

CLINIC FIELD CATTLE DISEASE VETERINARY GUIDE



Nexus Academic Publishers (NAP)
Lahore Pakistan

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For a full list of contents, please see the *complete table of contents*

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**Nexus Academic Publishers (NAP)
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DEDICATIONS

This book is dedicated to my parents who have given me the chance for an education and support throughout my life, to my brother's spirit (Dr.Ali Sadiek) who has been my friend guide, to my wife and children who have always stood by me and dealt with all of my absence from my family occasions with a smile.

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First of all, I would like to thank almighty god for his blessings and for all the achievement in my life. I am particularly indebted to my wife and children for inspiring me to this work. I take this chance to express my deep sense of gratitude to everyone who teaches me a letter.

Finally; I hope to acknowledge the services of the total team of publisher and everyone who collaborated in producing this book.

**With warm regards
Dr. Fathy Ahmad Osman**

Contents

ABSTRACT	1
INTRODUCTION	2
The most clinical Signs of Healthy Cattle	2
The Importance of Colostrums to Newborn Calve	3
Critical Point to Perform Physical Examine of Cattle	4
PART 1.THE MOST IMPORTANT DISEASE OF 1ST-4TH WEEK OF LIFE NAVEL-IL).....	10
Calf Scours	12
Pneumonia of Sucking Calves	14
Salmonellosis in Calves	15
Umbilical hernia	17
Septicemia of Newborn Calves.....	18
Worm Infestation of Sucking Calves	20
PART 2: THE MOST IMPORTANT DISEASE OF 5TH-12TH WEEK OF LI.....	20
Hypomagnesemic Calve Tetany (Whole Milk Tetany).....	21
Infectious Bronchopneumonia in Calves (Enzootic Pneumonia of calves).....	22
Shipping Fever Pneumonia (Hemorrhagic septicemia)	24
Tympani in calves	25
Cerebrocortical necrosis (Polioencephalomalacia).....	27
Vitamin E/Selenium deficiency (white muscle disease)	30
Coccidiosis	31
Rickets.....	33
PART.3.THE MOST IMPORTANT DISEASE OF REARING AND ADULT CATTLE	32
A. Metabolic disease.....	32
Indigestion.....	32
Grain Overload (Lactic acidosis, Carbohydrate engorgement, Rumen impaction)	34
Bloat (Ruminal tympany)	36
Osteomalacia (Creeps) (Adult rickets)	39
Post Parturient Hemoglobinuria (Hypophosphatemia, Heinz Body Hemolytic Anemia).....	40
Hypomagnesaemia(Lactation tetany, Grass Tetany, Grass staggers, Wheat Pasture Poisoning).....	42
Ketosis.....	44
Milk fever (Calcium Deficiency)	47

The Downer Cow Syndrome (Creper Cow)	49
Photosensitization	51
Traumatic Reticuloprotineties Hardware Disease (<i>Foreign Body Disease</i>).....	52
Vegal Indigestion (Chronic Indigestion).....	55
Abomasal Displacement.....	58
Bacterial Disease;	61
Pneumonia	61
Hypersomnia in Bull; [Infectious Septicemia Thrombosing Meningioencephalities (ISTME)].....	62
Tetanus.....	63
Mastitis.....	65
PARASITIC DISEASE	67
Verminous Pneumonia (Lungworm Infestation)	67
Fascioliasis (Liver Fluke Infestation)	68
Gastrointestinal Parasitic Infestation	70
Ringworm (Dermatophytosis)	71
Babesiosis (Tick fever, Cattle fever, Red water fever)	73
Theileriosis (East Coast Fever, Egyptian Fever, Tropical Theileriosis).	75
Anaplasmosis.....	77
Mange.....	78
VIRAL DISEASE	79
Influenza–Type Disease Complex	79
Infectious Bovine Rhinotracheitis 'IBR' (red–nose).....	80
Intestinal Diseases in Cattle (Bovine viral diarrhea and Mucosal disease complex)	81
Papillomas ' (Warts);	84
Foot and Mouth Disease	85
Bovine Ephemeral fever (Three day sickness).....	88
Lumpy Skin Disease.....	89
DISEASE DUE TO DIFFERENT CAUSES	91
Disturbed Urination	91
Diaphragmatic Hernia	93
Abortion.....	94
Foot Problems in Cattle	95
The Bovine Hoof anatomy	96

Types of Foot Problems	97
Torsion of Uterus.....	103
REFERENCE.....	95

Abstract

Cattle disease in field required several aspects to understand their pathogenesis and treatment, including complex subject matter typically with experience brought on by time and by attending educational part. For that these book provides information on disease that can affect individual animals or an entire herd. Including typical symptoms with typical images to help in identify the problem, advice for treatment and measure to prevent disease if it is available, the book include prepared items for this book as, the most clinical signs of healthy cattle, the importance of colostrums to newborn calves and the critical point to perform physical examination of cattle and the cattle disease were classified according to age of diseased animal into three parts as the following

- Part 1;The most important disease of 1st–4th week of life (Newborn disease).
- Part 2;The most important disease of 5th–12th week of life.
- Part.3.The most important disease of rearing and adult cattle.
- Part 3 divided into subclass according to causative causes. (Metabolic, parasitic, bacterial, viral and disease due to other causes).

The review supported by 66illustration, 61full color image and 5 diagram, related to information enhance all illustration don by the authors except of figure 4,7,31,39,42,55,59,60 and diagram 3,4 and 5.

I hope to provide a trusted source of field cattle disease for students and practicing veterinarians by this book, where it contain authoritative guidelines for the diagnosis,

INTRODUCTION

Cattle diseases are responsible for economic losses to cattle producers. These diseases are often either preventable or their effect on cattle production can be reduced. The diseases in cattle result in decreased production, poor growth, reduce feed conversion rates, reduced milk production, lower fertility rates and mortalities, and increased costs, costs of medications, labor costs and cost of replacement stock. Cattle like most organisms on earth are susceptible to a range of different diseases. These diseases each have varying causes, symptoms and treatments methods. In this article we will explore some of the diseases of cattle and their relevant information.

This book provides information about a listing of the common field internal disease of cattle including their symptoms, diagnosis, treatment and control. The health of a cattle herd depends on the caretaker being able to recognize disease symptoms of common ailments. Many common cattle disease symptoms can be an indication of several different health problems. Sorting the sick cattle and isolating them from the rest of the herd can keep the remaining cattle healthy even when several display common disease symptoms. Understanding the symptoms can help in making a correct diagnosis to begin an effective treatment.

The goal of this book is to profitably operate a cattle operation, introduce veterinarians to clinical medicine by reviewing the pathophysiological basis and associated clinical symptoms commonly noticed in respective fields and allow them to understanding the disease in simply picture, it is important to know about these disease and how to treat and control them. I hope to give a trusted source of animal health information for students and practicing veterinarians and consider this book as a small reference book where it contain authoritative guidelines for the diagnosis, treatment and prevention of cattle disorders.

The most clinical Signs of Healthy Cattle General behavior

Size and development corresponding to age; Lively, alert and inquisitive behavior, upright posture, lively facial expression, alert eyes and ear movements; Shining short coat. Calves readily approach visitors and alert to what is happening around them.

Body temperature	38.8–39.4 °C (102–103°F)
Pulse rate	72–92 per minute (strong and regular)
Respiratory rate	20–40 per minute,(calm ,regular)

Elasticity of the skin

Raised skin folds immediately level out again (within 1–2 seconds).

Urine

- Thin clear amber fluid.
- PH value.5.8–8.3.
- Specific gravity. 1.010 —1, 040.

Feces

- Yellow to light brown in the begging live which change to brown to olive green depending on feed, raining from pappy to formed and viscous without solid component in newborn calves.
- Daily quantity; 250–500g.
- First faces; Greenish–black, viscous, without solid components.
 - **Nose**
Wet and bright.
 - **Ears**
Warm

- **Eyes**
Clear, bright, no excessive tearing.
- **Vagina**
Some discharge may be present, normally odorless.
- **Udder**
Normal color, soft, Normal milk secretion from one or in all quarters.
- **Movement**
Normal gait and motion.
- **Hooves**
Healthy cattle stand straight and still while eating. Tipping or walking with lame gait are signs of poor hoof health, from bad rations, poor floors or lack of hoof treatment.
- **Ruminating**
Cattle should ruminate for seven to ten hours per day, ruminating 40 to 70 times on a cud. Taking less time indicates inadequate rations.
- **Rumen**
The rumen should be filled with feed. The left side of the stomach should protrude. If you press your fist into the rumen it should contract firmly about 10 to 12 times within five minutes.

The Importance of Colostrums to Newborn Calve

What is colostrums?

Colostrums is the thick, creamy–yellow, sticky milk first produced by cows initially.

Following calving and contains the antibodies necessary to transfer immunity onto their calves. It is essentially milk reinforced with blood proteins and vitamins. It has more than twice the level of total solids than in whole milk. It also contains a chemical allowing newborn calves to use their own fat reserves to immediately provide additional energy. Antibodies are proteins that are built by the immune system to prevent infectious disease and can only enter the blood from the intestines of the calf in the first hours after the calf is born. This ability to absorb antibodies decreases a few hours after birth and has gone by 24 hour and the calf is exposed to bugs immediately after birth; the bugs do not wait until the calf get colostrums. The quality of colostrums is defined by the concentration of antibodies. It can vary between cows. The second and subsequent milking of a dairy cow contains fewer antibodies and

should not be considered colostrums but as transition milk. Transition milk is milk that is not saleable from the first eight milking. Colostrums should not be mixed with transition milk and fed to new born calves.

How much should I give?

Typically provide at least 4 quarts of colostrums within the first 12 hours of life. Typically one feeding (2 qt) is given immediately and the additional 2 qt is given later. It is better to get the second feeding at 6–8 hours. Some producers feed 1 gallon at birth to get optimal absorption of the immunoglobulins. Also, colostrums are a great source of energy. Dairy calves only have enough fat to sustain them for about 18 hours. Getting colostrums into them provides them with high–quality fat which will help maintain their body temperature according to. Pete Erickson.(2010)..

How long should I feed the colostrums?

At least two feedings if you feed 2 quarts at a time, a gallon if you feed it all at once. However, data from Switzerland indicate that the longer you feed colostrums the healthier your calf will be according to Pete Erickson (2010).

How much IgG is present in the colostrums replacer?

Various amounts are available, from 60 g of IgG/dose to 100 g/dose. Our data indicates that a minimum of 100 g at birth will provide enough IgG for most calves to attain passive transfer. However, up to 200 g and provides better insurance to protect the calf. Pete Erickson.(2010).

How soon colostrums should be fed.

Calves need to receive individual attention and care immediately following birth. There is a direct link between good calf care and improved milk production and longevity in the milking herd. Calves are born with no immunity against disease. Until they can develop their own natural ability to resist disease, through exposure to the disease organisms in their surroundings, they depend entirely on the passive immunity acquired by drinking colostrums from their dam.

Colostrums are crucial for newborn farm animals. They receive no passive transfer of immunity via the placenta before birth, so any antibodies that they need have to be ingested (unless supplied by injection or other artificial means).The ingested antibodies are absorbed from the intestine of the neonate. The newborn animal must receive colostrums within 6 hours of being born for

maximal absorption of colostrums antibodies to occur. Recent studies indicate that colostrums should be fed to bovines within the first thirty minutes to maximize IgG absorption rates (Pakkanen et al., 1997). Colostrums vary in quality and quantity. In the dairy industry, the quality of colostrums is measured as the amount of IgG (Immunoglobulin G) per liter. It is recommended that newborn calves receive at least 4 liters of colostrums with each containing at least 50 grams of IgG/liter.

Calves are born with no immunity against disease until they can develop their own natural ability to resist disease through exposure to the disease organisms in their environment, they depend entirely on the passive immunity acquired by drinking colostrums from their dam. The concentrations of protein and vitamins A, D and E in colostrums are initially about five times those of whole milk, with a protein content of 17–18% compared with milk's 2.5–3.5%. However, within 2 days these are little different from those in whole milk. The levels of vitamins in colostrums are dependent on the vitamin status of the cow. The blood proteins transfer passive immunity from mother to offspring through maternal antibodies or immunoglobulins (Ig). The chances of calves surviving the first few weeks of life are greatly reduced if they do not ingest and absorb these antibodies into their bloodstream. It takes far fewer disease organisms to cause disease outbreaks in such calves than if they can acquire immunity from their dam. Calves without adequate passive immunity are four times more likely to die and twice more likely to suffer disease than those with it. Furthermore in certain situations blood levels of antibodies in heifer calves are directly related to their milk production in later life.

Current recommendations on colostrums feeding

Recommendations for colostrums feeding have changed dramatically over the last two decades. Twenty years ago, it was considered acceptable for all calves to run with their dams for 1, 2 or even 3 days and for her to pass on passive immunity through natural suckling. As producers learnt more about the causes and prevention of calf diseases, they became more 'colostrums conscious's. Current advice to farmers is to ensure all calves drink from their dam within the first 3–6 hr of life and, if not, to

provide additional colostrums from its mother or another freshly calved cow.

Age(days)	Period	Allowance (L)
1	≤ 6 hours	2.5
1	≤ 12 hours	4
2–3	Daily	3–4
Continued	Continued	Total milk

Table1: Indicated recommended colostrums allowance for newborn calves, where scours is a problem, continued feeding of good-quality colostrums is recommended up to 3 weeks of age.

Two feedings during the first day, 6–12 hr apart, and each of 2 L of good-quality colostrums used to be considered sufficient to provide passive immunity, mainly because of concern about the small capacity of the abomasums in newborn calves.

In case of dead mother cow or the calf is not able to suckle successfully. To ensure adequate quality, the colostrums consumed should be from adult cows, because heifers tend to have lower immunoglobulin levels in their colostrums. The amount of colostrums required to be consumed according to age is presented in Table 1. (Pakkanen et al, 1997).

Critical Point to Perform Physical Examine of Cattle

A physical examination is a routine medical procedure in which the physical symptoms of a patient are measured in order to determine if those symptoms fall within the normal range of that animal.

Physical exams should always follow a consistent routine – in most cases, every examination should follow the same order of steps even if you find a suspicious symptom, you should not stop the examination – there may be more symptoms to discover.

Steps of a Physical Examination

The first step of an animal physical exam is to speak with the owner in order to get a history of the patient.

The following 8 questions should always be asked:

1. What is the problem? Why did you call?
2. What symptoms have you observed?

3. When did this problem start?
4. Has this affected her feed consumption?
5. Has this affected her milk production or rate of gain?
6. How long ago did she calve?
7. How old is she?
8. Do any other animals have similar symptoms?

After you've taken the patient history, you should begin to examine the animal's head and neck.

You should assess the following parameters:

Ears – erect or drooping? Hot or cold?

If the ears are cold or warm? there is a problem. If her ears are warm, there might still be a problem where cold ears occur in hypocalcaemia (milk fever, or dangerously low levels of calcium in the blood).

If ears are erect, it is good status but drooping ears is bad.

Eyes – sunken or normal?

Eyes are a quick indicator of both hydration and of her emotional status. Are her eyes normal or sunken? If they sunken, the animal is dehydrated, also her eyes indicate if the animal in pain or not. Emotion status of animal can be read most easily from the eyes. Jaundice, anemia and parasitic infestation can be clinically diagnosed from the eyes.

Nose-Dry or moist ?

- Does animal have any mucus discharge and nature of discharge?
- Is it clear (good) or solid-colored (typically bad) .Is it clear (good) or bloody (bad).
- Nasal discharge (infection or illness).

Mouth

Cattle grinding her teeth or not? Circulation Jaw – does the animal have any swelling? Listen near her mouth. Is animal chewing her cud (very good)? Do you hear a high-pitched grinding noise (bad)? If so, this is a sign that the cow is in pain.

If animal compulsively licking or chewing, or are there any other signs of emotional /nervous problems.

Examination of mouth for presence of lesion as ulcers or vesicles (FMD, Sharp teeth), drooling salivation or not

Neck

Do cattle have swollen lymph nodes? Check her jaw – is there any swelling or fluid build-up (bad)? If so, this could be a sign of poor circulation or low electrolytes.

Neck – check the lymph nodes (they can be found along the jaw-line below the ears). Lymph nodes are glands full of white blood cells; they move lymphatic fluid (immune system-fluid) throughout the body if the lymph nodes are swollen, this is a clear sign that the animal is fighting an infection, illness or injury.

Swollen jaw (bottle jaw)?

Low electrolytes or blood protein levels or possible heart failure or parasitic infection (Fascioliasis or Haemonchiosis).

Skin – Is she dehydrated

To check for dehydration, you should perform a pinch test on the skin.

To perform the pinch test, firmly but gently grab skin between your thumb and forefinger. Pull the skin gently and then immediately release. Under normal conditions, the skin should immediately snap back to its original position. The return to its normal state should be instantaneous. If the animal is dehydrated, the skin will slowly return to its original state. It will behave sort of like cold bread dough;

Capillary Refill Time

When checking the mouth, you should also check the capillary refill time of the animal. Capillaries are the smallest blood vessels that link arteries to veins. They line the surface of the skin. To perform the Capillary Refill Time (CRT) test, gently and carefully lift the lip of the animal (make sure the animal is properly restrained). Gently push on the animal's gums and release. The color of the gums should go from a whitish color to its normal pink in 1–2 seconds. A slower time indicates shock or dehydration. A time under 1 second indicates heat stroke or shock.

After examining the head, you move to the left-hand side of the animal and examine their chest. At the chest, you will examine the

- Heart Rate: Should be 60–80 b p/m in normal case
- Respiration Rate. Should be 10–40 b.p/m in normal case
- Heart.



Figure1;

Auscultation the cranial part of the heart requires the head of the stethoscope to be some way underneath the elbow.

To measure the heart rate, you would use a stethoscope and place it behind the animal's left elbow. It may take a couple tries to find a good-sounding heart rate – move around until you find a good beat. When you check the heart rate, you will check for 2 things.

The apex of the heart lies caudal to the elbow at the sixth intercostals space. The base of the heart lies cranial to the elbow (figure 1). The aortic, pulmonic and mitral valves are ausculted on the left side of the heart. Insert your stethoscope deep under the axilla to find the pulmonic valve area between the shoulder and the elbow at the left third intercostals space. The aortic valve area is located at the left fourth intercostals space at the level of the shoulder. The mitral valve is located between the shoulder and elbow at the fifth intercostals space. Auscultation should take place in a reasonably quiet environment. The most successful way of examining the heart is to place the bell of the stethoscope sufficiently forward between the upper foreleg and the chest wall. The heart is located in the ventral part of the thorax between the third and sixth ribs. Careful interpretation of rate, rhythm, and heart sounds is necessary. Observe the jugular vein and mammary vein for any signs of distention or pulsation while you are ausculting the heart. Most dairy cows with cardiac disease have tachycardia at rest, but many diseases result in high heart rates (such as infectious diseases). Bradycardia (40–60 bpm) is often associated with vagal indigestion. Muffled heart sounds occur with pericardial and pleural effusion. Increased intensity of the heart sounds is associated with increased cardiac contractility. Cardiac diseases such as bacterial endocarditis and some cases of lymphosarcoma can be accompanied by fevers. The most common cause of murmurs is bacterial endocarditis. The most common valve is the tricuspid on the right side followed by the mitral valve on the left. Cardiac diseases may occur secondarily to GI diseases such as "hardware", traumatic reticulopericarditis. Muffled heart sounds with or without a washing machine-like murmur, distended jugular veins,

jugular pulse and brisket edema are compatible findings with hardware disease. Atria fibrillation causes a irregularly, irregular heartbeat, and is generally associated with a GI problem such as an LDA.

Rate

The rate should be 60–80 b.pm.

High heart rate (rate above 80 b.pm) – high heart rates can be a sign of a wide-range of problems, including infection, heat stroke, injury, nervousness, etc. The same is true for high breathing-rate (Up to 40 b/m).

Heart murmurs – if the heart beat does not have a 'lub-dub' sound (i.e. if you hear a whoosh or whistle), this is a sign are common symptoms you may encounter when examining the left side of the chest:

Quality

The heart should be a "lub-dub" sound. Whoosh' or 'Whistle' sounds are signs of other problems – these are called heart-murmurs.

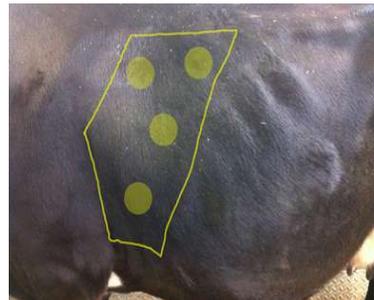
Respiration;

Figure 2; indicated the lung field extent of auscultation and percussion (auscultation locations shown in yellow circles).

To measure the respiration rate, you would use a stethoscope and place it above the animal's left elbow, it may take a few tries to find a quality and type of sounding respiration.

You will check for 2 things (Rate and Quality):

The assessment of abnormal lungs can be extremely difficult in the cow. The reason for this is that they have a smaller area to auscult due to the reduced number of ribs and a steeper angle of the diaphragm. The caudal border is the 11th rib. Note the area of auscultation that is outlined with tape in the picture to the left (figure 2).

Caution

Significant pathological lesions may be present without any abnormal lung sounds. Observation of rate and effort is important during auscultation. Cattle normally have a costoabdominal effort. Normal lung sounds are louder on inspiration than on expiration and are loudest in the ventral lung fields. Consolidation in the lungs caused by pneumonia often results in large airways sounds in the ventral lung field. Crackles and wheezes are abnormal lung sounds. Deeper breaths can be induced by holding off the nostrils. Palpate the tracheal to try and induce a cough.

Normal lung sounds are louder on inspiration than on expiration and are loudest in the ventral lung fields. Consolidation in the lungs caused by pneumonia after results in large airways sounds in the ventral lung field. Crackles and wheezes are abnormal lung sounds. Deeper breaths can be induced by holding off the nostrils. Palpate the trachea to try and induce a cough.

Rate

The rate should be 10–40 b.p.m

Quality

The breathing should be normal clear sound; it should not sound raspy or obstructed (dyspnea).

After examining the left chest, you would move to the left abdomen (stomach area).

In the left abdomen, you would examine the following:

Udder

The ideal udder has symmetrical quarters and teats that subjectively look ideal for milking. Pendulous and irregular quarters are generally the result of stretching due to repeated episodes of edema after calving or inflammation. These udders can be more difficult to milk but are probably not making the cow sick. Palpation of the udder gland is an art. The best starting place for inexperienced students is to compare the quarters to each other. It is rare that all 4 quarters have mastitis but common that all 4 quarters have edema. Generally an abnormal quarter will be obvious compared to the other 3 normal quarters. Abnormalities in consistency of the glandular tissue of the udder include edema, hardening and acute swelling. The palpation findings should be integrated with the examination of the mammary secretions and the physical exam findings. First, milk should be stripped from each quarter onto a strip plate, any deviation from normal milk in color and consistency is abnormal. Then strip each quarter's secretion on top of

each other and repeat in the reverse order. Any subtle abnormalities in secretion from quarter to quarter will be picked up in this method. This technique of stripping a quarter's milk onto a pooled milk sample is an excellent way to pick up watery milk.

It is critical to evaluate the mammary gland and integrate your findings with the rest of the physical exam to arrive at an accurate diagnosis and treatment plan. For example, a cow with clots in the milk or a watery secretion accompanied by a normal physical exam is handled differently than a cow with a watery secretion, elevated heart rate and temperature, rumen stasis and diarrhea.

Generally, contagious organisms such as, *Staphylococcus aureus*, *Streptococcus agalactia*, and *Mycoplasma bovis* tend to cause sub-clinical mastitis where udder palpates normally, the milk grossly looks normal, and the cow's physical exam is normal but with special tests such as California mastitis test or somatic cell counts would be needed to quantities any inflammation in the udder and bacterial culture would be necessary to make a diagnosis of the organism causing the sub-clinical mastitis.

Generally, the environmental organisms cause clinical mastitis (abnormal milk / swelling in the udder). The Strep group of organisms causes clinical mastitis 50% of the time and the coli form organisms (*Escherichia coli*, *Klebsiella*, *Enterobacter* sp., etc.) cause clinical mastitis 90% of the time. Probable diagnosis of the organism causing the mastitis can be made in certain cases. Secretions that smell foul and have a necrotic odor are usually caused by *Arcanobacterium pyogenes* or some anaerobe. Secretions that are very watery accompanied by a swollen udder are generally caused by coli form organisms. Secretions that are watery but red accompanied by a sick cow are generally caused by *Staph. aureus* and carry a poor prognosis for recovery. All other gradation of secretions between the normal milk and the extreme watery secretion are indistinguishable from each other and bacterial culture would be necessary to identify the organism causing the mastitis.

Rear Examination

After examining the udder, you would move onto examining the rear of the cow.

Rectal Temperature .Urine Ketones .Rear feet, legs and fecal sample (Grossly and microscopically);

Rectal Temperature

To take an animal's temperature, you will need a clean, lubricated rectal thermometer. Lift the tail and gently raise it as high as it will safely and humanely go. This will help to partially paralyze the cow's rear, reducing her likelihood of kicking while humanely restraining her. If done correctly, it should not cause pain. Gently insert the thermometer into the rectum using a twisting motion. Leave in place for at least 1 minute.

A cow's temperature should be 38–39 C^o while a higher temp (above 39C^o) indicates an infection or injury.

Rear Feet and Legs

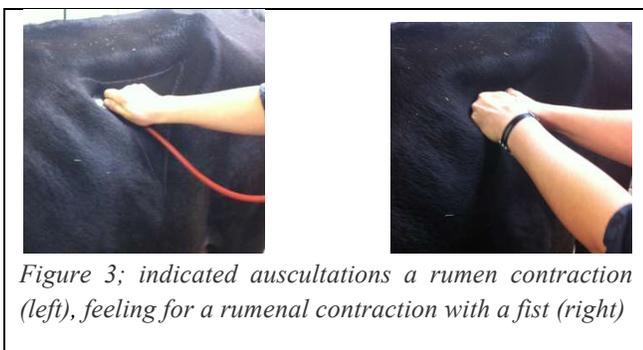
When examining the front and rear legs of the animal, you must attention for the following question:

- Cattle favoring one foot over the other or not?
 - How cattle legs when walking?
- Are there any noticeable signs of injury, lameness, or swelling?
- Is there any deformity?

Digestive Obstruction

To check for a digestive obstruction (such as a blocked or twisted colon), locate the hip of the cow (this is the bony part that juts out at the rear of the cow).

Rumen Contractions;



The rumen is the first chamber of the four chambered stomach of a cow.

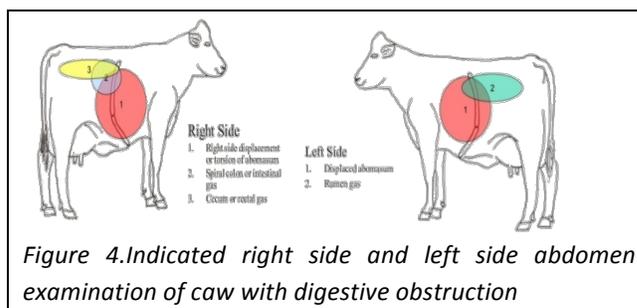
Does she have 1–2 contractions per minute?

The rumen has to contract at least 1–2 times per minute to sufficiently mix and break down the feed she has consumed.

To measure rumen contractions, place the stethoscope over the center of her body (Left flank as in figure 3). Listen for what sounds like low rumbles of thunder while watching for a minute to pass. A low rumen contraction is a symptom of digestive problems.

Listen to the rumen contractions by placing your stethoscope in the left paralumbar fossa. Normal rumen contractions (1–3/minute) are strong, and can be felt and seen. Palpate, auscult, and ballot the rumen to assess consistency. A gradation in consistency exists from the dorsal to the ventral sac of the rumen. The dorsal sac of the rumen generally has a gas cap and becomes more doughy in consistency (feed layer) towards the ventral fluid-filled sac of the rumen (figure 3). A rumen ping is differentiated from an LDA ping by its location over the left paralumbar fossa. It is possible to have a rumen ping and an abomasal ping at the same time, but they generally have different tones. If a ping with the same tone extends over both the rumen and the abomasal area, and rectal examination reveals a very small rumen with a ping with the same tone extends over both the rumen and the abomasal distended gas-filled dorsal sac, the ping is most likely a rumen void ping.

Abomasums placement;



The abomasums is the fourth stomach chamber of a cow. Does she have a displaced (twisted) abomasums?

It operates in the same way your stomach works.

If a cow stops eating or has other problems the abomasums can fill up with gas and twist over it. The abomasums will swell with gas like a balloon.

Left side: If the cow has a displaced abomasums or twisted stomach, you will be able to detect this by 'pinging' the cow. Place your stethoscope over what is the green area on cow left side (figure 4) and flick repeatedly with your finger and thumb.[Pinging sound is sometimes compared to the sound of basket ball hitting the cement].If the stomach has twisted, it will sound like a banjo through the stethoscopewhen you flick the area.

Right Side: Most of the symptoms that you will check would be found on the head or left-side of the animal. On the right hand side, you will check. Right displaced abomasums.

Check for 'pings' (figure 4) .If you hear a ping right around the hip, it is a sign of rectal gas. If you hear a ping to the right of the hip, it is a sign of a twisted intestine or intestinal gas.

Ping the left side of the cow by simultaneously percussion and ausculting the whole left side of the cow by firmly flicking your finger against the body wall of the cow. A "ping" represents a fluid-gas interface. On the left side of the cow, gas may be present in the abomasum (LDA), rumen, or peritoneal cavity. LDA pings are variable in tone, and often tinkling sounds are heard over the area of the LDA. Rumen pings are located over the rumen area and tend to be monotone. LDA and rumen pings often occur together and are usually distinguished by two distinct pings of different tones. The most confusing ping for us on the left side of the cow is a monotone ping that extends over the rumen and LDA area. Often, this ping is accompanied by a small gas-filled rumen on rectal palpation and is referred to as a "rumen-void" ping. These cows do not have surgical problems.

Wither Pinch test?

Wither is area consistency of the spine of the animal above the front feet.

If you gently pinch along the spine, the animal should have a reflex that resembles a "shrug" – the animal's spine should dip and their head should arch slightly. This is called a "dorsiflex".

If the cow does not respond to repeated withers pinches, it may have a internal pain or hardware disease. Hardware disease occurs when the animal accidentally consumes a piece of metal or other indigestible substance. The hardware will remain in the cow's reticulum (2nd stomach chamber).

Rectal Examination

The rectal exam should be performed last to avoid creating pneumorectum and confusing the interpretation of abdominal auscultation and percussion. The rectal examination is an extremely important part of the physical exam and even if a sure diagnosis is made prior to this procedure, it should never be skipped. A routine sequence of examination should be established and the examiner should use a lot of lubrication.

Manure and Uterine Discharge

Examine the perineal and tail area for evidence of blood, mucus, discharge or feces. Fresh blood on the tail suggests the cow has just gone through estrus. A moderate stream of clear, highly viscous (stringy) mucus suggests a cow may be close to or in estrus. Sometimes thin clear mucus may be seen with urine poolers (vaginitis) or cows with cystic ovarian disease. A variety of vaginal/uterine discharges may be evident depending on the stage of lactation. Fresh cows (cows that have calved recently) can have normal lochia (3–12 days postpartum) or depending on the severity of metritis may have thin, watery brown-red to tan discharge that is malodorous. The more mucoid the discharge, the less severe the metritis is for the cow. Normally cows should have a clean tail and perineal area. If there is a lot of manure on the tail, the cow may have diarrhea or perhaps tail paralysis. Inspect the manure for volume, color, fiber length, consistency, mucus-covering and odor. Absence of production of feces during the physical examination suggests reduced fecal output. Off-feed cows may produce scant, pasty feces and cows with intestinal stasis may have no feces or scant blood-tinged, thick mucus-covered feces.

The color of the feces varies with feed and bile secretions. A feces covered with orange mucus is typical of bile secretions seen in cows with fatty liver. Excessive amounts of mucus may be seen with constipation or inflammatory bowel disease. Digested blood appears black (melena) and is caused by blood in the abomasums or proximal small intestine, and would typically be found in a cow with a bleeding abomasal ulcer. Fresh blood indicates bleeding from the distal intestine, and may occur with colitis caused by coccidiosis or clostridium enteritis. Fibrin occurs in severe inflammation such as that caused by salmonellosis and may appear as casts. Diarrhea can be associated with fore stomach diseases

such as displaced or ulcerated abomasums, or small intestinal problems. In general, large quantities of liquid feces suggest a small intestinal problem such as Johns' disease, winter dysentery, salmonellosis, or enteritis of unknown origin (bad feed?). Large quantities of well digested soft feces may suggest a large intestine problem.

Amount – does she have diarrhea or is she constipated?

Color – is it a normal brown, or does it have red or black streaks in it?

Smell – does it smell like normal manure or does it have an especially–foul or –sour smell?

Consistency – is it runny, tarry, or extra stiff?

Symptoms in feces

- Brown, frosting–like consistency ,normal ,runny diarrhea
- Green & Watery – Jones disease.
- Sour–smelling – Salmonella infection or acidosis
- Slimy – excess fiber or protein
- Too little – twisted or obstructed colon or other digestive problem.
- Black or red streaks; internal bleeding (the redder the streaks, the later the injury in the digestive tract)

Pelvic examination

The pelvic/pubis brim and the iliac shafts. The pelvic brim is useful because of its central location. It is necessary to

do a thorough exam of the pelvic canal for gross changes that could be caused by severe vaginitis from birth trauma, abscesses or tumors. The reproductive tract including the cervix, and uterus should be located. The cervix is located in the middle of the floor of the pelvic cavity and should be freely movable. The uterine horns may be located in the pelvis or abdomen. Physiological and pathological conditions that affect the position and freedom of movement of the cervix are listed below:

Cervix predominantly pelvic and freely movable

- Normal no pregnant uterus
- Pregnancies up to 60 to 70 days
- Postparturient involution more than 14 days after parturition
- Pyometra and hydrometra with exudates or secretion <2 liters
- Chronic metritis without appreciable accumulation of exudates.

Cervix abdominal and fixed

- Pregnancies after 70 days
- Uninvolved postpartum tract
- Pyometra and hydrometra with exudates >2 liters
- Extensive adhesions
- Tumors (Lymphosarcoma)

Part 1: The Most Important Disease of 1st–4th Week of Life (Newborn disease)

Navel Ill (Omphalitis, Omphalophlebitis)

Infection of the umbilicus and its associated structures occurs commonly in newborn calves. Where the umbilical cord dries up within one week after birth

Omphalitis is inflammation of umbilical structures that may include the umbilical arteries, umbilical vein, urachus, or tissues immediately surrounding the umbilicus. The umbilicus consists of their types of structures and undergoes functional and anatomic changes at birth.

Umbilical abscess or infection of any of the three components of the umbilicus may produce local infection or be a source of septicemia

Etiology

A mixed of bacterial flora including: *E. coli*, *Proteus spp*, *Staph. Spp* and *Coryn. Pyogenes* may result in omphalitis, omphalophlebitis, omphaloarteritis and the infection of the urachus may be extended to the bladder causing cystitis.

The source of infection is most commonly the external environment coupled with failure of passive transfer. Bacteria isolated from calf umbilical cord remnant infection includes, *actinomyces pyogenes*, *E.coli*, *proteus* and *enterococcus spp*. The urachus is the most commonly affected structure in calves and the umbilical arteries the least.

Omphalophlebitis may extend the length of the umbilical vein into the liver and result in liver abscess formation.

Clinical Signs ;

Signs of navel or joint ill can occur as early as two days of age. If only the navel is involved, it will usually appear enlarged and wet. If the infection has moved into the bloodstream, the calf may appear depressed, have lameness or swollen joints, have cloudy eyes, have a poor appetite or diarrhea, or have a fever. Early in the disease, the navel may not be enlarged.

The umbilicus is enlarged and draining purulent material infection is easily noted (figure 5). In other cases the umbilicus may be dry and larger in diameter than expected, in addition neonates may have a completely normal – appearing dry external navel and severely ill from infection of the urachus umbilical arteries or vein.

The presence of pain on palpation of the umbilicus indicates inflammation.

Overt signs of infection are heat swelling, purulent discharge or pain. Concurrent signs of systemic infection such as joint infection, pneumonia, diarrhea or meningitis may be noted.

(1st) Omphalitis

Inflammation of the external aspect of the umbilicus and occurs commonly in calves 2 – 5 days after birth. The umbilicus is enlarged, painful and may be closed or draining purulent discharge. The calf is depressed, febrile and does not suck normally.

(2nd) Omphalophlebitis



Figure 5; Showing umbilical abscess.

- Inflammation of umbilical veins. Large abscess may develop along the course of umbilical vein and spread to the liver forming liver abscess. It usually occurs in calves 1 – 3 month age.
- Umbilicus is enlarged and containing purulent material.
- Inappetence, unthrifty, mild fever.

(3rd) Omphaloarteritis

Inflammation of umbilical arteries (less common), the clinical signs and treatment as omphalophlebitis.

- Accurate clinical signs of overt umbilical inflammation as described.
- Ultrasonography may help in evaluating a normal appearing navel.

The umbilical vein, arteries and urachus may be imaged in the newborn. The umbilical arteries leave the umbilical stalk and course on the other edges of the urachus in a parallel fashion. Persistent dilation of the umbilical vein or arteries with a hypoechoic fluid is seen with infection.

In calves the urachus normally retracts up into the abdomen at birth, and ultrasonographic identification of urachal remnant is abnormal.

Differential Diagnosis

The external appearance of an umbilical abscess is difficult to distinguish from that of an umbilical hernia, while in umbilical hernia an orifice in the abdominal wall is palpable but in umbilical abscess accumulation of fluid is palpable.

Lameness due to articular inflammation (septicemia) is also seen in salmonellosis and E. coli infection.

Treatment:

Sequel such as renal abscessation, joint inflammation, pneumonia and septicemia may develop if therapy is started too late or discontinued

1. **If the infection is limited to the navel area and has not invaded any joints**
 - Broad spectrum antibiotics for several days (Typically 10–14 days)
 - Surgical removals of the infected navel area have a good chance of being successful.
2. **If joints are involved and treatment is attempted,**

- Aggressive treatment by using approved broad-spectrum antibiotics for several days (typically 10–14 days or more).
- Oral or IV fluids therapy is given to treat and prevent dehydration. Their care may include heat lamps; adequate nutrition, clean dry stalls or bedding areas.
- Infected joints should be flushed by sedating the calf, clipping and thoroughly cleaning the skin over the joint and then using two large (14-gauge) needles placed on opposite sides of the joint to force sterile solution through the joint, first one direction and then the other. Joint flushing is usually repeated at 24- to 48-hour intervals for three treatments.

3. Closed abscess.

- The abscess was opened and the pus was drained as in figure 1.
- Washed with 1% potassium permanganate solution or hydrogen peroxide and application of drain (gauze with beta dine), in the umbilicus.
- On second day wound was cleaned with hydrogen peroxide and iodine tincture.
- The area surrounding the umbilicus was smeared with fly repellent ointment to prevent the infestation of the wound with maggot.

Prevention

Prevention is the key to this disease. Ensuring that the cow calves in a clean environment will significantly reduce the risk of joint ill (and many other diseases such as toxic mastitis and metritis). Proper planning and preparation can prevent the build-up of disease that occurs in too many calving areas.

Applying a disinfectant (such as iodine) to the navel can reduce the risk of bacteria entering via the navel, but it is no substitute for good hygiene. No amount of disinfectant can overcome being born in a dirty wet yard. Because of the anatomy, bulls navels tend to dry slower than heifers and they are thus at more risk of navel ill. Applying disinfectant two or three times to bulls can reduce the risk. It is also important to ensure that if cattle are born in a nice clean environment that they aren't moved to other pens or contaminated pastures until the navel has dried completely.

Calf Scours

Calf scours is not a disease, it is a clinical sign of a disease which can have many causes and causes more financial loss to cow-calf producers than any other disease-related problem they encounter. That is caused by a variety of infectious agents and conditions. In fact almost any disease agent which attacks calf during the first three weeks of life will result in some level of scours (diarrhea). The reason is because the agent is still so immature during that period and is the weakest point of the calf's systems. In diarrheas, the intestine fails to absorb fluids and/or secretion into the intestine is increased where a calf is approximately 70% water at birth. Loss of body fluids through diarrhea can produce rapid dehydration. Dehydration and the loss of certain body salts (electrolytes) produce a change in body chemistry and severe depression in the calf. Although infectious agents may be the cause of primary damage to the intestine, death from scours is usually due to loss of electrolytes, changes in body chemistry, dehydration and change in acid-base balance rather than by invasion of an infectious agent.

Causes.

- Calf scours is not actually a disease, but a sign of a disease which can have many causes. The known causes of scours are grouped into two categories:
- Noninfectious causes and infectious causes.
- The noninfectious causes are often referred to as “predisposing” or “contributing” factors, whatever they are called and there is a dramatic interaction between noninfectious causes and infection.

Infectious Causes of Calf Scours

Infectious causes of calf scours may be grouped as follows:

- Bacterial cause;
 - E. Coli ,*Salmonella Spp* ,*Clostridium perfringen* and other bacteria
- Viral causes;
 - Rotavirus ,Coronavirus, BVD virus, IBR virus
- Protozoan parasites; *Cryptosporidium* ,*Coccidia* Yeasts and molds.

Noninfectious Causes of Calf Scours;

- Noninfectious causes are best defined as flaws in management which appear as nutritional

shortcomings, inadequate environment, insufficient attention to the newborn calf, or a combination of these.

- Recent research has indicated that many scour cases can be directly related to colostrums intake by the newborn calf. A calf that is well mothered and consumes 1 to 2 quarts of colostrums in the first few hours after birth absorbs a higher level of antibodies. This calf is far less susceptible to scours and other calf hood diseases.
- The pathogens usually localize in the calf's gut, causing the all too familiar white/grey to yellow watery diarrhea. Under most circumstances, however, it is not as important to identify the specific pathogen as it is to treat the animal and immediately prevent new cases of calf scours from occurring.

Clinical signs;



Figure 6; New born calves with signs of watery diarrhea.

Rotavirus Scours. A reo-like virus can cause scours in calves within 24 hours of birth. However, when the infection is first introduced into the herd, it can affect calves up to 30 days of age or older. Infected calves are severely depressed. There may be a drooling of saliva and profuse watery diarrhea. The feces will vary in color from yellow to green. Calves lose their appetite and the death rate may be as high as 50 percent, depending on the secondary bacteria present (Figure 6).

Corona virus Scours occurs in calves that are over 5 days of age. When the infection first starts in a herd calves up to 6 weeks of age may scour. These calves are not as depressed as those infected with rotavirus. Initially, the fecal material may have the same appearance as that caused by rotavirus. As the calf continues to scour for several hours, however, the fecal material may contain clear mucus that resembles the white of an egg. Diarrhea may continue for several days. Mortality from corona virus scours ranges from 1 – 25 %.

Clinical signs associated with salmonella infection include diarrhea, blood and fibrin in the feces, depression, and elevated temperature. With Enterotoxaemia the disease has a sudden onset. Affected calves become listless, display uneasiness, and strain or kick at their abdomen. Bloody diarrhea may or may not occur.

A typical sign of coccidiosis in young calves is diarrhea with fecal material smeared over the rump as far around as the tail will reach. This may or may not contain blood. Death may occur during the acute period or later from secondary complications.

Nutritional scours is usually white scours caused by undigested milk passing through the intestinal tract. If a calf is not running around and feeding regularly chances are it is sick. Common symptoms are fever, weakness, separating them from the herd and laying down, as well as dehydration. You can tell if a calf has dehydration by pinching the skin on the calf's neck, if it does not immediately go back to the body of the calf then the calf is dehydrated.

Diagnosis

- A. Accurate history, clinical signs, and culture of internal organs for bacteria and serotyping of the organism.
 1. Diarrhea—Temperature may be normal or high increased.
 2. Dehydration signs. (Depression, sunken eyes, dry skin and the calf will probably be unable to stand).

The characteristic course of the diarrhea in the stock, particularly its very appearance in sucking calves during the first few days of life, is typical of this infection.

- B. The diagnosis through laboratory work of culture revealed that the cause could be single agent, such as salmonella and may be variety of agent. (E.Coli–Rotavirus .etc)

Differential diagnosis; Similar symptom is caused by;

- Salmonellosis but this disease does not occur in any stock until the second week of life.
- Inappropriate feeding also produces similar symptom.

Treatment

Treatment for scours is very similar regardless of the cause. It should be directed toward correcting the dehydration, acidosis, and electrolyte loss. Antibiotic

treatment can be given simultaneously with the treatment for dehydration. Dehydration can be overcome with simple fluids given by mouth early in the course of the disease. If dehydration is allowed to continue, intravenous fluid treatment becomes necessary.

When Should You Treat with Electrolytes?

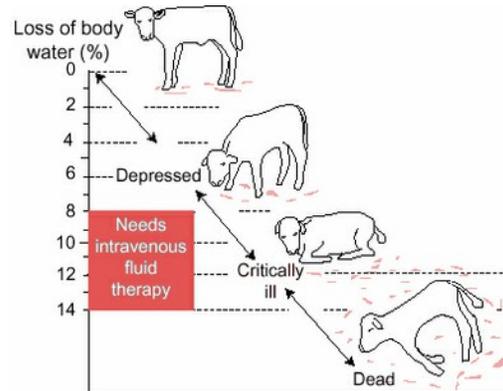


Figure7. Visual representation of percent dehydration as related to clinical symptoms and health of calves.

Calves can lose 5 to 10% of their body weight in water within 1 day of scouring. Fluid loss in excess of 8% requires IV treatment, and over 14% loss can result in death (Figure 7) .Adapted from Wattiaux M. A.(2005).

This is why it is extremely important to monitor calves daily and treat them quickly when signs of illness are observed. The amount of water lost by scouring calves can approximated using symptoms such as skin tenting, gum condition, attitude, and ability to stand or suckle as in (Table 2).

Dehydration	Symptoms
5–6%	Diarrhea, no clinical signs, strong suckling reflex
6–8%	Mild depression, skin tenting 2–6 seconds, calf still suckling, sunken eyes, weak
8–10%	Calf depressed, laying down, eyes very sunken, dry gums, skin tenting >6 seconds
10–14%	Calf will not stand, cool extremities, skin won't flatten when tented, comatose
Over 14%	Death

Table 2 :Clinical symptoms that help evaluate amount of dehydration in calves. Adapted from naylor J. M ,Can. Vet. J.

To evaluate hydration using skin tenting, pinch a fold of skin (best done on the neck) and count the seconds it takes to flatten. Flattening of skin in less than 2 seconds indicates normal hydration. If skin takes 2 to 6 seconds to flatten, the calf is about 8% dehydrated. Over 6 seconds indicates severe dehydration above 10%. Gums can be evaluated by looking at their color and feeling them for moisture. Normal gums should be pink and damp but if gums are white and dry this indicates 8 to 10% dehydration. One of the best measures of estimated dehydration and illness in calves is their attitude during milk feeding. Calves may show no symptoms of dehydration but if they need encouragement to drink, monitor them closely for scouring or other illnesses.

- | | |
|-------------------------|--|
| 1 –2 nd day | <ul style="list-style-type: none"> ➤ Replacement of milk or milk substitute by electrolyte solution (90 gm electrolyte salts+15g (cholortetracycline +vitamin AD3E+Sulfa drug) in water by mouth, twice daily between feed. ➤ Severely ill calves. In addition to drinking fluid, 250ml fluid saline therapy administrated by I/V. ➤ 1.5 ml Apicilline or sulfamethoxyprizine/10kg bodyweight. Twice daily at interval of 12 hours for three days. ➤ When internal parasites cause scours in older Grazing calves, treat these calves with an Anthelmintic drench. |
| 3 – 4 th day | <ul style="list-style-type: none"> ➤ Change over to milk.1 liter of milk and1 liter of electrolyte solution, and milk increases step by step to full intake in 6th day |
| 7 –14 day | <ul style="list-style-type: none"> ➤ 10 gm (chlortetracycline +sulfadimidine +vitamin A) per day/calf in the drinking water. |
| N.B | <ul style="list-style-type: none"> ➤ They must receive 2 liters fluids (Ringer solution, electrolytes and energy).Where the fluids are essential in order to allow the body organs such as the kidney, liver to continue to function, the fluid cannot be absorbed from the gut unless it contains electrolytes (salts) in the proper preparation. ➤ Most calves with scours tend to be acidic. It will help these calves to receive electrolytes that are alkaline (basic) in nature for 24–36 hours. Bicarbonate or lactate can be used. ➤ Determination the frequency of treatment needed by the amount of dehydrations present. ➤ Intravenous (IV) treatment is often necessary in severe or non–responsive cases. |

Prevention

General Principles

Ensure that all newborn calves receive colostrums. If the delivery was difficult, the dam may be tired or painful, and the calf may be weakened as well; this may result in a failure of the calf to nurse colostrums. In such cases, it is prudent to milk the colostrums from the dam and feed it to the calf via an esophageal feeder. How much colostrums should a calf receive? The calf must nurse or be given 2 quarts of colostrums during the first 2–4 hours after being born and a total of 4 quarts in 12 hours. It is often a good plan to obtain fresh colostrums from a local dairy and freeze it or purchase a colostrums replacer for occasions when the dam does not have colostrums. If sourcing colostrums from a local dairy farm, beware of potential pathogens that can be transmitted through colostrums such as Johne’s disease and Bovine Leucosis Virus.

Consider a vaccination program for your cow herd. Be sure to consult your local veterinarian about vaccine products and time of administration. Timing is critical as cloistral antibodies need to be in adequate concentrations in colostrums to provide ample passive immunity to the calf.

Maintain a clean calving area. Do not calve on pastures where cows have been kept in large numbers for long periods of time or sours have been recently diagnosed.

Calve in dry areas and drain pastures or corrals to minimize accumulation of moisture.

Pneumonia of Sucking Calves

Pneumonia is a respiratory disease that causes rapid breathing, wheezing, coughing and discharge from the nose. Force–fed calves can develop pneumonia as a result of the milk settling in the lungs.

The most common disease seen in calves. Initially caused by a range of viruses, but also by secondary bacterial infections, which can strike more easily when forms of stress occur that weaken immunity. Pneumonia leads to reduced growth performance in calves even after recovery. Calves generally cough, pant, discharge mucus, become depressed and have a high temperature of over 39.6°C. Further signs include sweating backs and a loss of appetite.

Pneumonia in sucking calves also called pneumococcosis is a generalized bacterial infection which takes very rapid course and affects mainly the respiratory organs and the disease occurs particularly in the third week of life, mainly during the winter.

Causes

The causative organism is *diplococcus pneumonia*, several types of which occur in man and animals, where the types occurring in man are also pathogenic to calves.

Methods of infection

The disease is transmitted by droplet infection (cough). In many cases the pathogens are introduced by staff working in the cattle house who have latent infection.

Clinical signs ;



Figure.8.indicated cough, breathing from mouth, extended head.(right) . Nasal discharge(left).

- The very rapid course of the disease (highly acute) usually lead to death within a few hours but with clinical pneumonic calf, the body temperature rises to 41.5 c., cough, nasal discharge and breathing from mouth (figure 8).
- Inflammation of the large airways (bronchi) and focal changes in the lungs are observed. With highly acute forms (septicemia) these signs may not be present.

Treatment

Treatment must be started immediately at the first signs of the disease.

- 1st-3th days**
- Administered feverish calf by antipyretic drug.(Analgin ,Novalgin)
 - Appropriate antibiotic is usually indicated.3 ml pencil line/10kg bodyweight by IM,as soon as possible.
 - Supportive treatment with non steroidal anti-inflammatory drug can be useful.
 - Fluid therapy may be used if

dehydration or toxemia is present.

- 1st-7th days**
- Affected calf and neighboring calves.4gm (chlortetracycline + streptomycin + penicillin +vitamin A)/10gm bodyweight daily for 7days via the drinking fluid.

Salmonellosis in Calves

Salmonella is a Gram negative bacteria comprehending more than 2000 species. Salmonellosis is a bacterial disease with a rising prevalence in the cattle industry. It is most common in dairy calves one to ten weeks of age, but can also be seen in adult dairy cows and beef cattle. Salmonellosis has a serious economic impact on the cattle industry worldwide. Livestock mortality, treatment costs, abortion, reduced production, discarded milk and reduced consumer confidence all contribute to the cost of salmonella to cattle industries.

Salmonellosis is an infection of the digestive tract caused by the bacterium, *Salmonella Enteric*. *Salmonella Enteric* has over 2,000 strains. Fortunately cattle are usually clinically infected by less than 10 of them. The majority of Salmonella that infect cattle are in groups B (species example –*S.Typhimurium*), C (example –*S. Montevideo*), D (example –*S. Dublin*), or E (example –*S. Anatum*).

Salmonellosis is an epidemic with a high mortality rate, which causes substantial losses. The pathogens, salmonella bacteria initially multiply in the intestine where they cause severe inflammation and diarrhea.

Incidence

The disease occurs throughout the world where all animal species and man may contact salmonellosis . Infection becomes possible due to the presence of adhesions and the different invasive ability in the various strains. Some strains produce enterotoxins and cytotoxic toxins. Salmonella has a good resistance; it can survive for one year in the ground. It is sensible to one percent formaldehyde solution, one percent glutaraldehyde solution and formalin. Salmonellosis is an important zoonoses deriving from contact with infected animals or contaminated equipment. In calves salmonellosis usually appears in epidemic form at the age of 2 to 6 weeks and causing heavy losses. Calves surviving the disease often become carriers. These are animals which survived the

infection in the intestine and continue to excrete salmonella without showing signs of the disease. They continually infect other animal.

Rout of Infection

- Purchase of calves is the main source. The danger is particularly acute if the animals purchased originated from several different sources.
- Man must be regarded as a further source of infection; salmonellosis is introduced by persons moving from cattle house to cattle house in the course of their work.
- Domestic and wild animals (dog, cats, mice, rats, deer, rabbit) may also transmit the disease. Salmonellosis pathogens may be also be introduced by the purchase of contaminated feedstuffs.

Clinical signs;



Figure 9. Signs of diarrhea in hind quarter and yellowish color diarrhea.

The disease sometimes causes sudden death without symptoms but usually diseased calf suffering from anorexia, fever, diarrhea and dehydration. Diarrhea is mucous at first and becomes bloody fibrinous with shreds of mucosa. During septicemia articulations, the lungs are also involved. The inflammatory lesions are markedly present in the small intestine but they could also be found in the abomasums and colon. There is congestion, whitish mucus, pseudo membranes easily removable or not and blackish or spotted mucosa.

Intestinal inflammation giving rise to diarrhea with yellow fluid and malodorous faces. The faces increasingly change color become grayish–green and contain mucosal fragments. With further progress of the inflammation the entire intestinal tract is affected and hemorrhages appears in the large intestine and rectum. The faces

become dark brown or black. The severs bloody intestinal inflammation alone can lead to the animal's death within a few days (Figure 9).

Right at the start of the intestinal disorder the calf shows dullness and a raised temperature however the animals continue to accept drink.

Diagnosis;

- Accurate clinical signs and case history.
- Laboratory examination.

It should be performed on fresh feces from not treated animals, the liver, the Liquid of the joints, intestine or whole carcass. The samples should be refrigerated but preferably not frozen. The bacteriological examination is performed through the use of different media: a pre–enrichment, a selective enrichment, solid selective; solid and non selective and media for confirm tests. Biochemical reactions and other tests are also usually employed. Serotyping of isolated salmonellae is carried out by a test of Fast agglutination on a slide.

Differential diagnosis;

The diagnosis should differentiate salmonellosis from colibacillosis, rotaviruses ,cryptosporidiosis, coccidiosis, mucous membrane disease, colisepticaemia and clostridiosis.

Treatment

- Treatment is aimed to restoring the correct hydro–saline balance and fighting the infection. Rehydrating therapy is always necessary while antibiotics are indicated only in cases of severe diarrhea with a rise in body temperature.
- Antibiotic resistance is increasing in anti–salmonellosis therapy and for this reason it is preferable to make the choice of the molecule on the basis of the literature reports, farm experiences. It is important to provide a mass treatment calculating the adequate pathology and administrating for at least four to five days.

Disease	Scours of sucking calves	Salmonellosis	Poisoning	Nutrition-Induced diarrhea
Cause	Rota and corona viruses. –Coli bacteria	–Salmonella bacteria	Furazolidone or poisons which inhibit blood coagulation	Incorrect use and composition of feed
Time of appearance	1- or 2- week of life usually 3- to5th day	2- to 6- week	All age groups, usually from the 3- week of life.	All age groups
Type of farm/system of keeping animals		Calf fattening and rearing	All system of keeping animals	All system of keeping animals
Symptoms	<ul style="list-style-type: none"> ➤ High temperature, occasionally abnormally low ➤ Drugs have particularly no effect on the diarrhea. ➤ Animals strains to defecates 	<ul style="list-style-type: none"> ➤ Moderate to high temperature. ➤ Diarrhea ➤ Swelling of joint in some animals 	Temperature often raised; hemorrhage in conjunctiva and convulsion possible. Bleeding following injury very difficult to stop.	Flatulence diarrhea; temperature normal or below normal.
Consistency of faces	Aqueous with undigested coagulated milk constitute	<ul style="list-style-type: none"> ➤ Mucous fluid and pappy malodorous admixture of blood. ➤ Color grey to dark green. ➤ Off-white mucoid flakes(fibrin) 	Soft faces with fresh blood	Fermentative diarrhea; fluid/pappy, yellow to dark brown

- Immediately**
- –Collected fecal sample before starting treatment and adapt treatment to result of antibiotic sensitivity test.
 - – Isolate affected animals from healthy one and disinfection the pasture.
- 1st– 5 days**
- Early treatment is essential for septicemia salmonellosis by use systemic antibiotic, as Ampicillin or cephalosporins to lyses of the bacteria and release of end toxin.
 - NSAIDs or flunixin meglumine may be used to reduce the effects of endotoxemia.
 - Oral medication is chosen, it should be given in drinking water. 4g sulfamethoxy pyzine or trimethoprim–sulfonamide combinations /10 kg body weight.
 - I/V administration of Fluid therapy to correct acid–base imbalance and dehydration may be necessary.

Umbilical hernia

The umbilicus in newborn calves consists of the urachus, a tube that connects the fetal bladder to the placental sac, and the remnants of the umbilical vessels that transported blood between the fetus and its mother.

Normally, just after birth, these structures shrink until only tiny remnants remain within the abdomen. If bacteria gain entry through the umbilicus, however, those remnants can become infected and require surgical removal. Additionally, if the area in the body wall through which these structures passed remains open, the intestines can protrude through the defect, resulting in an umbilical hernia. Umbilical hernias are the most common birth defect in calves and may be more common in the Holstein–Friesian breed.

Causes

Umbilical hernia is the most common form of congenital defect in calves. It appears that Holstein–Friesian breed cattle are more commonly affected. An umbilical hernia forms when the opening in the ventral abdomen through which the umbilicus protrudes (the umbilical ring) fails to close properly after birth.

Some factors that can increase the likelihood of this happening are cutting the umbilicus off close to the body wall, and excessive traction being applied to an oversized fetus during delivery.

A strangulated hernia develops when a loop of intestine slips through the umbilical ring and twists on itself, thus cutting off the blood supply to the tissue. The section of bowel is usually a loop of small intestine but it can be cecum or large colon. Umbilical hernia is often hereditary, but may also be caused by softening, over distension or injury to the abdominal wall.

In predisposed animals, a sudden increase in abdominal pressure lead to distention of the abdominal wall at the umbilical orifice and finally to visceral prolapsed. The abdominal wall and outer skin form the hernia sac.

Clinical Signs;



Figure 10; Showing umbilical hernia in calve and hernia ring.

Calves with simple hernias may seem completely normal except for a reducible umbilical hernia, where the hernia contents can be easily pushed back into the abdomen through hernia ring (figure 10).

Calves with infected hernias may be sick and show signs such as fever, in appetite, and poor growth rates and if the umbilical vessel remnants are involved you may see frequent urination in small amounts and urination through the umbilicus. Infection of the umbilical vein can spread to the liver, causing fever, and poor growth rates. Strangulating hernias are painful because the blood supply to the affected segment of bowel is compromised. This condition traps gas within the twisted segment and if left untreated, the segment will eventually die due to a lack of blood flow. The signs associated with a strangulated umbilical hernia include a warm, swollen, firm and painful hernia sac accompanied by signs of colic.

Diagnosis

Calves are physically examined to try to reduce the hernia contents. In some instances, it is possible to palpate an infected stalk within the hernia sac or around the umbilicus. Sedation may be required to place the calf on its back or side to better feel these structures.

Diagnosing a strangulated umbilical hernia can be done by observing signs such as a swollen, painful, firm umbilical sac along with fine needle aspiration of the swelling to differentiate it from an umbilical abscess. Umbilical hernia can also be differentiated from an umbilical abscess by palpation of the protrusion where if an umbilical abscess is present, an evenly distributed accumulation of fluid is felt, which cannot be pushed back into the abdominal cavity.

- Ultrasound examination is useful in determining whether there is any infection present and how extensive it is.
- Laboratory work such as white blood cell counts or fluid collection from the mass for a bacterial culture can also be useful detecting infection..

Treatment;



Figure.11. umbilical bandage and hernia management.

In calves with a small hernia, it is often sufficient to apply a hernia bandage for three weeks but if the hernia is large surgical repair is the treatment of choice where surgery can occasionally be performed under sedation with local nerve blocks (figure 11).

The umbilicus and its associated structures and all infected tissue are removed when the infection extends all the way to the bladder.

Septicemia of Newborn Calves

Septicemia is a systemic infection in which bacteria and their toxins get into the bloodstream and travel

throughout the body. Some types of toxin-forming bacteria gain entrance to the body via the GI tract, after damaging the gut lining and slipping through it, causing very rapid death. The calf goes into shock when internal organs are damaged and start shutting down. Any blood-borne infection may become life-threatening if the bacteria or their toxins damage vital organs. In some instances the infection may localize, creating internal abscesses, or may settle in the joints and causing a painful arthritis (“joint ill”).

Causes

Dr. Austin Hinds (food animal medicine and surgery specialist at the Cain Center, University of Idaho, Caldwell) says the most common reason calves develop septicemia is inability to fight the pathogen – and this is often due to poor colostrums. “If the calf was born early and the dam hasn’t bagged up yet (and has no colostrums) or for some reason the calf didn’t receive colostrums, he is at risk. For instance a cow might have twins and abandon one of them. Sometimes the cow will give birth to the first one, get up and walk away, lie down and have the second one. She may forget the first one and he doesn’t get colostrums. Heifers may not have good quality colostrums and/or not much of it. Some heifers may not have good mothering instincts so the calf is slow to nurse,” Hinds said. High risk calves also include calves that had a difficult birth or were delivered by C-section. “When a calf is born he has antibodies, so he gets these from his mother’s colostrums. When a calf is born his intestine is set up to absorb these large antibodies (IgG) for a short time. This is his source of immunity until his own immune system develops.

Bacterial infection may enter the body through various routes. It only becomes septicemia if the blood picks it up and takes it everywhere. A common location for blood-borne infection to localize is in the joints. “There is a lot of vasculature at the joints, and this is also a noticeable location because the joints swell and become painful and the calf is lame.

Rout of Infection

The navel and the intestinal tract are the main point of entry of the infection. Following an inflammation of the navel or intestine, the bacteria pass into the blood stream and spread.

Septicemia may develop from pneumonia or an intestinal infection. In the very young calf, diarrhea is generally caused by *E. coli*. If that bacterium crosses the GI tract into the bloodstream of a calf that has no immunity, it will become septic and hard to save without early and diligent treatment.

Clinical signs;



Figure 12 .Indicated inflamed joint in calve (left) and A septic calf may be unable to get up, with signs of shock including pale (or dark or red) gums (right).

Calf with septicemia will be weak, dehydrated and may or may not have a fever. In the later stages his temperature will drop and become subnormal as he goes into shock and calf may be unable to get up.

Signs of shock (pale gums or dark or red, rather than normal pink, cold feet, cold ears and the circulatory system is failing(The heart may be beating really fast, trying to get blood to vital organs as everything starts shutting down) .

If the calf is young and sick and maybe has swollen joints you might also check the navel for signs of infection such as swelling, heat, a thick umbilicus or pus discharge,

Reduce appetite, listlessness and prolonged periods of lying down are the first signs.

The joints are increasingly filled with products of inflammation, are hot to the touch and painful (figure 12).

Diagnosis;

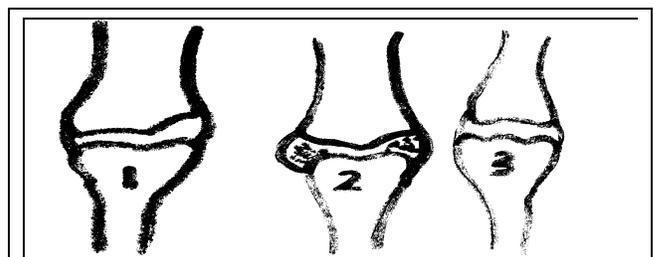


Diagram 1); 1- Normal joint.
 2-Inflamed joint, filled with synovial fluid.
 3-Rickets joint showing tumefaction of the bone at the joint

➤ **Clinical signs and case history;**

The disease must be differentiated from calves with rickets (Impaired calcium / phosphorus metabolism and/or vitamin D deficiency) where it show tumefaction of the bones at the joints, whilst joints with inflammatory changes are filled with synovial fluid (as in diagram 1).

Treatment

Septicemia is very hard to treat. “Often by the time you realize the calf is sick, it’s too late. The whole body is under attack and stress. When you find a septic calf you are usually way behind the eight ball,

Often when we see septicemia, it’s because a bacterial infection is not responding (not susceptible) to the antibiotic being used for treatment of scours, pneumonia, navel infection, etc. Bacteria have gained entrance to the bloodstream, often in spite of antibiotics we were using for something else. We need to do an antibiotic sensitivity test, to make sure we can treat with something that will work, or change to a different antibiotic that will work better than what we’re using,”

- 1–3· day** ➤ Antibiotics would be given. The best type of antibiotic would depend on the situation and cause of infection, and the stage of disease. 3 ml penicillin, streptomycin/10 kg bodyweight intramuscular at intervals of 24hours.
- No steroidal– anti–inflammatory, 10 ml phenylbutazone or dexamethasone.
- Fluid therapy is also important, and the stage of disease will determine whether it could be given orally, under the skin or IV.
- Calves that are in shock will need IV fluids as part of treatment. “The antibiotics and Banamine will also help, and we have to get some energy into the calf, if he’s not nursing. We need to get a little milk into him — unless the gut is completely shut down.
- 1- to 7- daily** ➤ Additional treatment via drinking fluid 20 g of (Chlortetracycline, streptomycin, penicillin, vitamin AD3E), per calf/day.
- Joints and Possibly** ➤ Apply and rub camphor, Ichthyol, iodine ointments twice daily.

Umbilical Hygienic Measure

- Cleaning and disinfection of place in cattle house or calf pen

Worm Infestation of Sucking Calves

Roundworms (*Toxocara vitulorum*) and threadworms (*Strongyloides papillosus*) infest calves particularly in the first few weeks of life.

Gastrointestinal parasites are typically a problem of young stock. While calves are drinking milk or suckling their mothers (beef) they are relatively safe from most worms. However from weaning until about 15 months old they are vulnerable.

Worms may absorb nutrients from the host’s food within the gut. They can destroy mucosal gut cells, resulting in inflammation, secondary bacterial infection and ulceration. They can suck blood, physically obstruct the gut lumen and damage other organs as they migrate through the body. Sometimes they may activate the immune system causing hypersensitivity of the gut.

Pathogenesis of infection

If cows ingest the eggs of roundworms or threadworms their larvae are hatched in the intestine, the larvae migrate through various organs, finally reaching the subcutaneous connective tissue and the udder where they lie dormant until the next calf is born while some of these larvae dead. As a result of hormonal changes at the time of birth, the dormant larvae regain their activity and migrate into the milk ducts and are excreted in the colostrums and milk for up to 3 weeks after birth. The calves thus become infected via the milk (Galactogenic infection) during the first few hours of life. Strongyliodes larvae may in addition penetrate the skin (percutaneous infection) and develop in the small intestine into mature worms and the eggs of which are excreted in the faces.

Clinical signs

In sever infestation the calves show varying appetite, constipation and diarrhea may be occurring. The coat becomes rough. Signs of colic may occur in sever worm infestation.

The symptoms may worsen and even lead to death unless they are treated. Animals of all ages may show symptoms, especially weight loss.

Small numbers can cause acute symptoms with blood and protein loss and can cause severe anemia or sudden death.

Diagnosis

- Accurate clinical signs and case history.
- microscopic examination of fecal samples for worm eggs.

Treatment and Control

In order to interrupt the life cycle of roundworms and threadworms and to clear the affected stock from infestation. This can be done using:

1. Use of low risk pasture/clean grazing.
2. Wormer treatments.
3. Integrated systems

However control of roundworms by clean grazing alone is impractical on many farms due to lack of available clean grazing. Wormer [anthelmintic] treatment is often necessary. Each calf is de-wormed at the age of 10 to 14 days; this treatment must be carried out by the 20th day at the latest.

- Carry out de-worming treatment in all calves which are born in one year.
- Approximately 14th day of life, addition to drink fluid or oral administration of 5g (Thiabendazole, piperazine citrate)/10 kg body weight, one dose only.
- Cattle anthelmintics are now available in a number of different forms: Drenches—Pour-ons —Boluses —Injectables.

Calves after turnout

Where calves are grazed independently of dams infection levels are liable to multiply to levels producing a reduction in weight gain and/or clinical symptoms.

If clean grazing is not available calves should be wormed regularly after turnout to suppress egg output in the early part of the season and limit the dangerous build up of infective larvae on the pasture in the second half of the grazing season.

Products with no persistent activity should be used every 3 weeks until the over-wintering infection has declined. Those with persistent activity can be used e.g. 3, 8 and 13 weeks after turnout or longer depending on the product used

To succeed and be cost-effective control strategies must be applied to set stocked systems (i.e. where the animals stay on the same field) otherwise the benefits are lost.

Treat all animals, as untreated animals would contaminate the pasture. With these systems, a considerable reduction in the numbers of infective larvae on the herbage in the second half of the grazing season will be obtained.

Calves at housing

Some larvae will always be present on pasture, so treat calves with an anthelmintic effective against arrested worms at housing, especially if taking place late in the season.

After de-worming the calves, their place in the house or the calf pen is thoroughly cleared of litter and dirt and then washed using brush.

Part 2: The Most Important Disease of 5th–12th Week of Life

Nutrition-Induced diarrhea

Calves often suffer from diarrhea which is not caused by an infection but is due to incorrect feeding where calves should be fed with great care where at the time of birth the first three stomachs of the sucking calf are not yet able to function and they develop in the course of the next few weeks through mechanical and chemical stimulation, for this reason colostrums and the right milk substitute are the only appropriate feed for calves during the initial period. Thus at the age of 1 to 3 weeks, feed like maize silage, dairy cattle fodder, whole meal or green forage cannot yet be given to calves where such fodder has a determinate effect on coagulation by chymosin and the change in acidity causes a shift of intestinal flora in favor of proteolytic bacteria, where digestive disorder caused by putrefaction while if saccharolytic bacteria predominate, we refer to a digestive disorder produced by fermentation

Causes

Error in the feeding methods may also cause digestive disorders as the following;

Errors in nature and composition of feed.

- Milk substitute not appropriate to age (e.g. Fat content too high).
- Feed deteriorated due to inappropriate storage, (e.g. rancid fat, moldy calf starter feed).
- Inappropriate feed because of wrong components. (unsuitable fats).
- Feed not appropriate to age (e.g. maize silage, dairy cattle fodder).

Error in preparing the feed.

- Incorrect stirring. Lumps in drink.
- Wrong temperature of drink above 39°C or below 35°C.
- Sudden change-over from milk to milk substitute.

Clinical signs;



Figure 13. Showing signs of diarrhea in calves.

- Diarrhea in the form of fluid or pappy, yellowish to grey, malodorous feces
- Fermentative diarrhea is characterized by bubbly, fluid or pappy, yellowish brown to black brown feces of acid smell.
- The animal shows deep-set eyes, reduced elasticity of the skin and dullness (figure 13).

Diagnosis

- **Clinical signs and case history;**
- **Differential diagnosis;** the difference between nutritional diarrhea and bacterial infection is then no longer clearly discernible. Other causes can be excluded by appropriate tests.

Treatment

General treatment protocol for calves with diarrhea illustrated in diagram (2), and then attention to deal with each case according to causes.



Diagram 2. Showing general protocol treatment with calf diarrhea.

- Immediately** ➤ The first step in the treatment is to eliminate errors in feeding.
 - Observe calf licking of wall.
 - Eating from trough of older neighboring animal (silage).
- 1st- 2nd day** ➤ Replacement of milk substitute or milk by electrolyte solution. 90 g electrolytes salts+15g (chlortetracycline, sulfadimidine, vitamins) in 2 liters of water twice daily.
 - Electrolyte solutions used for oral rehydration of calves suffering from diarrhea. Sodium chloride (kitchen salt), Sodium bicarbonate, Potassium chloride, Potassium dihydrophosphate, Sodium lactate, Potassium citrate, Glucose).
 - N.B- Glucose-Glycine electrolyte solution; 64 grams of this mixture should be dissolved in two liters of

warm water and given in one feeding.

In the third day 1 liter of milk substitute with 1 liter of electrolyte solution [45g electrolytes salts + 15g (chlortetracycline, sulfadimidine, vitamins)] in liter of water, twice daily.

- 4th- 10 day** ➤ Milk or milk substitute with 10 g (chlortetracycline, sulfadimidine, vitamins) twice daily.
- If diarrhea persists and signs of dehydration worsen and the calf shows severe signs of dehydration administer electrolytes and antibiotics intravenously. Dehydrated calves, even those near deaths, usually respond very well to electrolytes administered intravenously.

Hypomagnesemic Calve Tetany (Whole Milk Tetany)

Calves fed exclusively on milk are subject to hypomagnesaemic tetany, the tendency increasing as the calf grows older. This appears to be due to the fact that the level of magnesium resorption in the digestive tract diminishes with the age of the calf. In addition, the young animal possesses in its bone system a mobilizable reserve of magnesium which again tends to diminish with age. As a result the adult animal has no large reserve of mobilizable magnesium in its bones and this renders it more susceptible to hypomagnesaemia. It is similar to that of adult cows.

Causes

Grass tetany may not always arise from a simple deficiency of magnesium (Mg). The disorder can be quite complex and different circumstances can lead to a reduction in magnesium concentration in the blood and cerebrospinal fluid, producing signs of grass tetany as follows:

- Simple form – a deficiency of magnesium.
- Complex form – potassium is the most important factor which interferes with magnesium absorption from the rumen.
- The disorder can be quite complex, with many factors contributing:

- The age of animal – older cows with young calves are most vulnerable;
- Feeding on grass–dominant pastures or young cereal crops;
- High potassium soils or soils treated with inappropriate levels of potassium fertilizers;
- Environmental effects such as: wind, rain and exposure sudden lowering of temperature.

Clinical signs;



Figure 14. Deviated head in hypomagnesmic calve with signs of convulsion in calves.

The calves appear initially to be abnormally nervous with glassy eyes. This is followed by a period of hypersensitivity: the animal lays back its ears, clenches its jaws and nervously thrashes its tail; its appetite is poor. Convulsions generally appear only a few days after these initial symptoms and these as in the case of cows suffering from hypomagnesemia can be triggered off by noise or any external disturbance, generally suffer from marked muscular weakness and will be subject to one or more subsequent attacks to which it will eventually succumb. First signs are contraction of ear movements, distress movement and convulsion, ataxia, deviation of head. Muscles tremors especially with kinking abdomen, foot pushing, jaw movement and frothiness and cyanotic mucous membrane and death (figure, 14).

Treatment

A veterinarian will usually inject calcium and magnesium intravenously (i.e. into the vein) followed by a subcutaneous (i.e. under the skin) injection of magnesium. Farmers must be not disturbing the animal until you are ready to start the treatment.

Generally ➤ Calcium and magnesium solutions, that commercially available are injected intravenously (Ca –Barogluconate 25%, Magnesium lactate 3.3% or Magnesium gluconic 15% (50 ml by slow

intravenous route I/V).

- Followed by 60 to 200 ml of magnesium sulfate 50% injected subcutaneously in neck region for 5 days increased the level of magnesium in the blood within 15 minute.

Note

- Injection intravenous must be very slowly and avoid heart and respiratory abnormalities.
- Animals should not be moved during treatment, but once they have responded to treatment it is best to move them off the pasture. In some cases, repeat treatment may be needed.

Prevention and Control

- Magnesium should be given as feed additives;
- Daily oral supplements of magnesium oxide 60 gm to cattle and magnesium oxide 7–8 % mixed with molls or water and sprayed over hay and dry food.
- Let postures to complete its growth and decrease its potassium level supply.
- Adequate soil phosphorus decreases the grass tetany.
- Feeding free–choice mineral that supplies 13–15 gm of magnesium/head/day for 30 day and avoids stress factors.

Infectious Bronchopneumonia in Calves (Enzootic Pneumonia of calves)

The general term infectious bronchopneumonia (Enzootic Pneumonia) covers disease of the respiratory organs which have different causes, but show similar symptoms. Enzootic pneumonia of calves refers to infectious respiratory disease in calves. The term “viral pneumonia of calves” is sometimes used but is not preferred based on the current understanding of etiology and pathogenesis. Enzootic pneumonia is primarily a problem in calves <6 month old with peak occurrence from 2–10 wk, but it may be seen in calves up to 1 yr of age. It is more common in dairy than in beef calves and is a common problem in veal calves. It is also more common in housed dairy calves than in those raised outside in hutches. Peak incidence of disease may coincide with decline of passively acquired immunity. Morbidity rates may approach 100%; case fatality rates vary but can reach 20%. For an outbreak of the disease, stress factors

need to be present which reduce the resistance of the calves, enabling viruses and at a later stage, bacteria to multiply in animal organism.

Incidence

As a result of enlarged animal stocks, high density housing and intensive feeding, infectious bronchopneumonia in calves has appeared as a new disease complex. It has long been known and used to be regarded as harmless. However, in today intensive livestock farms purchasing animals and having as a result a large number of animals of the same age are particularly affected.

In the 4th to the 6th week of life, the calf has only a limited resistance to infection, which it originally received via the cloistral milk. This immunity acquired via the cloistral milk (passive immunity), steady decrease and has lost its effect by the age of 5 weeks. However; at that point the animal organism has not been sufficiently exposed to pathogens. Its active immunity is still in the process of being built up. At the age of 4 to 6 weeks the calf is particularly susceptible because its passive immunity is disappearing and its active immunity is as yet insufficient.

It is usually at this stage in their lives that calves are marketed and housed on calf or bull calf fattening farms. Marketing, subsequent transport and re-housing cause much additional stress to the calf and contribute to the increased appearance of infectious bronchopneumonia during this period.

Stress results from environmental and management factors, including inadequate ventilation, mixing by adding calves to an established group, crowding, and nutritional factors such as poor-quality milk replacers. Partial or complete failure of passive transfer of maternal antibodies is an important host factor related to development of disease. Changes in the environment, such as re-housing, change-over from straw litter to slatted floor must be regarded as precipitating factors.

Clinical signs

- Several days later a second viral attack produces a further increase in body temperature. The calves show losses of appetite, listlessness and aqueous or mucoid discharge from the eyes and the nose.
- Unless treatment is started at this stage, bacteria will aggravate the condition.

- Affected stand or lie listlessly in the pen. They refuse to feed, splay their forefeet and are audibly fighting for breath.
- Death occurs in many cases.
- The characteristic course with two attacks of fever and the outwardly visible signs of a respiratory disease point to the presence of infectious bronchopneumonia. It is particularly the discharge from the eyes and the nose and the high body temperature that indicate an infection with viruses and bacteria.

Diagnosis

Clinical signs and case history

- The demonstration of the viruses involved serves useful purpose only on farms where this condition occurs regularly where the virus can be demonstrated in the living animals at the start of the disease by taken swabs (nose, throat).
- Bacteriological examination is more important than the demonstration of the virus by taking swabs from the living animals and examining the organs of dead animals.
- The disease must be differentiated from a pneumococcal infection where the calves contract a pneumococcal infection mainly at the age of 3 weeks also takes such a rapid course that a discharge from the eyes is not normally seen.

Treatment

- The success of treatment greatly depends on the stage in which the disease is recognized and how quickly treatment is initiated.
- Antibiotic treatment has no effect on viruses, but kills bacteria (secondary infection) or inhibited their multiplication.

1st day *Severely affected calves*

- Long-acting antimicrobial drug as (cholormphenicol, enrofloxacin). Injection; 1.5ml/10kg body weight repeated each 12 hours for 3–5 day.
- Nonsteroidal anti-inflammatory drug (NSAID) has been shown to be a beneficial ancillary therapy in controlling fever
- Biosolvon; 10 ml/calf.
- Vitamin AD3E; 20/calf.

- Organophosphorus compound and vitamin B12; 10 ml/calf.
- 2nd day** Antibiotic dose + Biosolvon; 10ml/calf +Antihistaminic drug;5ml/calf
- 3rd day** Antibiotic dose + Biosolvon; 10ml/calf
- 1st–**
- 21 days**
 - Affected animal being feed on; 20g (chlortetracycline + sulfadimidine + vitamin AD3E)/day for 7 day.
 - Calves with development rumen; 10 g of (chlortetracycline + sulfadimidine +vitamin A and B) /day for 3 weeks.

Shipping Fever Pneumonia(Hemorrhagic septicemia);

Shipping fever pneumonia, or undifferentiated fever, is a respiratory disease of cattle of multifactorial etiology with Mannheimia haemolytica and, less commonly, Pasteurella multocida or Histophilus somni, being the important bacterial agents involved. Shipping fever pneumonia is associated with the assembly into feedlots of large groups of calves from diverse geographic, nutritional, and genetic backgrounds. Morbidity in feeder calves often peaks within 7–10 days after assembly in a feedlot. Morbidity can approach 35%–50%, and case fatality is 5%–10%; however, the level of morbidity and mortality strongly depends on the array of risk factors present in the cattle being fed.

Etiology

The pathogenesis of shipping fever pneumonia involves stress factors, with or without viral infection, interacting to suppress host defense mechanisms, which allows the proliferation of commensally bacteria in the upper respiratory tract. Subsequently, these bacteria colonize the lower respiratory tract and cause a bronchopneumonia with a cranioventral distribution in the lung. Multiple stress factors are believed to contribute to suppression of host defense mechanisms. Weaning is a significant stressor, and the incidence of this disease is highest in recently weaned calves. Transportation over long distances serves as a stressor; it may be associated with exhaustion, starvation, dehydration, chilling and overheating depending on weather conditions, and exposure to vehicle exhaust fumes. Additional stressors include passage through markets; commingling, processing, and surgical procedures on arrival at the

feedlot; dusty environmental conditions; and nutritional stress associated with a change to high–energy rations in the feedlot.

Clinical signs

- Early signs include depression, anorexia and dull eyes. When these symptoms occur, sort the cattle and check for fever, temperatures over 104°F can indicate the onset of BRD. Later symptoms include rapid or labored breathing, droopy ears, coughing, diarrhea, staggering, regular nasal discharge and sudden death.
- Left untreated, calves with severe Bovine respiratory disease (BRD), will die from asphyxiation,” according to the University of Minnesota recommendations.

Treatment

- Immediately**
- Early recognition and treatment of calves with BRD usually improves their outcome and overall performance.
 - Treatment options can vary but most involve the use of antibiotics specifically designed to treat calves with pneumonia. Most veterinarians now use antibiotics which are effective against the bacteria most commonly found in the lung tissue. These new antibiotics are also long acting, can be given under the skin and very effective against BRD.
 - In addition, some producers and veterinarians will also administer anti-inflammatory drugs for fever or vitamins.
 - Response to therapy is usually observed within 24 hours and a successful outcome is closely related to early recognition of BRD clinical signs.

Control and Prevention

Prevention of shipping fever pneumonia should focus on reducing the stressors that contribute to development of the disease.

Immediately

- Cattle should be assembled rapidly into groups and new animals should not be introduced to established groups.

- Mixing of cattle from different sources should be avoided if possible.
- Transport time should be minimized and rest periods with access to feed and water, should be provided during prolonged transport.
- Calves should ideally be weaned 2–3 wk before shipment. Cattle should be processed within 48 hr after arrival at the feedlot.
- Adaptation to high–energy rations should be gradual because acidosis, indigestion and anorexia may inhibit the immune response.
- Vitamin and mineral deficiencies should be corrected.
- Metaphylaxis with long–acting antibiotics such as oxytetracycline, tilmicosin, florfenicol, gamithromycin, tildipirosin, or tulathromycin has been widely adopted as a control measure given “on arrival” to cattle at high risk of developing shipping fever pneumonia.
- Metaphylaxis on arrival has been shown to significantly reduce morbidity and improve rate of gain and in some cases reduce mortality.
- Mass medication in feed or water is of limited value because sick animals do not eat or drink enough to achieve inhibitory blood levels of the antibiotic and many of these oral antibiotics are poorly absorbed in ruminants.

Tympani in calves

Tympani (bloat) is a rapid, excessive distention of the rumen, the dorsal sac or the abomasums. In this condition, for Variety of causes fermentation gases can no longer escape via the esophagus and gullet.

We had known three types of bloat which differ in origin, progress and treatment.

1. Suddenly occur (acute) tympani of the first three stomach compartments.
2. Chronic recurrent tympani of the first three compartments.
3. Tympani of the abomasums.

Suddenly occurring (acute) tympani

Causes

The sudden distension occurs as a result of an obstruction of the gullet by large lumps of fodder (pieces

of corn–cob, potatoes or pieces of turnip), may causes congestion of the gullet.

Clinical Signs;



Figure 15; Calf with bloat indicated ear back and open eyes.

When a lump of fodder becomes logged in the esophagus lead to spasm of the gullet muscles and the foreign body being wedged in even more tightly, since the fermentation gases which are formed can no longer be released by regular eructation and accumulate in the rumen.

- Gas bubble is formed which increase in size very rapidly and visible expands the left side of the body between the last rib and the hip bone.
- On palpation in the region of the rumen, clear sonorous sound can be heard.
- The animal refuse to feed anymore and being salivate profusely (figure 15).

Treatment;

Rapid action is imperative. An injection of anti–spasmolytic drugs (Buscopan) will facilitate the removal of foreign body.

- An attempt should be made either to pull the lump out carefully or to push it down into the body.
- If the distention is excessive, the gas is released by introducing tracer and cannula through the abdominal wall into the rumen (Diagram 3).



Diagram 3. Indicated instrument and drench treatment of calf tympani.

Chronic recurrent tympani

While the animals are being weaned from milk and after the changeover to roughage. They are very susceptible to recurrent bloat, where free gas formed a gas bubble and foamy fermentation is rarely observed. Tympani with the formation of gas bubble may be due to one of the following causes.

1. *Flora of the rumen not functioning*

For some calves the change-over from milk to roughage is carried out too quickly, where the time when milk feeding is terminated depends not only on age but also on the development of the stomach compartments.

2. *Chronic gastrointestinal catarrhal inflammation*

An inflammation of the mucosa of the first three stomach compartments and of the abomasums is caused by drinking cold or too hot fluid at irregular feeding time and by varying quantities. This disturbs the rhythm of rumination, since the movement of the three stomach compartments slow down or stop completely (atony of the rumen).

3. *Nerve damage.*

Late sequel of infectious bronchopneumonia where swelling and inflammation of lymph nodes occur in infectious bronchopneumonia lead to damage nerve responsible for the digestive movement lead to atony of the first three stomach compartments.

4. *Undigestible foreign body intake with feedstuffs*

5. *Inappropriate feedstuffs*

This includes the feeding of green forage while the calves still receive milk. Tympani caused by foamy fermentation that may occur due to the following causes.

- Young green forage which is deficient in crude fiber and rich in protein.
- If the crude fiber content is low, bacteria form enzymes (mucinases) which render the foam-suppressing substances of the saliva (mucins) ineffective in tympani caused by the formation of a bubble of free gas

Treatment

- **Acute bloat must be treated promptly by trocar:** In the last stages of severe bloat a few seconds delay may result in the animal's death. Equipment needed includes good handling facilities,

a stomach tube or rubber hose about 3/4 to 1 inch in diameter and 8 to 10 feet long, a supply of defoaming agent, and a large tracer. If the tracer fails to relieve the bloat, you will also need a sharp knife suitable for incising the skin and making an opening into the rumen.

- **The antifoaming agent:** The agent can be added through the tube or through a tracer and bloat needle. –Antifoaming agents would include emulsified oil (mineral oil) or oil containing an approved detergent such as dioctyl sodium sulfosuccinate.

Large bloat needles may be adequate for relieving feedlot bloat: They are about 6 to 7 inches long and come with a wire styled to unplug them, if necessary. Insert the needle at a point halfway between the last rib and hook-bone on the left side.

Choking on foreign objects (esophageal obstruction): This should be relieved with a tracer or big needle, if possible followed by gentle removal of the obstruction from the esophagus by a veterinarian surgeon.

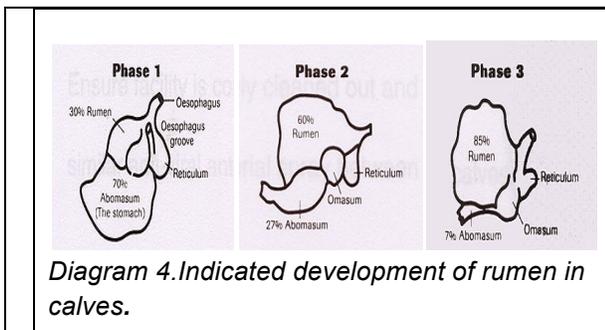
Chronic bloat: Chronic bloat caused by pressure on the esophagus due to muscle paralysis or other tissue pressure on the esophagus can be corrected by making a ruminal fistula. Generally, these openings are about 3/4 inch in diameter.

Prevention of recurrent tympany in calf

Let calves fast for one day but water must be freely available. Be careful with maize silage and continue feeding the calves with milk in the early stages of weaning.

- Give 2 tablespoonful of (Anti-bloat drug) three times daily.
- Transfer fresh rumen juice where warmed to body temperature.

Tympani of the abomasums ;



In sucking and fattening calves tympani of the abomasums is observed in isolated cases. With tympani of the abomasums, as distinct from bloat of the first three stomach compartments, protrusion of the abdominal wall occurs on the right-hand side in the lower part of the costal arch.

Causes

During the first few weeks of life, the abomasums cannot hold more than 4 liters / feed where the rumen is still undeveloped in newborn calf, where it to develop pass by many stages as illustrated in diagram 4).

- Bloat is caused in particularly by the ingestion of large volume of milk.
- Ulcers and inflammation of the abomasums occur if the drinking fluid taken up by the animal is too hot or too cold.
- Hair ball may obstruct the pylorus. The contents of the abomasums can then only partly pass into the intestine or not at all. The abomasums become congested and tympani results. With hasty eating, great deal of hair is swallowed at the same time, resulting in impairment of enzyme action on the multiplication of bacteria.
- Non-ruminant (birth to 21 days). The Abomasums comprises 70% of the total stomach. The rumen is underdeveloped and not functioning.
- 2- Dry feed intake in 22 to 84-day-old calves stimulates rumen growth and micro-organisms, which produce volatile fatty acids. These acids stimulate growth of rumen tissue.
- 3- At more than 84 days old the calf is considered a ruminant.

Clinical signs

- The calves show signs of sever colic.

- Arched back and kicking out with the legs are observed.
- Faces and urine are passed frequently and in small quantities.

Diagnosis

- On palpation of the abdominal wall in the region of the left flank a clear.
- This type of tympani cannot be remedied by the introduction of a tube through the esophagus.

Treatment

- Analgesic drug (Novalgen, Buscopan), where smooth muscles spasm are relived within a short period, enabling the gas to be released by eruct ion.
- In persistent cases puncture of the abomasums is necessary by veterinary surgeon.

Cerebrocortical necrosis (Polioencephalomalacia)

Cerebrocortical necrosis is a non-infectious metabolic disorder change in cerebral cortex which is caused by vitamin B1 deficiency and lead to disorder of the central nervous system, that characterized by softening of grey matter, and contributes to substantial economic loss to livestock industry. Animals of all ages can be affected but young animals appear to be more vulnerable. Several risk factors such as thiamine deficiency, Sulfur toxicity, lead toxicity, and water deprivation-sodium ion toxicity have been implicated in the development of Polioencephalomalacia (PEM). All these factors produce similar brain lesions. Regardless of the suspected cause of PEM, affected animals frequently respond to thiamine administration.

Incidence and pathogenesis

The disease is seen sporadically or as a herd outbreak. In general, younger animals are more frequently affected than adults. Animals on high-concentrate diets are at higher risk but pastured animals also develop cerebrocortical necrosis.

Polioencephalomalacia (PEM) has been associated with 2 types of dietary risks: altered thiamine status and high sulfur intake. Thiamine inadequacy in animals with PEM has been suggested by several types of observations, including decreased concentrations of thiamine in tissues or blood and deficiency-induced alterations of thiamine-dependent biochemical processes (decreased blood transketolase activity, increased thiamine pyrophosphate

effect on transketolase, and increased serum lactate). Unfortunately many of these biochemical features of altered thiamine status are inconsistently observed in cases of PEM, and decreased thiamine status has been observed in diseases other than PEM.

In adult ruminants, thiamine is produced by rumen microbes. Preruminant animals depend on dietary thiamine. Thiamine inadequacy can be caused by decreased production by rumen microbes or factors that interfere with the action of thiamine, e.g., plant thiaminases or thiamine analogs. Thiaminases can be produced by gut bacteria or ingested as preformed plant products. They can either destroy thiamine or form ant metabolites that interfere with thiamine function.

Up to the age of 10 weeks (100 kg body weight) calves needs 5mg vitamin B1 daily while in older animals this requirements rises to 5–10 mg /100kg body weight. Vitamin B1 is an essential biological substance in carbohydrate metabolism and has an important function in conduction in the nervous system, where central nervous system can function only if sufficient glucose and vitamin B1 as intermediary carrier (co–enzyme) is available.

Etiology;

1–Thiamine deficiency induced (PEM);

Thiamine deficiency induced PEM has been reported in cattle. Thiamine deficiency in ruminants has been associated with several factors such as an impairment of microbial thiamine synthesis, thiamine destroying activity of bacterial thiamin's, along with other dietary factors involved in thiamine destroying activity in the rumen. Bacterial thiamin's has been considered the main factor leading to thiamine deficiency in ruminants. Two types of thiamin's (Type I and II) are produced by different types of ruminant bacteria. Both types have a destructive effect on thiamine in the rumen. Thiamin's type I catalyze the nucleophilic displacement of the thiazole moiety of thiamine by another base known as a co–substrate and generate thiamine analogues that inhibit thiamine dependent reactions. Thiamin's type I requires ago–factor to accomplish its thiamine destroying activity.

Some medications such as promazines and levamisole along with substrates produced during fermentation appear to be act as cofactor to thiamin's

type I. Thiamin's type I is also present in plants such as bracken fern, horsetail and nard ferns. Animals exposed to these plants have subsequently developed PEM. Thiamin's type II splits thiamine by catalyzing the hydrolysis process and thereby may reduce the amount of thiamine absorbed from rumen. Several outbreaks of PEM in sheep and cattle with high thiamin's activity in the rumen have been reported.

Amprolium, a potent coccidiostat and thiamine analogue is believed to be another major factor associated with PEM. It inhibits the conversion of free–base thiamine to TPP, thereby depriving tissues (especially brain).

2–Sulfur–induced PEM; Sulfur toxicity has become increasingly accepted as a major cause of PEM and there are numerous reports regarding dietary S levels arranging from 0.45% to 0.6% on dry matter (DM) basis that caused clinical and experimental PEM. The hypothesis regarding high dietary associated sulfur.

Vitamin B1 deficiency which finally lead to cerebrocortical necrosis may be due to the following causes;

1. Feed does not contain sufficient vitamin B1
2. Mould fungi and bacteria in the feed form enzymes (Thiamine's) which destroy vitamin B1.
3. An abrupt change in feed disturbs the bacterial flora in the rumen, which then produces too little vitamin B1.
4. Sudden changes in temperature in the cattle house raise the vitamin B1 requirement considerably (additional requirement of up to 20%). A change to slatted floors, for instance may induce vitamin B1 deficiency.

Clinical Signs ;



Figure16; Animals press head into corners. Calves are blind and may show "star-gazing" behavior and calve lies on his side with convulsion (blindness)

PEM may be acute or sub acute, animals with acute form manifest blindness, recumbence; tonic–colonic seizures

and coma. Those with a longer duration of acute signs have poorer responses to therapy and higher mortality.

- Animals with the sub acute form initially separate from the group, stop eating, and display twitches of the ears and face.
- The head is held in an elevated position and there is a staggering, sometimes hypermetric gait, as the disease progresses, there is cortical blindness with a diminished menace response and unaltered palpebral and pupillary responses.
- Head pressing, opisthotonos, and grinding of the teeth may be observed.
- In the terminal stages, the calves lie down on the side with their legs stretched out and performing rowing movements and if treatment is not carried out in time, the animal die within 2 or 3 days (figure 16).

Diagnosis;

1. Accurate clinical signs and case history.
- 2, Differential diagnosis; The disease should be differentiated from acute lead poisoning, hypomagnesaemia, and vitamin A deficiency, infectious meningoencephalitis (ITEME) among other diseases of cattle with clinical findings referable to brain dysfunction or malfunction.

If calves and young cattle show central nervous signs disorder, vitamin B1 deficiency and cerebrocortical necrosis should be suspected.

3, Diagnostic treatment;

As an immediate measure, vitamin B1 should always be injected in high dose and any improvement seen after the injection will indicated the presence of vitamin B1 deficiency.

4, Laboratory examination; In the living animal the diagnosis is confirmed by determining the blood levels of pyruvic acid, lactic acid and glucose, as in table 3.

Blood Level.	Lactic Acid	Pyruvic Acid	Glucose
Animals.			
Healthy animal.	12mg/100ml	0.6–1mg/100ml	50mg/100ml
Diseased animal.	240mg/100ml	5.5mg/100ml	>200mg/100ml

Table 3; indicated the blood levels of lactic acid, pyruvic acid and glucose in healthy and diseased calf.

Differential diagnosis

Disease associated with central nervous symptoms should be differentiated from cerebrocortical necrosis. Such disease includes rabies, listeriosis, vitamin E and selenium deficiency and ISTME (hypersomania), where the most important characteristic of this disease are as the following.

Rabies	Profuse salivation, bloat, sudden collapse
Listeriosis	Raised body temperature and difficulty in breathing.
Vitamin E/Selenium deficiency	Pale appearance of mucosa, muscular weakness, hematuria
ISTME (hypersomnia)	Raised body temperature, no response to vitamin B1 injection

Treatment and prevention

Treatment should begin immediately after the onset of signs. Treatment of choice is thiamin hydrochloride at 10 mg/kg body weight given intravenously initially and followed by similar doses every three hours for a total of five treatments. Those animals that are clinically subpar and are still anorexic by the third day after the initiation of treatment will most likely not respond to treatment and should be put down. For the rest of the cattle, a dietary change and supplementation of thiamin at 1 gm/head/day for about 2 to 3 weeks are the preferred prophylaxis.

- 1st–3rd day**
 - The dosage of thiamine is 10–20, mg/kg, IM or SC,. Initial treatment may be administered I/V, 2–3 times daily for three days. I/M .
 - Reduction of cerebral edema can be attempted with administration of dexamethasone at a dosage of 1–2 mg/kg, IM or SC.
 - Symptomatic therapy for convulsions may be necessary.
- 1st–21st day**
 - Affected calve and calve of the same age; 10g (cholortetracycline + sulfadimidine +vitamin A+B1) and 1.5–2kg of good quality hay per day

Dietary supplementation of thiamine at 3–10 mg/kg feed has been recommended for prevention, but the efficacy of this approach has not been carefully evaluated. During a PEM outbreak, sufficient roughage should be provided.

When the problem could be associated with high sulfur intake, all possible sources of sulfur, including water, should be analyzed and the total sulfur concentration of the consumed dry matter estimated and dietary ingredients or water with high sulfur concentration should be avoided; if this is not possible,

then more gradual introduction to the new conditions can improve the chances of successful adaptation.

Vitamin E/Selenium deficiency (white muscle disease)

White muscle disease is also known as nutritional myopathy of calves. It is normally seen in young calves and is associated with deficiencies of selenium or vitamin E, or both. There are two forms of white muscle disease; a congenital form that affects the cardiac muscle, and a delayed form that is associated with either cardiac or skeletal muscle. Vitamin E has a capacity to protect feedstuffs with a high content of unsaturated fatty acids from being destroyed by oxidation. In this way the vitamin E contained in the fodder is used up and is no longer available to the animal.

- There is a close correlation between vitamin E and the trace element, selenium where the two substances have a mutual 'saving' effect but cannot replace each other.
- Vitamin E deficiency in the feed ration can cause changes in the cardiac and skeletal muscles which that are known as white muscle disease or white meat disease.

Incidence

If deteriorated milk substitutes (inadequate or excessively long storage) are fed during calf fattening, white muscle disease is seen with increasing frequency.

- If the calf is fed by its mother or another lactating cow. This condition will occur only if the vitamin E and selenium supply of the cows is grossly inadequate.
- The vitamin E content of all feedstuffs steadily decreases with the storage.

Causes

White muscle disease is a myopathy resulting from low levels of dietary selenium and vitamin E. Feeds grown in areas where the soil is deficient in selenium results in decreased uptake by the plant thus making the feed selenium-deficient. Vitamin E deficiency can be caused by large amounts of unsaturated fatty acids and other peroxide-forming substances in the diet. Another mechanism for selenium deficiency in cattle is the result of antagonistic effects of certain metals such as silver, copper, cobalt and mercury. The daily requirement of the

calf is 20mg vitamin E and 0.25mg selenium. If milk substitutes are fed which contain too many unsaturated fatty acids in large quantities? If milk substitute are stored for too long or incorrectly, where the fats decay and peroxides are formed lead to vitamin E deficiency.

Clinical signs;



Figure.17. stiff gait, arched back calf and lies down on the chest calf.

Calves affected by the congenital form of white muscle disease usually die within 2–3 days of birth due to cardiac muscle degeneration.

- Calf is affected severely it may die of starvation due to an inability to nurse properly due to weakness.
- Cattle affected by the delayed form of white muscle disease may exhibit signs ranging from general unthrift and stiffness to walking with an arched back and spending more time recumbent depending on the level of selenium in the diet.
- Growing calves may also develop a Heinz-body anemia. It has been shown that there is an increased susceptibility to infectious diseases during vitamin E/selenium deficiency.
- The animal may pass bright red to dark brown urine and the deficiency manifests itself particularly in muscular weakness.
- Stiff gait and arched back are usually the first signs of muscular changes in this condition, the animals frequently lie down and have an unsteady gait. With severe vitamin E/selenium deficiency the animal lie down permanently on the chest or side and show nervous disorder (figure 17).
- Clinical signs of low selenium/vitamin E status in adult cows are more difficult to determine. Adult cows with a selenium/vitamin E deficiency may be more likely to retain the placenta, have an abnormal

calving and are more susceptible to metritis and cystic ovaries.

Diagnosis

- Accurate clinical signs and case history;
- Laboratory examination;
 - Evaluation of serum level of vitamin E.

Normal animals. 0.15mg/100ml serum

Affected animals. 0.04mg/100 ml serum

- Necropsy examination

On examination post death, the heart will show white, chalky plaques that are most noticeable in the left ventricle.

The muscles of animals that have died or have been slaughtered show dark red patches with grayish white foci (necrotic foci), the carcass meat of such animals has a light pale color and classified as a low quality meat.

Treatment

- Cattle affected by white muscle disease have been treated with sodium selenite and vitamin E in sterile emulsion. This can be administered SC or IM, at 1 mg selenium and 50 mg (68 IU) of vitamin E per 18 kg (40 lb) body wt.
- If necessary, the treatment may be repeated two weeks later, but no more than four doses total should be given. In calves affected with simple vitamin E deficiency, treatment with dietary supplementation using α-tocopherol or substances rich in vitamin E can be used. Calves have been cured using 600–mg of alpha-tocopherol initially followed by daily doses of 200–mg. Any polyunsaturated fats should be removed from the diet as these may be causing the vitamin E deficiency.

1st to Immediately

- 3rd day
- Treatment should begin immediately after the onset of signs where treatment of choice is thiamin hydrochloride at 10 mg/kg body weight given intravenous initially and followed by similar doses every three hours for a total of five treatments.
 - Immediate treatment of the affected animals; 5ml vitamin E/Selenium per day/ intramuscular.

1st to 10 days

- –For the rest of the cattle, a dietary change and supplementation of thiamin at 1 gm/head/day for about 2 to 3 weeks are the preferred prophylaxis.
- 10 gm multivitamin/ calf/day to be stirred into the drinking fluid or admixed to the fodder.

Prevention

- To prevent white muscle disease within four weeks after birth, cows are given 15 mg of selenium, usually as sodium selenite four weeks before calving.
- To prevent the delayed type, calves are given 5 mg of selenium at two to four weeks of age and twice more at monthly intervals. A selenium and vitamin E mixture is advocated in some areas. Other procedures for selenium supplementation include administration of intraruminal selenium pellets, use of selenium–fortified salt or mineral mixtures; Adding selenium to feed for breeding animals or their young is useful in areas of known deficiency. The recommended supplemental level is 0.3 ppm selenium, calculated on the basis of total dry–matter intake. It is added as sodium selenite which contains 45.65% selenium. Because of the minute quantities involved and the toxicity of excess intake, premixing and thorough subsequent mixing is necessary.

Coccidiosis

Coccidiosis is commonly a disease of young cattle (1–2 mo to 1 yr) and usually is sporadic during the wet seasons of the year. “Summer coccidiosis” and “winter coccidiosis” in range cattle probably result from severe weather stress and crowding around a limited water source, which concentrates the hosts and parasites within a restricted area. Although particularly severe epidemics have been reported in feedlot cattle during extremely cold weather, cattle confined to feedlots are susceptible to coccidiosis throughout the year.

Outbreaks usually occur within the first month of confinement. Cows may contribute to environmental contamination of *E. bovis* oocysts through a pre parturient increase in fecal oocyst counts. Clinical disease due to coccidiosis does not typically occur in the first 3 wk of life. Coccidiosis is therefore not considered part of the neonatal diarrhea complex in calves.

Causes

Coccidiosis is caused by infection by protozoan parasites called *Eimeria spp.* which parasitize the lining of the

intestinal tract *E. bovis* is the most common and pathogenic. Infection causes a loss of absorptive capacity of the gut with consequent diarrhea and possibly dysentery. Outbreaks of disease are commonly seen 3–4 weeks after mixing groups of dairy calves.

Rout of infection

Infected animals pass coccidial oocysts in the faces. These oocysts mature the animal body into sporulated oocysts to reach maturity stage outside the body where the oocysts require moisture, oxygen and warmth. Mature oocysts are taken up in the fodder or drinking water and pass into the intestine where lost their outer shell and the sporozoities attack the intestinal mucosa and multiply in the intestinal mucosa cells by asexually and sexually multiplication.

N.B

Oocytes may remain infective for more than a year.

Environmental condition affected on the oocyst activity where moist areas in cattle houses and pastures offer favorable condition to development and survival of oocysts, while dry conditions and extremes of temperature (above 40c and below –7c) will quickly kill them off.

Clinical signs;



Figure 18. Straining with passage of only mucus and blood in calf.

The most characteristic sign of clinical coccidiosis is watery feces with little or no blood; animals show only slight discomfort for a few days.

- Severely affected calves develop thin bloody diarrhea that may continue for >1 wk or thin feces with streaks or clots of blood, shreds of epithelium and mucus but severe infections are rare. They may develop a fever, become anorectic, depressed, dehydrated and lose weight but tenesmus is common

because the most severe enteritis is confined to the large intestine (figure 18),

- Nervous signs (e.g, muscular tremors, hyperesthesia, clonic–tonic convulsions with ventroflexion of the head and neck, nystagmus) and a high mortality rate (80–90%) are seen in some calves with acute clinical coccidiosis and other calves die from secondary complications. Affected calves may die <24 hr after the onset of dysentery and nervous signs or they may live for several days.

Diagnosis

- Accurate clinical signs and case history
- Parasitological examination of the faces will show coccidial oocyst are present.
- Differential diagnosis; the diarrhea must be differentiated from that seen in Salmonellosis, poisoning, inappropriate diet and worminfestation by bacteriological and parasitological examination.

Treatment

The ideal coccidiostat suppresses the full development of the life cycle of the coccidia, allows immunity to develop and does not interfere with production performance.

N.B

Coccidiosis is a self-limiting disease and spontaneous recovery without specific treatment is common when the multiplication stage of the coccidia has passed.

Immediately

- Clinically affected animals should be isolated and given supportive oral and fluid therapy as necessary in addition together with sulfonamide therapy.
- Sulfonamide therapy may be indicated to control the development of secondary bacterial enteritis or pneumonia, which may develop in calves with coccidiosis during very cold weather. Sulfonamides in the feed at 25–35 mg/kg for ≥15 days are effective for the control of coccidiosis in calves.
- Amprolium (10 mg/kg) daily for 5 days, Sulfaquinoxaline (2.7 mg/kg) daily for 3–5 days. Where Sulfaquinoxaline is particularly useful for weaned calves that develop bloody diarrhea after arrival at a feedlot. While For prevention, amprolium is a good choice drug (5 mg/kg/day for 21 days).
- Monensin is an effective coccidiostat and growth promoting in calves. Post weaning coccidiosis in

beef calves has been controlled using monensin administered via intraruminal devices.

- Corticosteroids are contraindicated because they increase shedding of oocysts and have induced clinical disease in subclinical infected calves.
- Hygienic measures are taken (moving animals to another grazing ground, keeping feeding, watering point dry and changing them repeatedly, dry litter and keeping water troughs clean).

Rickets

Rickets is an inflammatory affection of young, growing bones, and mostly involves the ribs and long bones of the legs. It consists in a failure of the organism to deposit lime salts in bone, and for this reason the bones do not ossify so rapidly as they should. The cartilaginous ends of the bones grow rapidly, but ossification does not keep pace with it. The bones become long and their ends bend at the joints, the legs become crooked, and the joints are large and irregular. All the bones affected with this disease are thicker than normal, and the gait of the animal is stiff and painful. A row of bony enlargements may be found where the ribs articulate with the cartilages connecting. A calf is less frequently affected with rickets than dogs and pigs.

Causes

The most common causes are dietary insufficiencies of phosphorus or vitamin D. Calcium deficiencies can also cause rickets, and while this rarely occurs naturally, poorly balanced diets deficient in calcium have been said to cause the disease. As in most diets causing osteodystrophies, the abnormal calcium: phosphorus ratio is most likely the cause.

Clinical signs;



Figure.19. Rickets in a dairy calf.

The characteristic lesions of rickets are failure of both vascular invasion and mineralization in the area of provisional calcification of the physis. This pathology is most obvious in the metaphyses of the long bones.

- There may be a wide variety of clinical signs, including bone pain, stiff gait, swelling in the area of the metaphyses, difficulty in rising, bowed limbs, and pathologic fractures may occur (figure 19).
- On radiographic examination, the width of the physes is increased, the non-mineralized physeal area is distorted and the bone may show decreased radiopacity.
- In advanced cases, angular limb deformity can be seen due to asynchronous bone growth.

Diagnosis

- Accurate clinical signs and case history.
- Laboratory examination;

Plasma alkaline phosphates activity is commonly increased.

Concentrations of serum phosphorus and vitamin D may be altered depending on the primary cause of rickets.

In cases associated with phosphorus or vitamin D deficiencies, concentrations of these compounds in serum are subnormal.

Hypocalcaemia is seen in advanced stages.

Treatment;

Immediately

- Correction of the diet is the primary treatment for rickets. The prognosis is good in the absence of pathologic fractures or irreversible damage to the physes. If the animals are housed, exposure to sunlight (ultraviolet radiation) will also increase production of vitamin D₃ precursors.
- The affected animal should have nourishing feed containing a proper quantity of lime salts. Outdoor exercise and plenty of fresh air are indispensable. Limewater should be given once daily for drinking purposes and ground bone meal mixed with the food.
- Calves must be immediately injected with source for vitamin D (AD3E) for 3–5 days.
- 1 gm calcium phosphate given twice daily for 2-month old calf and proportionally increased for older animals that have proved efficacious in this disease.
- In some cases the long bones of the limbs are too weak at birth to support the weight of the animal and temporary splints, carefully padded and wrapped on with some soft bandages become necessary.



- If the animals are housed exposure to sunlight (ultraviolet radiation) will also increase production of vitamin D₃ precursors.
- The prognosis is good in the absence of pathologic fractures or irreversible damage to the physes.

Part.3.The Most Important Disease of Rearing and Adult Cattle

A. Metabolic disease

Indigestion(Simple indigestion)

Simple indigestion is a minor disturbance in ruminant GI function that occurs most commonly in cattle and characterized by failure of normal rumen movement where rumen motility slows but does not stop. Simple indigestion is a diagnosis of exclusion and is typically related to an abrupt change in the quality or quantity of the diet. Since the animals stops eating for one or two days, the rumen reaches a neutral pH level and recovery will follow.

Causes

Almost any dietary factor that can alter the intraruminal environment can cause simple indigestion. The disease is common in hand-fed dairy and beef cattle because of variability in the quality and quantity of their feed. Dairy cattle may suddenly eat excessive quantities of highly palatable feeds such as corn or grass silage; beef cattle may eat excessive quantities of relatively indigestible, poor-quality roughage during winter. During drought, cattle may be forced to eat large quantities of poor-quality straw, bedding, or grain. Simple indigestion can result from suddenly changing the feed, using spoiled or frozen feeds, turning cattle onto a lush cereal grain pasture, or introducing feedlot cattle to a high-level grain ration.

Simple indigestion is usually associated with a sudden change in the pH of the ruminal contents, such as a decrease in ruminal pH due to rapid fermentation of ingested carbohydrates or an increase in ruminal pH due to fore-stomach hypomotility and putrefaction of ingested feed.

1. Any dietary factor that can alter the ruminal environment.

2. Dairy cattle may suddenly eat excessive quantities of highly palatable feeds such as corn or grass silage, Excessive quantities of relatively indigestible, poor-quality roughage during winter.
3. During drought cattle and sheep may be forced to eat large quantities of poor-quality straw.
4. Suddenly changing the feed by using spoiled or frozen feeds, Introduced urea to a ration, oral antibiotic and sulfa, Turning cattle onto lush cereal grain pasture and introducing feedlot cattle to a high level grain ration.
5. Simple indigestion, which is primarily ruminal atone, may follow a sudden change in the pH of the ruminal contents caused by excessive fermentation or putrefaction of ingested feed. The simple accumulation of excessive quantities of relatively indigestible feed may physically impair rumen function for 24–48 hr.

Clinical Signs;

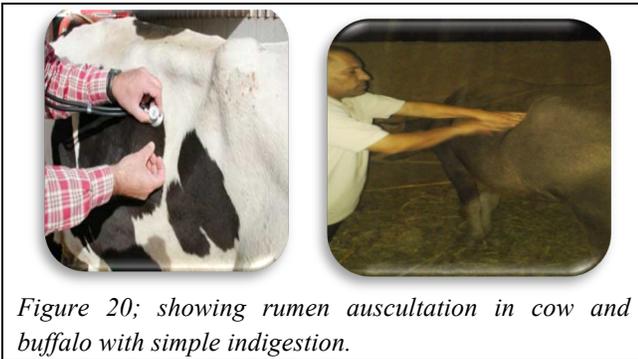


Figure 20; showing rumen auscultation in cow and buffalo with simple indigestion.

The animal will go off food, become slightly dull, depressed, hang the head or separate from the herd. Severe overeating is accompanied by systemic and often fatal acidosis. Rumen motility ceases and the contents become firm; mild bloat or swelling on the left flank, may be present (figure 20).

Constipation may be followed by diarrhea, muscle tremors, groaning, drunken behavior, increased heart and respiratory rates and fever. This is a life-threatening condition. The animals will grind their teeth as a response to pain or discomfort.

- Milk production will decrease and diarrhea may develop. They will generally recover within two days. Severe cases may be fatal within 24 hours (figure 20).
- Enterotoxemia may have similar symptoms upon onset but progresses more rapidly.
- In dairy animals in early lactation, the advanced stages of rumen acidosis may mimic toxic mastitis or milk fever. Animals that are down with milk fever will not be dehydrated or have diarrhea and will respond to the administration of calcium salts.

The Clinical Signs Depending on the Causes of Disorder

- a) **Silage overfeeding cause:** Anorexia and moderate drop in production, the rumen is usually full, firm and doughy. The feces are normally to firm in consistency but reduced in amount. Temperature, pulse and respiration are normal.
- b) **Excessive feeding of grain:** Anorexia and ruminal a stasis.
 - The rumen is not necessary full and may contain excessive fluid.
 - The feces are usually soft and foul smelling.

- Animal is bright and alert and usually begins to eat within 24hr.

Diagnosis

1. Clinical signs and a history of a change in the nature or amount of the diet.
2. Elimination of other possibilities,
3. Palpation of left flank is usually full, firm and doughy.

Differential diagnosis:

The disease must be differentiated from other possibilities as the following.

- Traumatic reticuloperitonitis by presence of systemic reaction and painful responses to deep palpation of the xiphoid region.
- Ketosis; by presence/albescence of ketonuria .
- Vagal indigestion, by respond to treatment or not.
- Abomasal displacement and grain overload.

Treatment

Treatment includes stopping access to food, remove all concentrate feed, increase consumption of fiber such as grass hay not alfalfa and give moderate amounts of water. Avoid free access to water as it will promote bloating due to the hyperosmolarity in rumen.

1. **Immediately:** Administration of 20–40 L of warm water or saline via a stomach tube, to vigorous dissolving the kneading of the rumen and may help restore rumen function.
2. **Give laxative drug as:** Liquid parafine, 1 liter/100kg body weight orally or 500–1000gm. Magnesium sulfate per mouth seems to be useful when excessive amounts of high-energy feeds have been ingested.
3. **The activity of the rumen increased by:** Give animal stomachic drug per mouth.
 - Vitamin B complex may be given for stimulating thiamine-producing bacteria.
 - Administration of 4–8 L of ruminal fluid from a healthy cow will help.
4. **Neutralize of animal acidosis:** Drench animals with something alkaline such as 6–9 ounces of sodium bicarbonate, which will help neutralize acid as there is a tendency for acidic conditions to develop in the rumen. A veterinarian mix 50–100g of sodium bicarbonate in a saltwater solution and offer

it to the animal. Alternate treatment for neutralization of acid is magnesium hydroxide or magnesium oxide.

5. **If too much urea or protein has been ingested:** Acetic acid or vinegar may be administered per mouth.
6. **Walk the animals and medicate with antibiotics:** Broad-spectrum antibiotic to restrict enterotoxemia and thiamin administration is recommended as polioencephalomalacia is potential sequel.

Grain Overload(Lactic acidosis, Carbohydrate engorgement, Rumen impaction)

Grain overload (acidosis, grain poisoning) occurs when cattle, sheep or goats eat large amounts of grain. The grain releases carbohydrate into the animal's rumen and this rapidly ferments rather than being digested normally. Bacteria in the rumen produce lactic acid, resulting in acidosis, slowing of the gut, dehydration and often death.

Etiology

Wheat and barley are the most common causes of grain overload but it occasionally occurs with oats and wheat grain. Crushing or cracking of grain by a hammer will increase the likelihood of grain overload, because these processes result in quicker release of carbohydrates.

Cases are often seen when stock are suddenly grain fed without being gradually introduced to the grain or pellets, there is a sudden change in feeding regimen or in the grains being fed. Stock graze newly harvested paddocks (where there may be spilled grain or unharvested areas), stock get unplanned access to grain or pellets, such as around silos.

The disease is most common in cattle that accidentally gain access to large quantities of readily digestible carbohydrates; particularly grain. It also is common in feedlot cattle when they are introduced to heavy grain diets too quickly. Wheat, barley and corn are the most readily digestible and oats less digestible. Less common causes include engorgement with apples, grapes, bread, batter's dough, sugar beets, mangles, or sour wet brewer's grain that was incompletely fermented in the brewery. The amount of a feed required to produce acute illness depends on the kind of grain, previous experience of the animal with that grain, the nutritional status and condition of the animal, and the nature of the micro flora.

Cattle accustomed to heavy grain diets may consume 30–45 lb (15–20 kg) of grain and develop only moderate illness while others may become acutely ill and die after eating 20 lb (10 kg) of grain.

Pathogenesis

Ingestion of toxic amounts of highly fermentable carbohydrates is followed within 2–6 hr by a change in the microbial population in the rumen. The number of gram-positive bacteria (*Streptococcus bovis*) increases markedly which results in the production of large quantities of lactic acid. The rumen pH falls to ≤ 5 , which destroys protozoa, cellulolytic organisms and lactate-utilizing organisms and impairs rumen motility. The low pH allows the lactobacilli to utilize the carbohydrate and to produce excessive quantities of lactic acid. The superimposition of lactic acid and its salt lactate on the existing solutes in the rumen liquid causes osmotic pressure to raise substantially which results in the movement of excessive quantities of fluid into the rumen causing dehydration.

The lactic acid causes a chemical rumenitis, and its absorption results in lactic acidosis. In addition to acidosis and dehydration, the pathophysiologic consequences are hem concentration, cardiovascular collapse, renal failure, muscular weakness, shock, and death. Animals that survive may develop mycotic rumenitis in several days, hepatic necrobacillosis several weeks or months later, or chronic laminitis, as well as evidence of ruminal scars at slaughter.

Clinical Findings;

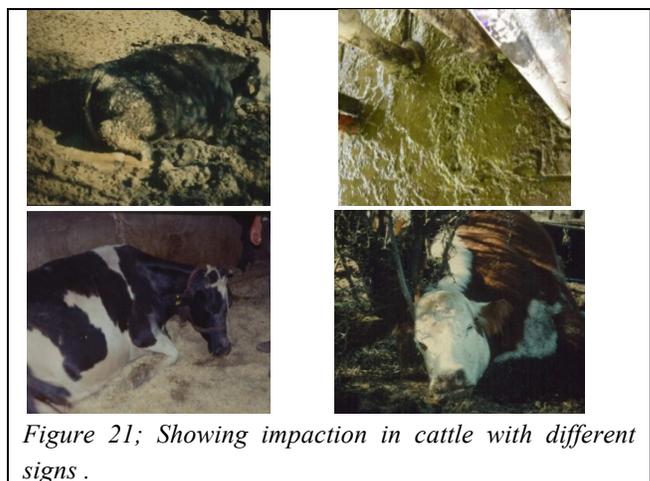


Figure 21; Showing impaction in cattle with different signs .

Carbohydrate engorgement results in conditions ranging from simple indigestion to a rapidly fatal acidosis where the interval between overeating and onset of signs is shorter with ground feed than with whole grain and severity increases with the amount eaten. A few hours after engorgement the only detectable abnormality may be an enlarged rumen and possibly some abdominal pain (manifest by belly kicking).

In the mild form: The rumen movements are reduced but not entirely absent the cattle are anorectic but bright and alert and diarrhea is common. The animals usually begin eating again 3–4 days later without any specific treatment. In cattle that have become ill on smaller amounts of grain, the rumen will feel not necessarily full, but rather resilient because of the excessive fluid. Acute laminitis may be present and is most common in those animals that are not severely affected; chronic laminitis may develop weeks or months later. A urea is a common finding in acute cases and diuresis after fluid therapy is a good prognostic sign. Death may occur in 24–72 hr and rapid development of acute signs particularly recumbence, indicates an urgent need for radical treatment (figure 21).

In severe form; some animals within 24–48 hr of the onset of severe overload will be recumbent, some will be staggering and others will be standing quietly; all will be completely off feed. Immediately after consuming large quantities of dry grain cattle may engorge themselves on water but once ill they usually do not drink at all. The primary contractions of the rumen are completely absent, although the gurgling sounds of gas rising through the large quantity of fluid are usually audible on auscultation. Ballottement and auscultation of the left flank may elicit fluid–splashing sounds in the rumen. The contents of the rumen as palpated through the left flank may feel firm and doughy in cattle that were previously on a roughage diet and have consumed a large amount of grain. Severely affected animals stagger and may bump into objects; their palpebral reflex is sluggish or absent and the pupillary light reflex is usually present but slower than normal. They commonly lie quietly often with the head turned into the flank and their response to any stimulus is much decreased so that they resemble cases of parturient paresis. Body temperature is usually below normal (36.5–38.5°C); however, in animals exposed to the sun in hot

weather it may be increased to (41°C). Respirations tend to be shallow and rapid up to 60–90/minute, heart rate usually is increased in accordance with severity of the acidosis; prognosis is poor for cattle with rates of 120–140/min. In pregnant cattle with severe form of the disease, abortion may occur 10–14 days later.

Diarrhea is common and usually profuse where the feces are soft to liquid yellow, have an obvious sweet–sour odor and contain undigested kernels of the feed that has induced the overload (figure 21).

Diagnosis

The diagnosis is usually obvious if the history is available. It may be confirmed by the clinical findings, a low ruminal pH (<5), and examining the micro flora of the rumen for the presence of live protozoa.

Clinical signs;

As static rumen with gurgling fluid sounds, diarrhea, ataxia, and a normal temperature are characteristic.

Laboratory Examination;

To avoid an increase in pH on exposure to air, rumen fluid obtained by stomach tube or paracentesis should be checked promptly.

- Normally, the pH in cattle on roughage is 6–7; in those on a grain diet, 5.5–6. Values below those ranges are strongly suggestive of overload and a pH < 5 indicates severe acidosis.
- Ruminal fluid may be examined microscopically; 5–7 protozoa are normally seen under low power. In acidosis, the protozoa are virtually absent.
- A gram's stain of the fluid will reveal a change from predominantly gram–negative bacteria (normal) to predominantly gram–positive bacteria in acidosis.
- Increased blood lactate and inorganic phosphate levels, mild hypocalcemia and reduced urinary pH are also seen, but it is seldom necessary to check such values to make a firm diagnosis.

Treatment

For all cattle suspected of having eaten large quantities of concentrate, it is important to restrict water intake for the first 18–24 hr.

- If overload is serious, slaughter for salvage should be considered, it may well be the most economical choice.
- Mortality is high in severely affected animals unless vigorous therapeutic measures are initiated early. In

such animals removal of rumen contents and replacement with ingests taken from healthy animals is necessary.

Initially

In–Severe cases

- Rumen lavage may be accomplished with a large stomach tube if sufficient water is available. A large–bore tube (2.5 cm inside diameter, 3 m long) should be used and enough water added to distend the left flank; gravity flow is then allowed to empty out what it will. Repeating this 15–20 times achieves the same results (and requires about as much time) as using rumenotomy to empty and wash out the rumen with a siphon.
- Emptying the rumen should be followed by rumen inoculation before signs of severe toxicosis are evident by rigorous fluid therapy to correct the acidosis and dehydration and to restore renal function.
- 5% sodium bicarbonate solutions should be given I/V (5 L/450 kg). During the next 6–12 hr, a balanced electrolyte solution, or a 1.3% solution of sodium bicarbonate in saline may be given IV, up to as much as 60 L/450 kg body wt.
- Urination should resume during this period. Usually, it is unnecessary and even undesirable to also administer antacids PO (or intraruminally).
- Animal administrated with antihistaminic drug.

In less severe cases:

- Emptying the rumen is unnecessary. In these, magnesium sulfate (500 g/450 kg body wt) should be added to warm water pumped into the rumen and mixed there in via kneading the flank. This may be all that is necessary if the rumen pH is >5 and the animal is still standing and reasonably alert several hours after the engorgement.
- Animal administrated with antihistaminic drug.

N.B: If good appetite returns within 3 days, the prognosis is good. However, if treatment was not started early enough to prevent acidification of the ruminal contents, and mycotic infection of the rumen wall ensues, relapse is likely within 3–5 days, and the prognosis is bad and advertised to slaughters.

Prevention

Accidental access to concentrates for which the cattle have developed an appetite, in quantities to which they are unaccustomed, should be avoided. Feedlot cattle should be introduced gradually to concentrate rations over a period of 3 wk, beginning with a mixture of ≤50% concentrate in the milled feed containing.

Bloat(Ruminal tympany)

Bloat is simply the buildup of gas in the rumen. This gas is produced as part of the normal process of digestion, and is normally lost by belching (eructation). Bloat occurs when this loss of gas is prevented. There are two sorts of bloat. The least common type is gassy bloat, which occurs when the gullet is obstructed (often by foreign objects such as potatoes) or when the animal can't burp (such as with milk fever or tetanus). The second type of bloat is frothy bloat, which happens as the result of stable foam developing on top of the rumen liquid, which blocks the release of the gas. This is by far the most common form of bloat, and unlike gassy bloat, it is highly seasonal with peaks in the spring and autumn. This is because the foam is formed by breakdown products from rapidly growing forages (particularly legumes such as clover and alfalfa). These increase the viscosity (stickiness) of the rumen fluid and prevent the small bubbles of gas formed by rumen fermentation from coming together to form free gas that can be belched off. Bloat is an over distention of the rumen reticulum with the gases of fermentation, either in the form of a persistent foam mixed with the ruminal contents, called primary or frothy bloat, or in the form of free gas separated from the ingest, called secondary or free–gas bloat.

It is predominantly a disorder of cattle, susceptibility of individual cattle to bloat varies and is genetically determining.

Etiology

Primary Ruminal Tympany(frothy bloat)

- The cause is entrapment of the normal gases of fermentation in stable foam. Coalescence of the small gas bubbles is inhibited and intraruminal pressure increases because eructation cannot occur.
- Both animal and plant influence the formation of stable foam where soluble leaf proteins, saponins, and hemicelluloses are believed to be the primary foaming agents and to form a monomolecular layer

around gas rumen bubbles that has its greatest stability at about pH 6.0. Salivary mucin is antifoaming, but saliva production is reduced with succulent forages. Bloat-producing pastures are more rapidly digested and may release a greater amount of small chloroplast particles that trap gas bubbles and prevent their coalescence.

- The immediate effect of feeding is probably to supply nutrients for a burst of microbial fermentation. However, the major factor that determines if bloat will occur is the nature of the ruminal contents where protein content, rates of digestion and ruminal passage reflect the forage's potential for causing bloat. Over a 24-hr period, the bloat-causing forage and unknown animal factors combine to maintain an increased concentration of small feed particles and enhance the susceptibility to bloat.
- Bloat is most common in animals grazing legume or legume-dominant pastures, particularly alfalfa, ladino, and red and white clovers, but also is seen with grazing of young green cereal crops, rape, kale, turnips, and legume vegetable crops. Legume forages such as alfalfa and clover have a higher percentage of protein and are digested more quickly.
- Frothy bloat also is seen in feedlot cattle, and less commonly in dairy cattle, on high-grain diets. Where the cause of the foam in feedlot bloat is uncertain but is thought to be either the production of insoluble slime by certain species of rumen bacteria in cattle fed high-carbohydrate diets or the entrapment of the gases of fermentation by the fine particle size of ground feed. Fine particulate matter, such as in finely ground grain can markedly affect foam stability as can a low roughage intake. Feedlot bloat is most common in cattle that have been on a grain diet for 1-2 months, timing may be due to the increase in the level of grain feeding or to the time it takes for the slime-producing rumen bacteria to proliferate to large enough numbers.

Secondary ruminal tympany(free-gas bloat);

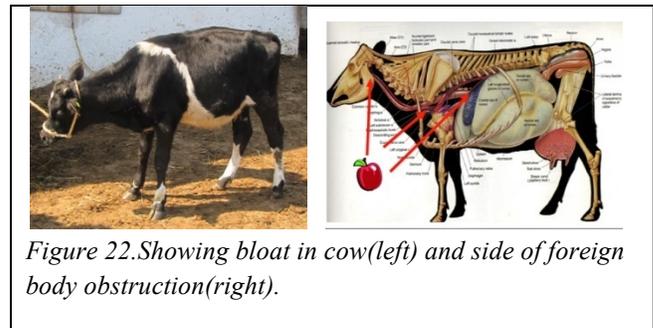


Figure 22. Showing bloat in cow(left) and side of foreign body obstruction(right).

- Physical obstruction of eructation is caused by esophageal obstruction due to a foreign body (e.g., potatoes, apples), stenosis or pressure from enlargement outside the esophagus (as from lymphadenopathy), as illustrated in figure (22).
- Interference with esophageal groove functions in vagal indigestion and diaphragmatic hernia may cause chronic ruminal tympany.
- It also occurs in tetanus.
- Tumors and other lesions of the esophageal groove or the reticular wall are less common causes of obstructive bloat. There also may be interference with the nerve pathways involved in the eructation reflex. Lesions of the wall of the reticulum (which contains tension receptors and receptors that discriminate between gas, foam, and liquid) may interrupt the normal reflex that is essential for escape of gas from the rumen.
- Tympani also can be secondary to the acute onset of ruminal atony that occurs in anaphylaxis and in grain overload where this causes a reduction in rumen pH and possibly an esophagitis and rumenitis that can interfere with eructation, hypocalcaemia Unusual postures, particularly lateral recumbence, are commonly associated with secondary tympani..
- Chronic ruminal tympani is relatively frequent in calves up to 6 month old without apparent cause; this form usually resolves spontaneously.
- Animals may die of bloat if they become accidentally cast in dorsal recumbence or other restrictive positions in handling facilities, crowded transportation vehicles, or irrigation ditches

Clinical Signs

In primary bloat

Bloat is a common cause of sudden death where cattle not observed closely such as pastured and feedlot cattle and dry dairy cattle usually are found dead.

- Cattle with bloat may display the following signs:
- The left flank may be so distended that the continuer of the Paralumber fossa protrudes above the avertable column (distended left abdomen);
- Loss of appetite, a reluctance to move, appear distressed – vocalize, eyes bulging and strain to urinate and defecate; rapid breathing – mouth may be open with tongue protruding and staggering (figure 22).

In secondary bloat

There is tympanic resonance over the dorsal abdomen left of the midline.

- Free gas produces a higher pitched ping on percussion than frothy bloat.
- The distension of the rumen can be detected on rectal examination.
- In free-gas bloat, the passage of a stomach tube or trocarization releases large quantities of gas and alleviates distention.
- In advanced cases the animal will go down and death is rapid at this stage due to the swollen rumen compressing the lungs so interfering with breathing and tissue oxygenation, and obstructing blood flow (figure 22).

Diagnosis

- An accurate clinical signs.
- History of access to lush pasture.
- Passing a stomach tube will distinguish between gassy and frothy bloat. If it's gassy bloat, stomach tube passed into the rumen will allow the gas build-up to escape through the tube. No such gas is seen in frothy bloat.

Treatment

Passing a stomach tube is the best treatment for gassy bloat. Once the gas has been released, the cause of the obstruction should be looked for.

In a few cases a tracer and cannula punched through the side into the rumen will relieve gassy bloat when a stomach tube has not worked. But such cases are rare, and as the tracer provides a tremendous opportunity for introduction of infection, it should only be used as a last resort.

In life-threatening cases

- An emergency rumenotomy or a tracer and cannula may be used for emergency relief.
- When the animal's life is not immediately threatened, passing a stomach tube of the largest bore possible is recommended.

For Frothy Bloat

- Antifoaming agents that disperse the foam should be given by stomach tube. Old-fashioned remedies such as linseed oil and turpentine are effective but newer treatments such as dimethicone or polaxolene are easier to give as the effective dose is much smaller. 100 ml/animal/orally or intraruminal.
- In frothy bloat, it may be impossible to reduce the pressure with the tube and an antifoaming agent should be administered while the tube is in place.
- A variety of antifoaming agents are effective, including vegetable oils (eg, peanut, corn, soybean) and mineral oils (paraffin), at doses of 250–500 ml. Dioctyl sodium sulfosuccinate (docusate), a surfactant, is commonly incorporated into one of the above oils and sold as a proprietary anti-bloat remedy, which is effective if administered early.
- Poloxalene (25–50 g, PO) is effective in treating legume bloat but not feedlot bloat.
- If the bloat is not relieved quickly by the antifoaming agent, the animal must be observed carefully for the next hour to determine if the treatment has been successful or if an alternative therapy is necessary.
- Placement of a rumen fistula provides short-term relief for cases of free-gas bloat associated with external obstruction of the esophagus.

If an Outbreak of Frothy Bloat Occurs

All cattle on that pasture should be removed immediately and put onto a high fiber diet (hay or straw), and any cows showing bloating signs treated with an anti-foaming agent. The pasture should not be grazed for at least ten days.

Controls and Prevention

Prevention of pasture bloat can be difficult

Management practices that have been used to reduce the risk of bloat include feeding hay before turning cattle on pasture where hay to be effective it must be at least one-third of the diet. The only satisfactory method available to prevent pasture bloating is continual administration of

antifoaming agent during the risk period. This is widely practiced in grassland countries. The most reliable method is drenching twice daily (e.g., at milking times) with an antifoaming agent.

The antifoaming agent can be added to the feed or water or incorporated into feed blocks, but success with this method depends on adequate individual intake. The agent can be “painted” on the flanks of the animals from which it is licked during the day, but animals that do not lick will be unprotected.

Available antifoaming agents include oils and fats and synthetic nonionic surfactants. Oils and fats are given at 60–120 ml /head/day; doses up to 240 ml are indicated during most dangerous periods.

To prevent feedlot bloat, rations should contain about 10–15% cut or chopped roughage mixed into the complete feed. Preferably, the roughage should be a cereal, grain straw; pelleted rations made from finely ground grain should be avoided. The addition of tallow (3–5% of the total ration) may be successful occasionally, but it was not effective in controlled trials.

Osteomalacia (Creeps) (Adult rickets);

This is a condition of bone brittleness or softening of bone found usually in adult life. It consists in the decalcification of mature bone, with the advancing diminution of the compact portion of bone by absorption. The periosteum strips very easily from the bone. This disease is seen in cows during the period of heavy lactation or in the later stages of pregnancy and the greater the yield of milk the more rapid the progress of the disease. Heifers with their first calves are frequently affected, as these animals require a considerable quantity of mineral salts for their own growth and for the nourishment of their offspring.

Osteomalacia has a pathogenesis similar to that of rickets but is seen in mature bones and associated with disruption of normal bone remodeling. Because bones mature at different rates, both rickets and osteomalacia can be seen in the same animal. Osteomalacia is characterized by an accumulation of excessive unmineralized osteoid on trabecular surfaces, the long bones may show irregular, diffuse thickening along the

length of their diaphyses; but the bone is soft, easy to cut or saw, and may become permanently deformed.

Disease of bone in adults where there is defective bone mineralization. The condition is characterized by excessively wide seams of osteoid on bone forming surfaces.

Causes

Much the same as given under rickets. Phosphorus deficiency is a common cause of reduced bone mass (osteopenia) and improper bone mineralization (osteomalacia) in cattle. Dystrophies Associated with Calcium, Phosphorus, and Vitamin D disorders.

Clinical Signs;



Figure 23. Indicates atrophy of the supra and infra spinatus muscles and increased volume of the shoulder joint.

The clinical signs observed in the cattle are lameness, stiff gait, postural changes, reluctance to walk, prolonged recumbence and difficulty to stand-up, in marked cases there is a gradual emaciation and symptoms of gastrointestinal catarrh with depraved appetite (the animal eating manure, decayed wood, dirt, leather, etc). Muscular weakness is prominent together with muscle tremors which simulate chills but are not accompanied with any rise of temperature; there is pain and swelling of the joints, and constant shifting of the weight from one leg to another. The restricted movements of the joints are frequently accompanied with a crackling sound which has caused the name of “creeps” to be applied to the disease.

The coat is dull and rough and the skin dry. The animal may be subject to frequent sprains or fracture of bones without apparent cause as in lying down or turning around and when such fractures occur they are difficult to unite.

The bones principally involved are the upper bones of the legs, the haunch bone and the middle bones of the spinal column where the long bones may show irregular

diffuse thickening along the length of their diaphyses; but the bone is soft, easy to cut or saw, and may become permanently deformed. Since the physes are "closed" and there is no bone growth, metaphyseal changes are absent or minimal (figure 23).

Diagnosis

1. Accurate clinical signs and case history.
2. To establish a firm diagnosis of osteomalacia, the diet should be evaluated for calcium, phosphorus and vitamin D content. There is radiographic evidence of generalized skeletal demineralization, loss of lamina duradentes, subperiosteal cortical bone resorption, bowing deformities, and multiple folding fractures of long bones due to intense localized osteoclast proliferation. Levels of hydroxyproline, an amino acid released into blood during bone mineralization, can be determined to assess the extent of ongoing bone mobilization. If dietary calcium and phosphorus content cannot readily be determined (e.g., in grazing animals), soil or fecal samples can be analyzed as crude proxies for dietary intake of these minerals.
3. Laboratory values used to assess renal function should be within normal limits in animals with nutritional osteodystrophy.

Treatment

- Animals with osteomalacia should be confined for several weeks after initiation of the supplemental diet. Response to therapy is rapid; within 1 wk the animals become more active, and their attitude improves. Jumping or climbing must be prevented, because the skeleton is still susceptible to fractures. Restrictions can be lessened after 3 wk, but confinement with limited movement is indicated until the skeleton returns to normal (response to treatment should be monitored radiographically).
- Complete recovery can be achieved within months in animals with no or only minor limb and joint deformities.

Post Parturient Hemoglobinuria (Hypophosphatemia, Heinz Body Hemolytic Anemia)

Post parturient hemoglobinuria (PHU) is an acute sporadic disease of high yielding buffaloes and cow, characterized by hypophosphatemia, intravascular hemolysis mostly

affected buffaloes either in advanced pregnancy or in early lactation with majority of animals are in their 3rd or 6th the lactation. It is characterized by development of per acute intravascular hemolysis and anemia with potentially fatal outcome. Beef and non lactating cattle are rarely affected. The exact cause is unknown, but phosphorus depletion or hypophosphatemia has been incriminated as a major predisposing factor. Severe intracellular phosphorus depletion of RBCs is known to increase osmotic fragility of the RBCs, possibly predisposing to intravascular hemolysis. Although marked hypophosphatemia is commonly diagnosed in affected animals, most cases of severe hypophosphatemia are not associated with intravascular hemolysis, suggesting that hypophosphatemia is not the sole causative factor responsible for post parturient hemoglobinuria. A similar condition reported in New Zealand was associated with copper deficiency, potentially making RBCs more susceptible to oxidative stress. Other potential causes are hemolytic or oxidative plant toxins (often from *Brassica* spp, sugar beets, or green forage).

Occurrence

The transition between late pregnancy and early lactation, from calving until 3–4 week postpartum. It is widely distributed in Egypt especially in winter and usually affected high lactating cow 2–6 week after parturition and may affected buffalo's cow before or after parturition.

Etiological causes

1. Not really understood but sever drop in serum phosphorus level as a result of its secretion in milk and intensive milk production may predispose to the condition
2. Heavy feeding on hemolytic or oxidative plant toxins (e.g. green forage) and plants low in phosphorus (Barseem) may predispose to the case..

Phosphorus function

Phosphorus is essential for.

1. Intracellular function e.g.– Glycolysis, oxygen transport, muscle contraction and Protection of cell membrane from oxidative damage.
2. 2– Phosphorus is a component of bone, milk and ruminant saliva. Phosphorus deficiency is usually

primary and result in organ and system dysfunction and finally progressive demineralization of bone.

Suggested Pathogenesis of Post Parturient Hemoglobinuria

The pathogenesis of post parturient hemoglobinuria briefly occur where animal with heavy lactation and/or oxidative plant toxin and/or low phosphorus level feed suffering from decrease in the blood level phosphorus, that increase osmotic fragility and oxidative injures of RBCs which lead to hemolytic of RBCs and appearance of hemoglobin in urine (Hemoglobinuria).

Clinical Signs;

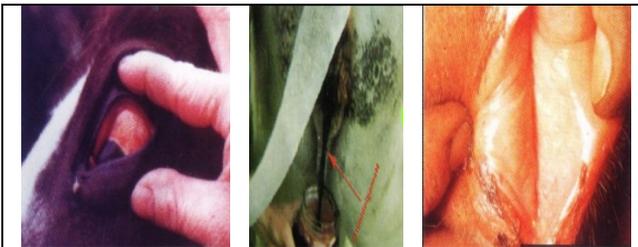


Figure 24; Hemoglobinuria in hypophosphatemia in cattle cow and buffaloes.

Cattle suffering from post parturient Hemoglobinuria have decrease appetite, severe decrease in the milk yield, Hemoglobinuria in the next day (Red color urine), temperature is normal or slight increase. Signs of anemia are pale mucous membrane, Jaundice, cardiac impulse is seriously augmented and recumbence (figure 24).

- Acute cases stay 3–4 days then the cow becomes severely dehydrated and weak.
- Cow may die later from anoxic anemia but also recovery occurs after 3 week with fatality rate of (10–30%).

Diagnosis

- Case History: Time of calving and time of occurrence and plane of nutrition
- Clinical Signs: sudden appearance of red urine (Hemoglobinuria) around the pregnancy stage and normal to moderately rise of body temperature.
- Laboratory: Serum phosphorus level is \square 4mg/dl, low RBCs, HB, PCV and Excessive urine urobilinogen.

Differential Diagnosis

The disease must be differentiated from leptospirosis, water or salt poisoning, Brassica or onion intoxication, chronic copper intoxication, Babesiosis and Bacillary hemoglobinuria.

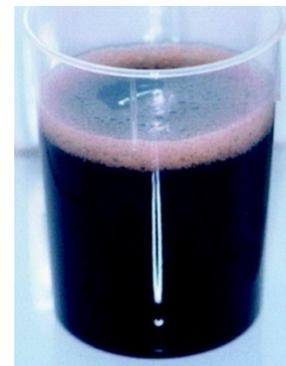
We must be differentiated between hematuria and hemoglobinuria as the following.

Hematuria

- Urolithiasis
- Cystitis
- Acute pyelonephritis
- Water or salt poisoning
- Enzootic hematuria
- Oxalate poisoning

Hemoglobinuria

- Leptospirosis
- Postparturient hemoglobinuria
- Brassica or onion intoxication
- Chronic copper intoxication
- Babesiosis
- Bacillary hemoglobinuria



Treatment

Transfusion of large quantities of whole blood is the best treatment for severely affected cows. Crystalloid fluids may be beneficial if blood is not available and may protect the kidneys against toxic and anoxic damage but monitoring the PCV and the total protein concentration is required to prevent third spacing due to the decreased intravascular pressure.

Immediately

1. A study on treatment trials of hemoglobinuria in buffalo cows and cattle indicated that subsequent treatment with sodium acid phosphate 20% (60 g in 300 ml of water), administered concurrently by i/v, s/c. and oral routes, approximately 100% of affected animals recovered in 1–3 days.

2. 100 g of bone meal administered as a drench two times a day,
 3. Blood transfusion should be attempted in severe anemia (PCV $\leq 16\%$) 4–10 liter/cow from the same family of diseased animal.
 4. Intravenous fluids therapy to sustain hydration.
 5. Basically the diseased animals must be given antioxidant drugs as vitamin C or vitamin E.
- Improvement of any phosphorus insufficiency or disproportion in the ration together with removal of incriminated feeds might prevent further cases. Because of incompatible results with phosphate therapy and the copper–deficient status of affected cows
 - 3–Copper glycinate (120 mg available copper) has been recommended in cases in which copper deficiency is suspected as the underlying cause.

Treatment with Toldimfos Sodium and Tea Leaves and Sodium Acid Phosphate

- Result of this treatment was based on the recovery of urine discoloration;
- The efficacy of toldimfos sodium was 85% followed by tea leaves 56%, and sodium acid phosphate 18%. Following treatment with tea leaves and toldimfos sodium urine was clear next day, and with treatment with sodium acid phosphate urine was clear on third day. Zameer et al., (2010)

Control

1. Pay attention to phosphorus content of the ration at beginning of lactation.
2. Phosphorus supplement or bone meal could be added.

Hypomagnesaemia (Lactation tetany, Grass Tetany, Grass staggers, Wheat Pasture Poisoning).

Hypomagnesaemia is an electrolyte disturbance in which there is an abnormally low level of magnesium in the blood. It is not equal to magnesium deficiency where hypomagnesaemia can be present without magnesium deficiency and may result from a number of conditions including inadequate intake of magnesium, chronic diarrhea, malabsorption, alcoholism, chronic stress and medications such as diuretics use among other.

Occurrence and animal susceptibility

1. Adult lactating cows are most susceptibility due to the loss of magnesium in milk.
2. Hypomagnesmic tetany case is rare in non lactating cattle but has occurred when undernourished cattle were introduced to green cerebral crops but can occur in lactating beef cows in doors feeding silage.

Etiology

Sharp decrease in serum magnesium which may occur when.

1. Absorption of dietary magnesium is unable to meet the requirements for maintenance.
2. Occurs in high lactating cow raised on pastures rich in potassium and nitro fertilizer.
3. Magnesium absorption from the rumen may be reduced when potassium and nitrogen intakes are high while sodium and potassium intakes are low.

Stress and contributing factors

1. Weather stress where the problem is more common during cool, cloudy and rainy condition.
2. When animals grazing at cool season grasses or small grain pastures in spring.
3. The problem mostly occurs in animal's grasses on pastures grown on soils low in available magnesium and high in available potassium.
4. Heavy application of broiler house litter or other high nitrogen and potassium manures may increase the hazard of grass tetany.

Nutritional Stress

1. Forages containing less than 0.2% magnesium and more than 3% potassium and 4% nitrogen are likely to cause grass tetany.
2. Turning cattle onto winter pasture directly from low quality pastures.
3. Grazing on young forage, small grain or rapidly growing lush pastures are the most dangerous.

Medications

- Antibiotics (viomycin, tabomycine, gentamycine and aminoglycosides) where, 30% of diseased animals using these antibiotics have bloke loop of henele resorption in the hypomagnesaemia.
- Digitalis causes an increased intracellular concentration of sodium, which in turn increases intracellular calcium by passively decreasing the action of the sodium–calcium exchanger in the

sarcolemmam where increased intracellular calcium gives a positive isotropic effect.

- Adrenergic drug. Displace magnesium into the cell.
- Insufficient selenium, vitamin D, sunlight exposure or vitamin B6.

Gastrointestinal Causes

The distal tracts digestives secrete high levels of magnesium therefore; secretory diarrhea can cause hypomagnesaemia as in ulcerative colitis, malabsorption, acute pancreatitis., Hydrogen fluoride and poisoning.

Pathogenesis

1. Disease occurs when magnesium drop to 1mg/dl that may associate with hypocalcaemia (< 8mg/dl).
2. Magnisium plays a role as transmitters of impulses to the muscular system leading to secretion of acetyl–choline that activate choline esterase respiratory for muscular contraction.
3. Magnesium deficiency result in muscular tremors, twitch and spasm..

Clinical Signs

The clinical signs may be divided into three stages (acute, sub acute and chronic).

- Restlessness, staggers, over–alerts appearance, excitable or aggressive.
- Animals may fall down and go into convulsions.
- In many cases animals may die without any prior sign of disease.

Acute form

1. Animals off feeding on pasture suddenly, Tremors of muscles and ears.
2. Hyperesthesia followed by frenzy movement, blowing and ataxia, Convulsions, nystagmus, and jaw movement with frothiness of mouth.
3. Cow lay dawn with frequent attempt to rise with slight rise in temperature, pulse and respiration. Increased force of heart beat.
4. Animals may die in an hour's if not treated immediately.

Sub acute clinical Signs Form;



Figure 25. A. Affected cows become separated from the group and have a startled expression with frequent teeth grinding, an exaggerated blink reflex and frothy salivation with muscular tremors and mild convulsion and extended neck

Cow stays for 3–4 days with in appetite, sad face with depression, increased fore and hind limb movement. Animals are refusing to movement with throw head away.

- Increased frequency of urination and defecation associated with straining. Muscular tremors and mild convulsion especially on hind legs and tail with ataxia. In the late stage cows may recover or lay dawn (figure 25).

Chronic Form Clinical Signs

Loss of appetite and loss of body weight decrease in the peak of lactation and cow lay dawn resemble milk fever signs but don't respond to calcium therapy.

Diagnosis

- **Accurate clinical Signs and case history:** Grazing of adult lactating cows on lush green pastures rich in potassium and sodium fertilizers, bad weather and mostly occur in 15 day after parturition.
- **Laboratory diagnosis**
 1. Drop in magnesium and calcium level in blood and cerebrospinal fluid
 2. Mild decrease in serum phosphorus level and increase in serum potassium level
 3. Decreased magnesium level in urine
 4. Decreased magnesium level in urine
- **Differential Diagnosis**
 1. Acute lead poisoning.(Blindness and frenzy movement).
 2. Rabies. (Down paralysis, Dog biting, No convulsion).
 3. Nervous form of ketosis.(Ketenemia ,Ketenuria).

Treatment and control

- **Immediately**

The animal should not be stimulated during treatment, because this could trigger fatal convulsions and it is essential to administer a sedative drug to control the cow's seizure activity and prevent a fatal convulsion, and to facilitate intravenous treatment.

○ ***Animals showing clinical signs require treatment***

- Combined solutions of calcium and Magnesium (400 ml of 40% calcium borogluconate), preferably given slowly i/v, continue injection treatment if necessary
- Chloramphenicol may be replaced by sulphamethoxypyrazine, Ampicillin, penicillin and streptomycin response to treatment is slower in animals with hypomagnesaemia tetany than in animals with hypocalcaemia alone, because of the time it takes to restore Mg in the CSF.

➤ **200ml of 25% Magnesium Sulfate**

It is best to inject under the skin in the area behind the shoulder and over the ribs. Massage the area well after injecting the solution to spread the fluid and aid its rapid absorption into the blood stream.

- **The cow should then be raised into sternal recumbence and left quietly.**
- The administration of magnesium sulfate by injection will only increase plasma levels for 6 to 12 hours, therefore it is essential to offer concentrates/hay to ensure adequate dietary intake to prevent relapse.
- The remaining cows are very likely to have subclinical hypomagnesaemia and will be at risk from acute grass staggers. Blood sampling of a group of at least five cows could be performed to check the herd magnesium status.

Preventive measures immediately.

- 200 ml of magnesium sulfate 50% injected under the skin(s/c) increased the level of magnesium in the blood within 15 minute.
- Correction of mineral deficiencies and elimination of plant toxins from the diet may help prevent recurrence.

Note

- Intravenous injection must be very slowly and avoid heart and respiratory abnormalities.

Ketosis

Ketosis is a common disease of adult cattle. It typically occurs in dairy cows in early lactation and is most consistently characterized by partial anorexia and depression. Rarely, it occurs in cattle in late gestation, at which time it resembles pregnancy toxemia of ewes. In addition to in appetite, signs of nervous dysfunction, including pica, abnormal licking, in coordination and abnormal gait, bellowing, and aggression are occasionally seen. The condition is worldwide in distribution, but is most common where dairy cows are bred and managed for high production.

Etiology and Pathogenesis

To satisfy the requirements of milk production, the cow can draw on two sources of nutrients, food intake and body reserves. During early lactation, the energy intake is insufficient to meet the energy output in milk and the animal is in a negative energy balance. In conventional farming, this is considered to be a normal metabolic situation in high-yielding dairy cows. Cows in early lactation are therefore in a vulnerable situation and any stress that causes a reduction in feed intake may lead to the onset of clinical ketosis. If the feed intake of the cow is not sufficient to meet the demand for energy, there is insufficient ruminal production of propionic acid, the main precursor of glucose in ruminants, which results in hypoglycaemia. Hypoglycaemia leads to a mobilization of free fatty acids and glycerol from the fat stores. However, the liver cannot deal with the acetyl-CoA which results from the oxidization of these fatty acids because of a lack of energy. The excess acetyl-CoA is converted into the ketone bodies acetoacetate and β -hydroxybutyrate and, to a small extent, acetone. Tissues other than the liver can use ketone bodies, but if their production exceeds the rate at which they are used by muscle and other tissues, they accumulate, and ketosis is the result. Ketone bodies are excreted in milk and urine. The reduction of propionic acid production is usually the result of underfeeding or a reduced feed intake. A period of in appetite is normal around calving, but may be exacerbated in the early post-partum period by a deterioration of forage quality, sudden changes in diet or excessive fatness at calving. Other risk factors are month (more likely in winter), increasing parity, milk fever, ketosis in the previous lactation, increased 305-day milk yield in the previous lactation

and the average milk protein percentage in the previous lactation. Cows with milk fever have a greater decrease in feed intake after calving which exacerbates the negative energy balance, increasing the risk of ketosis.

Butyrate is a precursor of acetyl-CoA and is therefore ketogenic agent.

Secondary Ketosis

Secondary ketosis is common and is the result of diseases causing a reduction in appetite in early lactation, such as displaced abomasum, mastitis, metritis, etc. In areas of cobalt deficiency, ketosis may be diagnosed in grazing cattle. Cobalt is required for rumen microbial synthesis of vitamin B12 and is also essential for adequate utilization of propionic acid. Ketosis has also been reported as the major presenting sign in a dairy herd with fasciolosis.

Epidemiology

- All dairy cows in early lactation (first 6 wk) are at risk of ketosis. The incidence in lactation is estimated at 5–16%, but incidence in individual herds varies substantially. Ketosis occurs in all parities (although it appears to be less common in precipitous animals) and does not appear to have a genetic predisposition, other than being associated with dairy breeds.
- Cows with excessive adipose stores (body condition score ≥ 3.75 out of 5.0) at calving are at increased risk of ketosis, compared with those with lower body condition scores. Lactating cows with hyperketonemia (subclinical ketosis—serum BHB concentrations > 12 mg/dl) are at increased risk of developing clinical ketosis, compared with cows with lower serum BHB concentrations.

Clinical Findings

There are two major forms of ketosis occurring in dairy cattle are the wasting and nervous forms where the wasting form is much more common

1. Wasting form of ketosis (Digestive form)

- Initially there may be a gradual decline in appetite over two to five days. Often the appetite is lost in an unusual manner and the cow may eat grass and hay but will not eat grain or silage. The appetite may appear depraved with cows eating any objects, including dirt and stones. Consequently, milk yield falls quickly to a fraction of its initial level, but never

ceases completely. By this stage the affected animal is obviously ill and is disinclined to move, may be stagger or unsteady on its legs and the head is often carried low to the ground.

- Temperature, pulse, and respiration rates of the cow remain fairly normal as the animal loses weight. The coat is described as having a "woody" appearance, presumably due to the loss of fat reserves under the skin.
- The ketones produced by the cow in this disease have a characteristic sweet "sickly" smell, which may be detected on the cow's breath and less commonly in milk samples. Very few affected animals die without treatment; recovery is slow with milk yields gradually improving over one month but never fully returning to normal levels.

2. Nervous form of ketosis

- This form of ketosis is less common and in typical cases the signs are quite striking.
- Affected cows can show a range of signs including apparent blindness and strange movements of the tongue leading to incessant licking of the skin.
- Affected cows may also walk in circles and bellow loudly for no apparent reason. These kinds of behavior can last for one or two hours with the signs starting more suddenly than the wasting form of the disease.

3. Milk Fever Form

- Losses of consciousness, tremor, paresis and not respond for calcium treatment
- Appearance of ketenes on urine and expired airs.

Diagnosis

- An accurate signs and Case history where the clinical diagnosis of ketosis is based on presence of risk factors (early lactation),
- Clinical signs, the clinical signs and ketene bodies in urine or milk when a diagnosis of ketosis is made,
 - **Laboratory Test**

1. Cow-side tests for the presence of ketone bodies in urine or milk are critical for diagnosis. Caution should be exercised in the use of such tests within 48 hr after calving. Due to the large sugar in plasma at calving, a positive test for ketones is very common during this period.

2. Dipstick tests are convenient, but those designed to detect acetoacetate or acetone in urine is not suitable for milk testing. All of these tests are read by observation for a particular color change. In a given animal, urine ketone body concentrations are always higher than milk ketone body concentrations. Without clinical signs, such as partial anorexia, these results indicate subclinical ketosis.

3. Milk tests for acetone and acetoacetate are more specific than urine tests. Positive milk tests for acetoacetate and/or acetone usually indicate clinical ketosis.

➤ **Differential Diagnosis**

Physical examination should be performed because frequently ketosis occurs concurrently with other peripartum diseases. Especially common concurrent diseases include displaced abomasums, retained fetal membranes, and metritis. Rabies and other CNS diseases are important differential Diagnoses.

Treatment

4. Ketosis cases occurring within the first 1–2 wk after calving frequently are more refractory to therapy than those cases occurring nearer to peak lactation

5. The initial aim of treatment is to restore the lack of glucose in the body.

6. A quick-acting glucose supplement is required immediately. Follow-up treatment is aimed at providing a long term supply of glucose.

a. Glucose replacement

7. Intravenous administration of a dextrose solution by a veterinarian is effective in the short term, but follow-up treatment is essential if relapses are to be avoided.

8. Treatment is aimed at reestablishing normoglycemia and reducing serum ketone body concentrations by IV administration of 500 ml of 50% dextrose solution is a common therapy.

9. This solution is very hyperosmotic and, if administered perivascularly, results in severe tissue swelling and irritation, so care should be taken to assure that it is given IV.

10. Drenching with propylene glycol or glycerine has longer term effects. It also has the benefit of ease of administration. Treatment should be continued for

two to four days. Several commercial compounds contain propylene glycol and glycerin

11. Glucose therapy generally results in rapid recovery, especially in cases occurring near peak lactation.

b. Hormonal therapy

12. Many of the long-acting corticosteroids have beneficial effects in ketosis. They are administered by the veterinarian as a single injection.

13. Corticosteroids have the ability to break down protein in muscles to produce glucose, which immediately replenishes the depressed blood glucose levels.

14. When using corticosteroids, it is important to supply an adequate amount of glucose either as a high carbohydrate diet and/or propylene glycol drenches to prevent excessive breakdown of muscle protein.

15. Administration of glucocorticoids including dexamethasone or isoflupredone acetate at 5–20 mg/dose, IM, generally results in a more sustained response.

Long-Acting Insulin Preparation

16. 150–200 IU/day may be beneficial, given IM, should be given in combination with glucose or a glucocorticoid to prevent hypoglycemia.

N.B

17. Glucose and glucocorticoid therapy may be repeated daily as necessary. Propylene glycol (250–400 g/dose, PO) acts as a glucose precursor and may be effective as ketosis therapy, especially in mild cases or in combination with other therapies. This dose may be administered twice per day.

18. Overdosing propylene glycol leads to CNS depression..

Prevention and Control

➤ Prevention of ketosis is via nutritional management.

➤ Body condition should be managed in late lactation, when cows frequently become too fat. The dry period is generally too late to reduce body condition score.

➤ Reducing body condition in the dry period may even be counterproductive, resulting in excessive adipose mobilization prepartum.

➤ A critical area in ketosis prevention is maintaining and promoting feed intake. Cows

tend to reduce feed consumption in the last 3 wk of gestation.

- Feed intake should be monitored and rations adjusted to maximize dry matter and energy consumption in late gestation. After calving, diets should promote rapid and sustained increases in feed and energy consumption.
- Rations should be relatively high in non fiber carbohydrate concentration, but contain enough fiber to maintain rumen health and feed intake. Neutral-detergent fiber concentrations should usually be in the range of 28–30% with non fiber carbohydrate concentrations in the range of 38–41%.
- Dietary particle size will influence the optimal proportions of carbohydrate fractions. Some feed additives, including niacin, calcium propionate, sodium propionate, propylene glycol, and rumen-protected choline, may be beneficial in preventing and managing ketosis. To be effective, these supplements should be fed in the last 2–3 wk of gestation, as well as during the period of ketosis susceptibility.

Milk fever(Calcium Deficiency)

Milk fever is the most common metabolic disorder in dairy cattle. The condition usually occurs just before during or immediately after parturition. This condition is also likely to be the most common cause of apparent sudden death in dairy cows and is a common cause of dystocia and stillborn calves. Milk fever is a disorder mainly of dairy cows close to calving. It is a metabolic disease caused by a low blood calcium level (hypocalcaemia). Between 3% and 10% of cows in dairying districts are affected each year, with much higher percentages occurring on some properties. Jersey cows that are mature and fat and graze lush, clover dominant pasture before calving are most susceptible.

Etiologic causes

Predisposing factors

- Breed, this is probably due to the relatively high production level for small breeds. The incidence of the disease also increases with lactation number. The reason is thought to be the increasing requirement for calcium at parturition

as milk yield increases with each lactation and the ability to mobilize calcium.

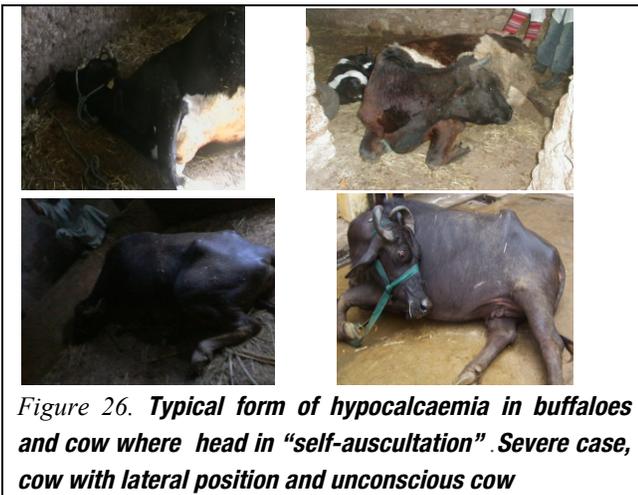
- Some degree of hypocalcaemia occurs in all cows at parturition, but only when this becomes severe do clinical signs develop. Hypocalcaemia is often accompanied by hypophosphataemia and hypermagnesaemia, although milk fever may also be caused by a low magnesium intake in which case there may be hypomagnesaemia.
- The underlying factor in the aetiology of hypocalcaemia at parturition is the sudden increase in calcium requirements for the production of colostrums. The cow adapts to this increased demand by increasing calcium absorption from the gut and the mobilisation of calcium reserves in bone. These processes are controlled by the parathyroid hormone (PTH) through the production of 1, 25-dihydroxycholecalciferol from vitamin D₃, which stimulates increased gut absorption of calcium and probably the mobilisation of calcium from bone.

Factors affecting this adaptation process

- Age of the cow where older cows are less able to mobilise calcium from the skeleton. Heifers are rarely affected. Old cows increase in susceptibility up to the fifth or six calving because they produce more milk and are less able to replace blood calcium quickly.
- High levels of oestrogen around parturition inhibit calcium mobilisation.
- Feed intake and thus calcium intake are often reduced around parturition.
- Bone reabsorption of Ca is inhibited in cows fed high K or high Na diets as a result of metabolic alkalosis.
- High calcium intake during the dry period reduces the response to higher demands.
- Low magnesium intakes reduce the ability of the gut to absorb calcium where about 80% of cases occur within one day of calving because milk and colostrums production drain calcium (and other substances) from the blood, and some cows are unable to replace the calcium quickly enough.

- High producer's cows are more susceptible because the fall in their blood calcium level is greater. Selecting cows for high production may therefore increase the problem with milk fever.
- The feeding management of dry cows in the 2 weeks before calving is very important, because it affects both the amount of calcium available to replace blood calcium and the efficiency with which the available calcium can be used.
- When the amount of calcium in the diet is greater than is needed the efficiency of absorbing calcium from the intestine and the efficiency of transferring calcium from the skeleton both become very sluggish and the chance of milk fever is greatly increased,
- Fat cows are at a greater risk than thin cows due to fat cows produce more milk at calving time also some cows get milk fever several days or even weeks before or after calving. This is usually due to the feed, especially the dietary calcium, being insufficient to meet the heavy demand due to the rapidly growing fetus or milk production in early lactation.

Clinical Signs;



The clinical signs of milk fever include

- Cows that are dull and lethargic ears are cold to the touch, stiffness in the legs. The rumen becomes static, feces tend to bulge in the rectum, these well-recognized signs are due to

the rapid decrease in calcium concentrations in the blood which occurs close to parturition.

- Milk fever depresses rumen contractility and other disorders such as retained placenta, metritis, dystocia, displaced abomasums and ketosis are associated with it.
- Feed intake can drop and worsen the energy status of the already affected cow. This negative energy balance in the postpartum cow will have a strong influence on subsequent fertility.
- In typical cases cows show some initial excitement or agitation and a tremor in muscles of the head and limbs. Then they stagger and go down to a "sitting" position, often with a 'kink' in her neck, and finally lie flat on their side before circulatory collapse, coma and death. A dry muzzle, staring eyes, cold legs and ears, constipation and drowsiness are seen after going down. The heart beat becomes weaker and faster.
- The body temperature falls below normal, especially in cold, wet, windy weather. These signs are due mainly to lowered blood calcium levels. Sometimes there are additional signs due to complicating factors. Bloat is common in cows unable to "sit up" because the gas in the rumen is unable to escape. Pneumonia and exposure may affect cows left out in bad weather (figure 26).

Treatment

Treatment generally involves calcium injection by intravenous, intramuscular or subcutaneous routes. Before calcium injection was employed, treatment comprised inflation of the udder using a pneumatic pump. Inflation of the udder worked because the increased pressure created in the udder pushed the calcium in the udder back into the bloodstream of the cow. Intravenous calcium is potentially fatal through "heart blockade", or transient high calcium levels stopping the heart so should be administered with care.

In unclear cases of downer cows, intravenous calcium injection can lead to diagnosis. The typical reaction will be a generalized tremor of the skeletal muscles, and sometimes cardiac arrhythmia. Defecation, urination and eructation are frequent during the

treatment due to pharmacological effect of calcium on the smooth muscles. In stages 1 and 2, the cow can stand up approximately 10 minutes after the end of the intravenous injection. But in stage 3, it may take two or three hours. If the cow is in stage 3 and lying on its side, it is important to put the cow in a sternal position or else risk aspiration.

The prognosis is generally good even in advanced cases however some cows can relapse the following day and even a third time the day after.

Treatment should be given as soon as possible and all equipment should be kept sterile to avoid abscess formation at the site of injection.

1. Immediately

- The animal injected by, 300–500 ml of a 40% solution of calcium borogluconate, with, I/V injection by very slowly to avoid sudden death.
- 600 ml of combined solutions contain additional ingredients such as magnesium, phosphorus and dextrose (for energy) e.g Ringer solution. Which may also be at low levels in the blood of cows have milk fever.
- Animal injected by AD3E, 10 ml I/M daily per animal.
- Dexamethasone.5–12ml. I/M per animal.
- 5–10 ml adcoferine (as a heart tonic).
- Some cows that have been comatose may have regurgitated and inhaled rumen content into the lungs. If there is ruminal material around the nose one should be suspicious that this may have happened and intensive antibiotic treatment should be commenced as soon as possible as inhalation pneumonia is often fatal.
- **Note**
 - Injection of the solution by farmers should be in several places under the skin on the neck or behind the shoulder, unless the cow is in a coma or there are other reasons for desiring a quick response.
 - Injection into a vein should be left to a veterinarian as it can cause sudden death if not carried out properly. Veterinary assistance is also advisable if there is not a quick response to treatment, because other problems may also be present.

- Cows that are "flat out" should be propped up into a normal resting position to relieve bloat. If weather conditions are bad, or the response to treatment is slow, transfer the cows to shelter to prevent exposure and other complications. Provide feed and water. Ragging helps.
- Recovered cows should not be milked for 24 hours; then the amount of milk taken should be gradually increased over the next 2–3 days.

Prevention

Changing the cow's diet during the transition period (from 4 weeks before calving until 4 weeks after calving) can reduce the occurrence of milk fever and other metabolic diseases, and optimize production and fertility. The simplest approach is to restrict the amount of green feed in the last 2 weeks of pregnancy and provide hay from sources that not recently been treated with potash fertilizers. At the other end of the scale cows may be fed a total mixed ration that includes a balance of dietary cations and anion.

Feeding hay prior to calving and restricting access to green feed results in acidic blood which favors calcium mobilization from bone and improves calcium absorption from the intestines, both of which are important factors in preventing the occurrence of milk fever.

The Downer Cow Syndrome(Creeper Cow)

The Downer Cow Syndrome is condition occurs in cattle usually following hypocalcaemia parturient paresis. It is characterized clinically by prolonged recumbence even after two successive treatments with calcium. Postmortem reveals there are traumatic injuries to limbs muscles and nerves, ischemic necrosis of the muscle limbs, myocarditis, fatty infiltration and degeneration of the liver.

Causes

Definite cause is not clear. It may be:

- Complication of hypocalcaemia cause traumatic injuries due to spread lying of the hind legs during hypocalcaemia, if they forced to get up or walk on slippery floor.
- A difficult parturition due to oversize calf resulted in pelvic oedema, injuries, and failure of cow to get up following parturition.

- The increase level of serum glutamic oxalacetic transaminases (SGOT) indicates the increase muscle damage.
- Prolonged recumbence of hypocalcaemia case predisposing the cause (4– 6 hours).
- Serum electrolytes imbalance or deficiency have suggest as the cause of prolonged recumbence.
- Hypophosphataemia is regarded as a common cause in some regions and responds to phosphorus therapy.
- A long term low level of magnesium has been suggested as a cause especially when accompanies hypocalcaemia.
- Hypopotassaemia (hypokalaemia) with Hypophosphataemia, the most common quoted cause especially in the so called creeper cows which are bright and alert and crawl about but unable to rise.

Occurrence

The disease occurs most commonly in the first 2 or 3 days after calving in heavy milk producers and many cases occurs concurrently with parturient paresis.

Clinical Findings;



Figure 27. Downer cow syndrome with splayed legs.

It may occur independently follow apparent recovery after treatment of hypocalcaemia.

In the typical cases:

Affected cows either make no effort or unable to stand following treatment for

1. Parturient paresis with not raise for up to 24 hours following as illustrated in (figure 27).
2. Loss of appetite.
3. Temperature is normal and heart rate may be normal or elevated (80–100/b/m).
4. Tachycardia and arrhythmia may occur in some cows immediately following calcium treatment.
5. Proteinuria is common and marked protein urea indicates extensive muscle damage.
6. Frequent attempts to raise result in the cow or creeping along the ground with both hind legs.
7. In some cases, the hind legs are extended on each side of the cow and reach up to elbow joint on each side as illustrated in (figure 27).
8. In some cows marked tendency to lateral recumbence with head down back.
9. Some tetany of limbs appears.

Diagnosis

Accurate clinical signs: Rectal exam – determine pelvic involvement or fracture, Coxofemoral dislocation, determine the position and relative size of the rumen and uterus (bloat / torsion / displacement), determine if the calf is stuck and pressing on nerves etc.

1. Vaginal exam – determine if there is a retained fetus / retained membranes, the nature of any discharge / manipulate the calf's position etc.
2. Test particular nerves, joints and bones for function / determine their involvement.
3. Assess temperature, heart rate, respiration rate, pulse quality and mucous membranes.
4. Administer Calcium Borogluconate (CBG) intravenously.
5. Administer Magnesium, Glucose, Phosphorus and Potassium intravenously.
6. Take blood or urine samples for testing.
7. Give you an idea of the cow's prognosis based on the results of these procedures.

Laboratory clinical Pathology Diagnosis

- Ca, P, Mg and glucose levels of the blood are within normal range.
- The levels of CPK and SGOT are usually markedly elevated (18–24 hours after the onset of recumbence).
- Increase CPK indicates muscular damage.

Treatment

Immediately

1. Nursing care – roll the cow several times a day to alternate sides to avoid muscle damage / compartment syndrome. Often cows will remain down due to secondary muscle damage and pressure on nerves and vessels.
2. Inflatable bags to elevate hindquarters can be used.
3. Soft substrate / good footing to aid if cow will stand.
4. Hip clamps can be used to elevate the cow, reduce dependant swelling and provide preliminary support as the cow regains the ability to stand. Hip clamps must be used carefully so as not to cause further injury.
5. Give oral glucose or Propylene Glycol to fix an energy imbalance.
6. Give oral calcium (Calcium chloride or Calcium proprionate)
7. Use of solution containing K, Ca, Mg, and P has been recommended.
8. Treated cases (prognosis) are of poor prognosis when recumbence still 7–10 days.

Control

The detection of hypocalcaemia and their treatment reduce the occurrence of the case.

- If recumbence occur, treatment as soon as possible.
- Avoid slippery ground surface.

Photosensitization

Photosensitization occurs when skin becomes more susceptible to ultraviolet light because of the presence of photodynamic agents. Photosensitization differs from sunburn and photo dermatitis, because both of these conditions result in pathologic skin changes without the presence of a photodynamic agent. In photosensitization, unstable, high–energy molecules are formed when photons react with a photodynamic agent. These high–energy molecules initiate reactions with substrate molecules of the skin, causing the release of free radicals that in turn result in increased permeability of outer cell and lysosomal membranes. Damage to outer cell

membranes allows for leakage of cellular potassium and cytoplasmic extrusion. Lysosomal membrane damage releases lytic enzymes into the cell.

Photosensitization is typically classified according to the source of the photodynamic agent.

1. Primary (type I) photosensitivity,
2. Aberrant endogenous pigment synthesis (type II) photosensitivity,
3. Hepatogenous (secondary, type III) photosensitivity.
4. Idiopathic (type IV) photosensitivity has been described.

Causes

A wide range of chemicals, including some that are fungal and bacterial in origin, may act as photosensitizing agents. However most compounds that are important causes of photosensitivity in veterinary medicine are plant–derived, Photosensitization occurs worldwide and can affect any species but is most commonly seen in cattle,

Primary Photosensitization

Ingestion of plants that contain light–sensitive substances .Some plants contain compounds which become, toxic when activated by light. St John’s wart is one such plant which has glands on leaves, stems and petals containing the light–sensitive compound hyperacid. If enough compounds accumulate in blood vessels at the surface of exposed skin, sunlight transforms it into a toxin and skin damage results.

Secondary Photosensitization

Liver damage from toxins the green pigment chlorophyll in plants is metabolized in the animal to a light–sensitive compound phylloerythrin. The liver excretes phylloerythrin in bile to the intestine. When the liver is damaged, phylloerythrin cannot be excreted and it builds up in the bloodstream. If a sufficient quantity of phylloerythrin is present in the blood vessels at the surface of exposed skin, sunlight transforms the phylloerythrin into a toxin which severely damages the skin.

Clinical signs;



Figure 28; Typical cases of photosensitization affect non-pigmented skin which may ooze serum, becomes dry, parchment-like and sloughs.

Only those areas exposed to sunlight will be affected that is non-pigmented skin with little hair, skin damage also occurs along the back and on the sides of the udder that are exposed to the sun with damage limited to the white areas and sparing of the colored areas being characteristic. The skin becomes red, weepy and swollen, swelling causes the ears to droop and eyelids to close, swollen lips make it difficult for the animal to eat, also may be swelling under the jaws, the animal may have difficulty breathing when there is extensive swelling around the nostrils and throat, while in the most severe cases the surface skin may crack, die and turn black. This dead skin may slough off. There is intense irritation, pain, agitated, scratch and rub against fixed objects, shake their head, seek shade and lose their appetite (figure 28).

Affected cattle will have a sharp drop in milk production with their udder affected will often kick at their belly.

Diagnosis

- **Accurate clinical signs** are easily recognized in cases of marked photosensitivity but are similar to the primary a clinic effects of sunburn in early or mild cases.
- Reference to the specific diseases in which photosensitization is an objective sign may assist in diagnosis of the underlying disease.
- **Laboratory diagnosis;** Evaluation of serum liver enzymes and liver biopsies may be necessary to confirm the presence of hepatic disease. Examination of blood, feces, and urine for porphyrins can also be performed.
- **Differential Diagnosis**

Photosensitization may be confused with simple sunburn and other causes of dermatitis. It is important that the disease 'bluetongue' (an exotic viral disease of ruminants) is ruled out.

Treatment

Immediately

1. Affected animals should be removed from pasture suspected to contain the photodynamic agent and should only be allowed access to grazing at night.
2. Any drugs or medications currently received by the animal should be stopped.
3. Corticosteroids anti-inflammatory drugs such as flunixin meglumine and topical steroid creams may be useful to decrease the severity of cutaneous lesions in the acute stage of the disease.
4. Secondary bacterial infections of cutaneous lesions are common and should be treated with broad-spectrum antibiotics and correct wound management. Lesions generally heal well and surgical debridement is reserved for only the most severely necrotic lesions.

If the animal is affected by secondary photosensitization,

5. Supportive treatment should be provided. A low-protein, high-energy diet rich in branched-chain amino acids is recommended.
6. Intravenous fluid therapy with 5% dextrose may be necessary.
7. Prognosis and eventual productivity of an animal is related to the site and severity of the primary lesion and/or hepatic disease and to the degree of resolution.

Traumatic Reticuloproteinieties Hardware Disease(*Foreign Body Disease*)

Traumatic reticuloproteinieties is common disease in adult cattle caused by the ingestion and migration of foreign body in the reticulum. Cattle are more likely to ingest foreign bodies than small ruminants since they don't use their lips for prehension and are more likely to eat a chopped feed. The disease caused a great economic loss among cattle (decreased milk yield, decreased feed conversion and deaths'. The typical foreign body is metallic object such as apiece of wire or nail often greater than 2.5cm in length or any sharp needles etc. that

can cattle swallow it during grazing, hand feeding. Dairy cattle older than beef cattle since they are more likely to be chapped feed, such as silage or roughage.

The disease caused great economic losses among cattle (decreased milk yield, decreased feed conversion and deaths) and the typical foreign body is metallic object, such as a piece of wire or nail, often greater than 2.5cm in length. Or any sharp needles etc. that can cattle swallow it during grazing, hand feeding.

Dairy cattle older than 2 years of age more commonly affected than beef cattle since they are more likely to be feed chopped feed, such as silage or roughage.

A large number of adult dairy cattle have metallic foreign bodies in their reticulum without signs of clinical disease; where tenesmus of gravid uterus is likely predisposing factors causing migration of the foreign body into the reticular wall.

Pathogenesis of traumatic reticuloperitonitis

Lack of oral discrimination in cattle lead to ingestion of foreign bodies. That may be lodged in the upper esophagus and cause obstruction, may be in the esophageal groove and causes vomiting, may remain fixed without penetration and without serious results and will gradually be voided away or mostly it may pass to the reticulum where the vigorous contraction, the nail penetrate it, rarely may it be passing toward the liver or spleen.

Sequels of traumatic perforation of reticulum

- a. Acute local peritonitis, diaphragmatic hernia.
- b. Acute diffuse peritonitis, toxemia, Depression and recumbence.
- c. Pericarditis, death with chronic heart failure.
- d. Chronic local peritonitis, vagus indigestion.
- e. Recovery.

Clinical Signs;

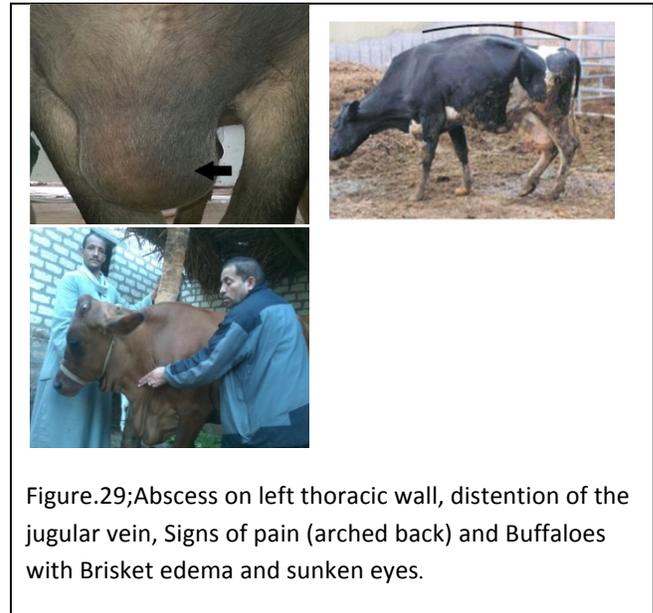


Figure.29; Abscess on left thoracic wall, distention of the jugular vein, Signs of pain (arched back) and Buffaloes with Brisket edema and sunken eyes.

The initial attack is characterized by sudden onset of rumino-reticular atone, fecal output is decreased, sharp fall in milk production, the rectal temperature is often mildly increased, The heart rate is normal or slightly increased and respiration is usually shallow and rapid.

1. The cow exhibits an arched back; an anxious expression; reluctance to move and careful gait. Forced sudden movements as well as defecating, urinating lying down getting up and stepping over barriers may be accompanied by groaning.
2. A grunt may be elicited by applying pressure to the xiphoid or by elevating this area firmly and then pinching the withers which causes extension of the thorax and lower abdomen.
3. In chronic cases, feed intake and fecal output are reduced and milk production remains low. Signs of cranial abdominal pain become less apparent and the rectal temperature usually returns to normal as the acute inflammation subsides and peritoneal contamination is walled off. Some cattle develop chronic vagal indigestion, possibly due to the adhesions that form after foreign body perforation, particularly those on the ventromedial reticulum.
4. Cows with pleuritis or pericarditis due to foreign body perforation usually are depressed, tachycardic (>90r /m) and pyrexia (40°C). Pleuritis

is manifest by fast shallow respiration; muffled lung sounds and possibly pleuritic friction rubs.

5. Thoracentesis may yield several liters of fluid.
6. Traumatic pericarditis usually is characterized by muffled heart sounds, possibly with pericardial friction rubs and occasionally by gas and fluid splashing sounds on auscultation. Jugular vein distention with a pronounced jugular pulse is present early in the course and congestive heart failure with marked submandibular and brisket edema is a frequent sequel (figure 29).
7. Penetration through the myocardium usually results in extensive hemorrhage into the pericardial sac and sudden death.

Complications of TRP

Reticular abscesses are a common complication of TRP. Also, if the foreign body migrates through the diaphragm and into the pericardium, it can cause septic pericarditis and subsequent congestive heart failure. Less common complications include reticular fistulation, vagal indigestion, and diaphragmatic hernia.

Diagnosis

Accurate clinical signs and case history. This can be based on history (when available) and clinical findings if the cow is examined when signs initially appear. Without an accurate history and when the condition has been present for several days or longer, diagnosis is more difficult. Other causes of peritonitis, particularly perforated abomasal ulcers, can be difficult to distinguish from traumatic reticuloperitonitis.

Differential Diagnoses

The case must be differentiated from the conditions that can produce variable or nonspecific GI signs, e.g., indigestion, lymphosarcoma, or intestinal obstruction.

- Abomasal displacement or volvulus should be ruled out by simultaneous auscultation and percussion.
- Pleuritis or pericarditis of no traumatic origin produces signs similar to those associated with foreign body perforation.

Laboratory Tests

1. In many cases there is a neutrophilia with a left shift. Fibrinogen and in chronic cases total plasma protein concentrations may be high. The acid–base status and serum electrolyte levels are typically normal because abomasums and small–intestinal absorption

can remain normal. However marked hypokalemic, hypochloremic metabolic alkalosis can be seen, presumably because adynamic ileus from peritonitis can affect abomasums and GI motility and resorption of abomasums secretions. The metabolic alkalosis can be created or exacerbated by treatment with alkalizing agents such as magnesium hydroxide used as a laxative.

2. Peritoneal fluid analysis can be helpful in determining if peritonitis is present, the nucleated cell count and the protein level return to normal as the contamination is walled off.

Radiographs

May be used to detect metallic material in the reticulum and to determine whether the reticulum has been perforated, the foreign body must be visible beyond the border of the reticulum or be positioned off the floor of the reticulum.

Depression in the cranioventral aspect of the reticulum or identification of an abscess (by gas accumulation outside a viscous), soft–tissue masses, or a fluid line in the cranial abdomen are also reliable radiographic findings. A perforating foreign body will remain in the ventral aspect of the reticulum and be surrounded by gas. Electronic metal detectors can identify metal in the reticulum but do not distinguish between perforating and non perforating foreign bodies. Ultrasonography of the heart and thorax is useful in the diagnosis of pleuritis.

Treatment

Treatment of the Typical Case May Be Surgical or Medical

Conservative treatment consists of instillation of a magnet to recover or immobilize the metal foreign body if the foreign body is composed of magnetic metal

1. Medical treatment involves administration of antibiotic to control the peritonitis and a magnet to prevent recurrence. Because of the mixed bacterial flora in the lesion, affected cattle should also receive 3–7 days of systemic antibiotic therapy (Penicillin, ceftiofur, Ampicillin, or tetracycline), stall rest and other supportive therapy as indicated. a broad–spectrum antimicrobial agent such as ox tetracycline (6.6–11 mg/kg) should be used. Penicillin (22,000 IU/kg, IM, bid) is used widely and

is effective in many cases despite its limited spectrum.

2. Affected cows should be confined for 1–2 wk, placing them on an inclined plane (elevated in front) may limit further penetration of the foreign object.
3. Supportive fluid therapy and/or calcium borogluconate, should be administered as needed (oral or occasionally I/V fluids and S/C).

Affected Cattle should be Re-Evaluated in 48–72 Hours

If a magnet is already in place or conservative therapy is not successful, an exploratory laparotomy /rumenotomy is indicated for removal of the foreign body

- Surgery involves rumenotomy with manual removal of the object or objects; if an abscess is adhered to the reticulum, it should be aspirated (to confirm that it is an abscess) and then drained into the reticulum.
- Slaughter should be advised if the animal does not respond to treatment.

Prevention

Preventive measures include avoiding the use of baling wire, passing feed over magnets to remove metallic objects and keeping cattle away from sites of new construction, and completely removing old buildings and fences. Additionally bar magnets may be administered by mouth, preferably after fasting for 18–24 hr. usually the magnet remains in the reticulum and holds any ferromagnetic objects on its surface. There is good evidence that giving magnets to all herd replacement heifers and bulls at 1 year of age minimizes incidence of traumatic reticuloperitonitis.

Vegal Indigestion (Chronic Indigestion);

Cattle suffering from vagus indigestion develop progressive intermittent then chronic abdominal distention. Improper four stomachs emptying due to a functional outflow problem can be caused by damage to the ventral vagal trunk. Mechanical inhibition of motility from adhesions or abscesses can also decrease four stomachs emptying. The vagus nerve runs along both sides of the esophagus and terminates in branches that innervate the four stomachs and abomasums.

Inflammation or traumatic damage to the nerve can result from pharyngeal trauma or abscesses,

Etiology and Pathogenesis

Various diseases can cause vagal indigestion due to injury or pressure on the vagal nerve and conditions resulting in mechanical obstruction of the cardia or reticulo–omasal orifice (e.g. papillomas or ingested placenta)

There are 4 types of vagal indigestion based on the site of the functional obstruction.

- Type I is failure of eructation or free–gas bloat
- Type II is a failure of omasal transport
- Type III is abomasal impaction
- Type IV is partial obstruction of the fore stomach.

i. *Type I vagal indigestion or failure of eructation results in free–gas bloat*

It is most commonly due to inflammatory lesions of the vagus nerve such as localized peritonitis adhesions (usually after an episode of traumatic reticuloperitonitis) or chronic pneumonia. While less common causes include pharyngeal trauma, which affects a more proximal part of the vagus nerve and esophageal compression by abscesses or neoplasia such as lymphosarcoma, also Free–gas bloat can also be seen with esophageal obstruction by intraruminal foreign bodies or masses.

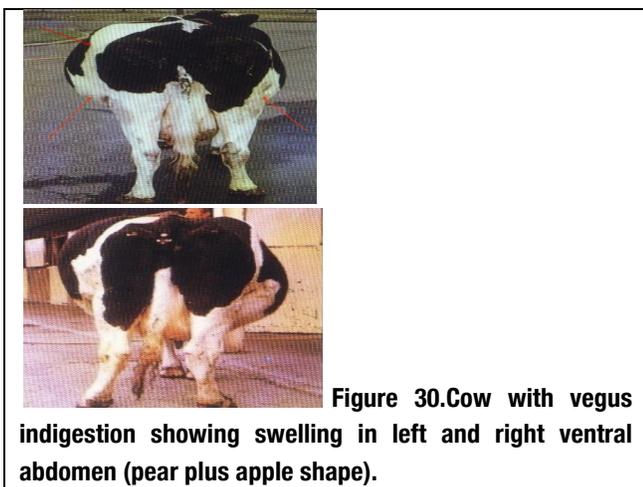
ii. *Type II vagal indigestion or failure of omasal transport*

Develops as results of any condition that prevents ingest from passing through the omasal canal into the abomasums and adhesions or abscesses (reticular or single liver abscesses) are the most common causes. Where reticular abscesses and adhesions are almost invariably the result of traumatic reticuloperitonitis.

Mechanical obstruction of the omasal canal by ingested material (e.g., placenta), pathological masses (e.g., lymphosarcoma, squamous cell carcinoma, granulomas, or papillomas) or ingestion of plastic bags, skin, cloths, dust, sand, licking of hair and wool (phytobezoares). What is a phytobezoar? A phytobezoar is also known as a fiber ball. They are accumulations of indigestible plant material in the digestive system of cattle. These occur in areas where fibrous feeds make up a substantial part of the cows diet.

1. Initially these fibrous materials accumulate in the rumen, contractions of the rumen then cause these fibers to roll around and form a ball. The balls can be variable in size, from marble size to grapefruit even football size.
 2. Phytobezoars are commonly seen in cattle grazing large amounts of fibrous weeds such as onion weed or nut grass. Onion weed mostly dominates pastures in the autumn following a dry summer but obstruction due to phytobezoars occur most commonly in the following spring or summer. This is thought to be associated with lush feed and increased gut activity, where which can also cause chronic ruminoreticular distention due to failure of omasal transport.
- iii. ***Type III vagal indigestion is abomasal impaction***
Develop due to feeding of dry course roughage such as straw in a chopped or ground form with restricted access to water and usually during extremely cold temperatures abomasal impaction and can develop in cattle after abomasal volvulus without abomasal impaction.
- iv. ***Type IV vagal indigestion;***
It typically develops in cattle during gestation which may be related to the enlarging uterus shifting the abomasums to a more cranial position which inhibits normal motility.

Clinical Findings;



Clinical Signs Typically Appearing in this Disorder

Physical exam includes rectal palpation of an L shaped rumen with the ventral rumen sac to the right of the midline. The left flank and right ventral abdominal quadrant will be distended resulting in the characteristic "papple" shaped abdominal contour (The left side looks "apple" shaped and the right side looks "pear" shaped) and Palpation of the pharynx and esophagus can help to locate inflammation, cellulitis or/and a foreign body as illustrated in (figure 30).

Hydration status can be assessed by tenting the skin above the eye and observation of the eye's location in the orbit. Passage of stomach tube will determine if rumen distention is due to free gas or ingests. Abdominal distention, anorexia, loss or decreased appetite, bloat in ruminants, Bloody feces, decreased amount or absent feces and constipation. Rumen hypomotility or atony (decreased rate of motility and strength)

Pain on external abdominal pressure, decreased or absent milk production and decreased respiratory rate (dyspnea, difficult, open mouth breathing, grunt and gasping). Mucous nasal discharge, bradycardia, slow heart beat or pulse, generalized weakness, Inability to stand, downer and prostration in late stage.

Laboratory finding

Hematologic findings vary where the PCV can be increased because of dehydration or decreased because of bone marrow depression (anemia of chronic disease). The WBC may be normal, increased, or decreased. If an inflammatory condition such as peritonitis is present, the neutrophil to lymphocyte ratio is typically reversed, and a neutrophilia may be present. Lymphocytosis can be seen with vagal indigestion due to lymphosarcoma. Leucopenia may be present with diffuse peritonitis. Increased serum globulin and total protein can be seen with abscesses. Metabolic status is normal or metabolic alkalosis may be present. The chloride level varies with the site of the obstruction. Low chloride indicates reflux of chloride from the abomasums into the rumen and obstruction at the level of the abomasums. The chloride levels of the rumen fluid may be increased.

- Metabolic alkalosis is typically present if serum chloride is decreased. The chloride is usually normal if the lesion is cranial to the abomasums.

Potassium is usually low due to decreased potassium intake in the feed.

- Calcium is often moderately decreased because of ongoing milk production; however, it can be low enough to cause recumbence.
- BUN and creatinine increase with dehydration due to pre renal azotemia.

Diagnosis

Diagnosis is based on the presence of sub acute to chronic ruminoreticular and abdominal distention. Because vagal indigestion is by definition a sub acute to chronic disease, this diagnosis should not be made in cattle that have not been sick for at least several days, which rules out acute rumen tympani and acute frothy bloat. Diagnosing the specific cause of vagal indigestion is more difficult but is important because of differences in treatment and prognosis. Physical examination, rectal examination, CBC, blood acid–base determination, and serum chemistry values are often useful. Peritoneal fluid analysis can support the diagnosis of peritonitis if total protein or nucleated cells are increased. Radiographs of the reticulum should be taken to identify a radiopaque linear foreign body (e.g., wire) or reticular abscess. Definitive diagnosis often requires exploratory surgery (left paralumbar fossa laparotomy and rumenotomy).

Differential Diagnosis

Other causes of abdominal distention, such as ascites and uterine enlargement, are included in the differential diagnosis and can almost invariably be ruled out by rectal palpation due to the absence of ruminoreticular distention.

Occasional cases of longstanding obstruction of the cecum or small intestine can cause severe ruminoreticular and abdominal distention; however, palpable cecal or small–intestinal distention is also palpable rectally. In addition, the rumen is distended but not L–shaped, and a characteristic ping is present in the case of cecocolic volvulus

Treatment and Prognosis

If the value of the animal justifies treatment, surgery is almost always needed to identify the underlying cause.

Generally

- **Medical Management Alone is Ineffective**
 - A left flank laparotomy(rumenotomy) provides the opportunity for definitive treatment in some cases

where emptying the rumen at the time of surgery may help restore normal rumen motility, stimulation of low–threshold tension receptors in the reticulum occurs under normal circumstances and causes reflex reticuloruminar contractions (figure 30).

- Supportive or symptomatic therapy should be provided in all cases which typically involve correcting dehydration as well as calcium and electrolyte deficits, commonly with oral fluids and electrolytes. Severely dehydrated animals and those with longstanding disease require I/V fluids therapy.
 - Transfaunation at surgery and/or via stomach tube may help reestablish normal rumen flora in cattle with chronic anorexia.
 - Antibiotics should be given if the underlying cause is infectious or if a rumen fistula is created, choice of antibiotic should be based on culture results if possible.
1. **Type I vagal indigestion (failure of eructation);**

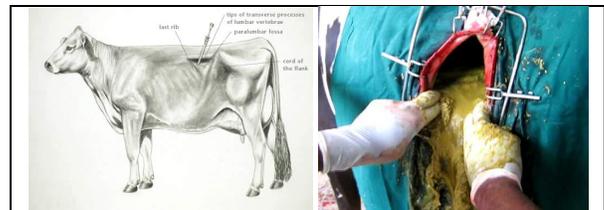


Figure 31. Rumenotomy and side of tracer and canula in cattle.

- Creating a rumen fistula to allow free gas to escape. If surgery is not economically feasible and the underlying cause of vagal indigestion has been identified a rumen tracer can be placed temporarily (figure 31).
- The tracer should not be removed for at least 2 wk to allow firm adhesions to form between the rumen and body wall.
- The prognosis for animals with type I vagal indigestion is usually favorable where after creation of a rumen fistula the signs resolve in nearly all cases.

- Animals with chronic respiratory disease or pharyngeal trauma may not recover from the underlying condition.
- 2. Type II vagal indigestion (failure of omasal transport);**
- Rarely responds to supportive or symptomatic therapy without surgical intervention.
 - Left flank laparotomy (rumenotomy) can be used to identify adhesions in the vicinity of the reticulum, reticular or hepatic abscesses or obstruction of the omasal canal, removal of foreign bodies, wires, and some masses at surgery affords an excellent prognosis as illustrated in (figure 31)
 - Reticular abscesses identified at surgery should be cautiously drained into the reticulum, and antibiotics given for 10–14 days. Reportedly, 83% of cattle with reticular abscesses respond favorably to treatment.
 - Hepatic abscesses must be drained by a second surgery. Large-bore cannulas placed through the body wall, through the adhesions, and into the abscess will drain the pus. However recurrence is more of a problem with hepatic abscesses than with reticular abscesses.
 - A diagnosis of lymphosarcoma at surgery warrants a grave prognosis and identification of adhesions in the vicinity of the reticulum warrants a fair to good prognosis with surgery, antibiotic therapy, and appropriate supportive treatment.
- 3. Animals with type III vagal indigestion (abomasal impaction)**
- If the diagnosis is made at surgery or if the abomasum impaction is thought to be dietary diacyl sodium sulfosuccinate or magnesium sulfate can be infused directly into the abomasum via the reticulo-omasal orifice after emptying the rumen.
 - Anasogastric tube can be passed into the abomasum at surgery and left in place for continued treatment.
 - Abomasotomy and removal of abomasum contents using a right paracostal approach with the cow in left lateral recumbence can be performed as a last resort. However, recurrence of the impaction is common.
 - Pyloric obstruction in cattle is rare and is most often due to a foreign body obstructing the lumen.

Pylorotomy is almost never effective in resolving abomasum impactions.

4. Type IV vagal indigestion (partial fore stomach obstruction),

- Therapeutic abortion has been recommended for treatment of cattle with type IV vagal indigestion (partial fore stomach obstruction), and some cows have improved with this treatment; however, because type IV vagal indigestion is a poorly defined condition, the prognosis is always guarded.
- A more specific prognosis is based on response to therapy and identification of a specific lesion at exploratory celiotomy and rumenotomy.

N.B

- Cattle with secondary impactions due to traumatic reticuloperitonitis or as a sequel of right abomasal displacement or abomasal volvulus seldom recover.
 - Animals with foreign bodies (e.g, phytobezoars) obstructing the pylorus have a good prognosis if the obstruction is removed.

Prevention

The most common cause of vagal indigestion is traumatic reticuloperitonitis, which causes adhesions and abscesses that interfere with vagal nerve function. Therefore, prevention of traumatic reticuloperitonitis is important.

- Good management practices will prevent some cases of vagal indigestion resulting from chronic pneumonia.
- Early diagnosis of right-sided abomasal displacements and abomasal volvulus, and surgical correction the day the diagnosis is made, may prevent some cases.
- Prompt removal of the placenta from the cow's enclosure after parturition will keep it from obstructing the cardia, reticulo-omasal orifice, or pylorus.

Abomasal Displacement;

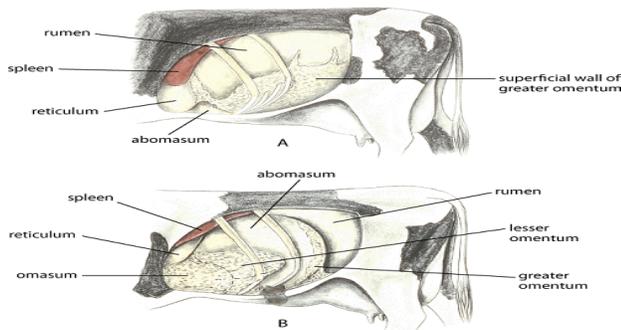


Figure 32.A). Normal topography of left abdominal viscera, cow. B) Left displacement of abomasums. Illustration by Dr. Gheorghe Constantinescu. Adapted, with permission, from DeLahunta and Habel, *Applied Veterinary Anatomy*, W.B. Saunders, 1986.

The abomasums (true stomach) normally lies on the floor of the abdomen but can become filled with gas and rise to the top of the abdomen when it is said to be 'displaced'. The abomasums is more likely to be displaced to the left (LDA) than the right (RDA), (figure 32). Cattle have a specialized digestive system that helps them process their high fiber diet where they have four distinct compartments to their stomachs, the rumen, reticulum, and omasum are the fore stomach compartments. The abomasums is the "true" stomach of the cattle and carries out similar function to the stomach in humans. The abomasums is suspended by a loose attachment which means it can potentially move out of its normal position where it can fill up with gas and prevent normal flow of feed through the digestive track (figure 32). Abomasums displacements typically occur in high production dairy cows.

Causes

No one knows exactly what causes the abomasums to move out of its normal position.

- **Calving:** The majority of cases occur soon after calving. During pregnancy the uterus displaces the abomasums, so that after calving the abomasums has to move back to its normal position, increasing the risk of displacement.
- **Atony of the abomasum:** If the abomasums stops contracting and turning over its contents, accumulation of gas will occur and the abomasums

will tend to move up the abdomen. This tends to be a cause of inadequate nutrition.

- Three types of commonly seen abnormal positions include:

Left Displaced Abomasums (LDA), most common;

➤ **Right Displaced Abomasums (RDA).**

➤ **Abomasal "volvulus"** (twisting) on the right side (RVA).

The abomasums can also twist around itself (called an abomasal volvulus), which can compromise blood supply to the abomasums and the affected cows become much more ill much more quickly.

Clinical signs

Up to 90% of LDAs occur within the first 4 weeks after calving. Affected animals go off feed and become depressed. Producers will frequently notice a drop in appetite and reduced milk production. Symptoms often resemble ketosis with ketones in blood, milk, breath and urine

Animals with right displacement can show more severe signs including colic, elevated heart rate scant feces and diarrhea; if torsion occurs, animals can go downhill very rapidly showing signs of severe shock with cold extremities and extreme dullness.

Abomasal Displacement;



Figure 33; B. indicated tympani of lower left abdomen in cows with a displaced abomasums.

- Loss of appetite, decreased manure production, decreased milk production, mild colic, hypotonic rumen, Cold ears and widely dilated pup and tymani of the lower abdomen "slab-sided" abdomen (figure 33).

Abomasal Volvulus.

- Dull, depressed, loss of appetite and high heart rate.
- Large amounts of gas and fluid collection on the right side.

- Minimal manure production and dehydration.

Diagnosis;



Figure. 34. LDA: Ping & Splash As cult and per cuss .Ping high pitched. Ballotement for splash of fluid. All pings are not created equal – rumen ping.

- Accurate clinical signs
- Rectal examination (can't)
- Simultaneous "auscultation and percussion" or "pinging" of the abdomen. A pinging noise is heard when there is a gas-filled organ up against the body wall, such as a displaced abomasums (figure 34).
- Passage of a nasogastric tube, through the nose into the stomach, to test the fluid from the rumen.
- Blood analysis.
- Ping the left side of the cow by simultaneously percussion and auscultating the whole left side of the cow by firmly flicking your finger against the body wall of the cow. A "ping" represents a fluid-gas interface. On the left side of the cow, gas may be present in the abomasums (LDA), rumen, or peritoneal cavity. LDA pings are variable in tone, and often tinkling sounds are heard over the area of the LDA. Rumen pings are located over the rumen area and tend to be monotone. LDA and rumen pings often occur together and are usually distinguished by two distinct pings of different tones. The most confusing ping for us on the left side of the cow, is a monotone ping that extends over the rumen and LDA area. Often, this ping is accompanied by a small gas-filled rumen on rectal palpation and is referred to as a "rumen-void" ping. These cows do not have surgical problems (figure 34),
- Ping the whole right side of the cow carefully. Pings on the right side are confusing, so care must be taken to delineate the borders of the ping. On the

right side of the cow, gas may be present in the cecum, spiral colon, small intestine, duodenum, uterus (after calving), peritoneum, rectum, and abomasum (RDA or RTA). Pings on the right side can be confusing, and location and repeatability of the ping are probably the most useful indicators as to the organ involved. While most pings on the left side of the cow are LDA most pings on the right side of the cow are from spiral colon and cecum. Commonly, cows that are sick, especially with GI disturbances have pings in the right paralumbar fossa. A distended or displaced cecum will produce a consistent, large ping that is always palpable per rectum. RDA and RTA (right abomasal torsion) pings are usually cranial to the 8th to 10th rib. The RDA/RTA pings on the right side are more cranial in location compared to the LDA ping on the left side, which can be heard as far caudal as the cranial aspect of the left paralumbar fossa. Most right-sided abomasal problems have some degree of torsion associated with them, but the overall condition of the cow (heart rate, and degree of dehydration) indicates the severity and degree of torsion of the abomasums. Ballot the right abdomen to detect any abnormal mass or fetus (figure 34).

Treatment

The goal of treatment for abomasal displacement or volvulus initially includes stabilizing the cow with fluid therapy, calcium and/or dextrose (sugar) solutions and then to move or replace the abomasums to a normal position, Prevent it from displacing again and keep the procedure a traumatic for the cow.

Closed (Nonsurgical) Techniques for Abomasal Displacement

Less expensive and relatively quick and simple techniques to perform include:

- **Rolling:** Flipping the abomasums back in place (not an effective long-term treatment because most cows will have a recurrence).
- **Securing the abomasums** to the body wall with a blind tack (a holding stitch placed in the abomasums without opening the belly) or a toggle pin (a special device to fix the abomasums in place). Both blind tacking and toggle pin techniques prevent recurrence of the displacement, but they can have very high

complication rates, including infection or damage to internal organs.

- **Laparoscopic placement**(of a small camera inside the abdomen to see what you are doing) can avoid these complications.

Open (Surgical) Techniques for Abomasal Displacement:

- Surgery for an abomasums displacement can be performed with the cow standing or lying down depending on the veterinarian's preference, temperament of the cow, its physical condition, and the surgery facilities that are available. Veterinarian may refer you to an ACVS board–certified veterinary surgeon.
- An incision is made into the abdomen through the side or the bottom of the abdomen to suture the abomasums to the body wall internally.

Open (Surgical) Techniques for Abomasal Volvulus

- The only option for cows with an abomasal volvulus is to undergo an open, surgical procedure that includes: Evaluating the blood supply to the abomasums, returning it to its normal position and securing it to the body.

Prevention

Prevention should be aimed at ensuring dry matter intake is maintained in early lactation:

- Ensure cattle are not too fat at calving (i.e. >3.5 BCS);
- Feed high quality feeds, with good quality forage;
- Feeding a total mixed ration as opposed to concentrates;
- Ensure plenty of space at feeding sites;
- Minimize changes between late dry and early lactation ration;
- Prevent and promptly treat, diseases such as milk fever, metritis, toxic mastitis and retained afterbirth which reduce feed intake;
- Maximize cow comfort, minimize stress.
- It is likely that a farm with numerous DA problems is feeding the late dry and/or early lactation cows wrongly.

Bacterial Disease; Pneumonia

Pneumonia is an acute or chronic inflammation of the lungs and bronchi characterized by disturbance in respiration and hypoxemia and complicated by the systemic effects of associated toxins. Pneumonia is a common problem, particularly in calves during the housing period.

Etiological agents

- Most cases of pneumonia are caused by bacterial, viral and parasitic infection.
- Injury to the bronchial mucosa and inhalation or aspiration of irritants may cause pneumonia directly and predispose to secondary bacterial invasion.
- In many cases, high humidity, dust, dumb bedding, excessive heat, tight buildings with inadequate ventilation and irritating gases such as ammonia compromise disease resistance and natural defense mechanism.

Bacterial Causes

Pasterulla haemalytica

Pasterulla multocida

- Bacterial organisms carried in the respiratory tract of many animals.
- Most newborn are exposed to these organisms, but don't develop the disease because of natural resistance, healthy environment and ingestion of antibodies in colostrums. That helps control the infection.

Viral agent

Parainfluenza–3 (PI3) is common

Parasitic agent

Parasitic invasion of the bronchi as by

1. Dictycaulous. spp (Lung worm).
2. *Eiemira species* can also causes lung problems in animals may result in pneumonia. Protozoan parasite e.g., *Toxoplasma gondii*. 3

Aspiration pneumonia

Usually occur in animals during drenching. Aspiration pneumonia may result from persistent vomiting, abnormal esophageal motility, or improperly administered medications (e.g., oil or barium) or food forced feeding. It may also follow suckling in a neonate with a cleft palate.

Clinical signs;



Figure 35...Nasal discharge from calves showing cough and opening nos.

- Fail to suck with moderate fever in young nursing animals that develop pneumonia.
- Clear to yellow, runny to thick nasal discharge, coughing and/ or rapid breathing. –Harsh lung sounds heard when listening with stethoscope and fever
- Young animals that recover are susceptible to relapse during the feeding period and are more likely to suffer from heat stress. If the pneumonia remains undetected, serious lung damage will result and treatment will not be effective as illustrated in (figure 35).

Disease Transmission

Most of the infectious organisms are spread by direct contact with body fluids (saliva, nasal discharge ...etc) and fecal material. These problems can also be transmitted from one animal to another by contaminated hands, buckets and feeders.

Diagnosis

The following list contains some key area to help determine a diagnosis.

1. Accurate clinical signs, harsh lung sound when using stethoscope.
2. Culture and sensitivity test performed on nasal secretion or on samples taken at necropsy can help identify the specific cause of the infection.
3. Transtracheal was performed by a veterinarian with culture and sensitivity test may be necessary.

Treatment

Treatment must be based on early identification of affected individuals and depends on whether the cause is bacterial, viral or parasitic.

- **Immediately**
 - Fluid therapy (Normal saline or Ringer solution) often helps the recovery rate.

- The owner should be sure that sick newborns are nursing or that they are provided supplement milk via stomach tube.
- **Pneumonia due to Bacterial Causes**
 - Culture and sensitivity testing is recommended for the perfect antibiotic.
 - Treatment with antibiotics such as (Penicillin, Tetracycline, Gentamycine) and even long acting antibiotic.
- **Pneumonia due to Viral Causes**
 - Treatment for all viruses' infection involves treating the symptoms, not killing the virus with this in mind.
 - Anti-inflammatory agent (Dexamethasone) and broad-spectrum antibiotic for secondary bacterial infections are recommended.
- **Pneumonia due to Parasitic Causes**
 - Most parasites can be treated using anti-parasitic drugs.
 - Routine de-worming will also help prevent the parasitic causes of pneumonia.(see verminous pneumonia)

Prevention

- Good management is the key to preventing respiratory problems.
- Most commonly the areas lack ventilation resulting in high humidity and noxious gases such as ammonia.
- Well-ventilated (but cool) housing is probably ideal.
- New born animals should be provided supplemental heat only until they are dry and have nursed.
- Avoid overcrowding and don't keep the birthing area too warm.

Hypersomnia in Bull;[Infectious Septicemia Thrombosing Meningoencephalities (ISTME)]

Hypersomnia is a disease of the brain and spinal cord which is caused by bacteria. Its scientific name is infectious septicemia thrombosing meningoencephalities (ISTME).

Incidence

The disease is observed particularly in the autumn and winter and mainly in beef cattle weighting 200–400kg. Stress condition like transport, re-housing, crude fiber deficiency and vitamin B1 deficiency favor the

appearance of the disease, 90% of the affected animals die of the disease.

Causative agent

The causative organism (*Haemophilus somnus*) is present in many beef cattle stock, but the disease only appears through the effect of stress factors. The pathogen can be isolated from the brain and cerebrospinal fluid of animals that have died.

Clinical signs

The clinical symptoms and the progress of the disease depend on the site and the extent of the brain damage and therefore differ from case to case where the disease appears in two stages.

The first stage

Reduce food intake, the animal stands about sleepily, the animal appears as if tired. Stand listlessly in the pen, tend to lie down or wander about restlessly.

- Aqueous nasal discharge and bouts of coughing may be observed in some bulls.
- The body temperature is within or slightly above normal limits.

The second stage

After 10–40 hours the animals lie down all the time showing central nervous system, initially the bull lies on the chest with one or both forelegs stretched forward.

- The head turned sideways and the eyes half closed.

In the Advanced Stage

The animal lies on the side with stretch or slightly bent legs, the body temperature of the animal lying down is below normal, paralysis of the tail, the anus and the rectum is observed and the mortality rate is very high.

Diagnosis

- The clinical signs and case history
 - Because of the inflammation, the spinal cord is under increased pressure. This is why the cerebrospinal fluid escapes spontaneously on puncture; the fluid is yellowish white turbidity and coagulates after short time
- **Laboratory examination**
 - Indicated increase in the protein content to above 300mg/100ml (normal animal level; 10–40mg/100ml).
- **At autopsy;**
 - The typical changes caused by the disease 'Thromboses' are found in the brain.
 - **Differential Diagnosis**

The nervous signs seen are also observed in vitamin B1 deficiency, listeriosis, warble fly infestation and phosphoric acid ester poisoning. The most important distinguishing characters are.

Vitamin B1 deficiency.	➤ Unsteady gait with splayed leg, head raised upward and backward (stargazing).
	➤ Early treatment with vitamin B1 produced rapid improvement.

Listeriosis	➤ –Raised body temperature.
	➤ –Animal wandering about in circling.
	➤ The head is always turned to the same side.
	➤ The animal dies within 4–14 days.

Lameness due to warble fly infestation.	➤ –Migrating warble-fly larva in the vertebral canal may cause lameness in the hind legs.
	➤ At autopsy. Warble-fly is found in the spinal canal.

Poisoning with phosphoric acid esters.	➤ Phosphoric acid ester used in treatment of mange and warble-fly infestation.
	➤ In case of overdose treatment, diarrhea with admixture of blood and colic occur within 2–24 hours after treatment.
	➤ Frequent urination and salivation are typical.

Treatment

1st– 2nd days	➤ 3 ml Ampicilline/10 kg bodyweight intramuscular at interval period is 12 hours.
	➤ Intramuscular injection of 1ml vitamin B complex.
	➤ All animals of the same age are taken 1 bolus of sulfamethazine/150 kg bodyweight by mouth in water.
1st– 21 days	➤ The animals of the same age are given 10 gm (chlortetracycline + sulfadimidine + vitamin A and B)/ animal/day.

Tetanus

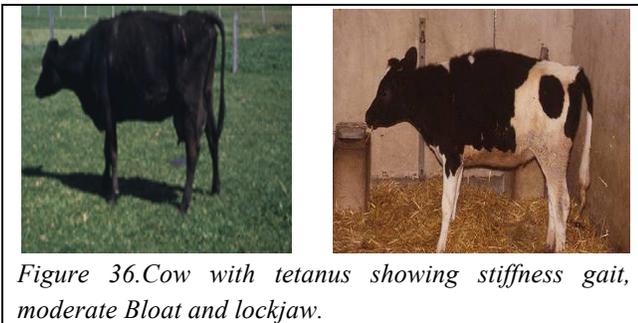
Tetanus is a fairly common disease occurring in all animals of livestock. It is relatively rare in cattle, but cattle can get tetanus. Tetanus is a highly fatal disease caused by

toxins produced by the bacterium called, *Clostridium tetanus*, generally found in soil, feces of domestic animals, dust and contaminated environment. The disease starts when the organism gets into wounded or damage tissue as a result of contamination.

Etiology

This organism is widespread in the environment but only rarely causes disease in cattle. Tetanus usually occurs when an animal has had a penetrating wound, has been castrated or tail docked, or had a difficult calving. It can take days to weeks from receiving the original wound until the appearance of tetanus. The tetanus organism must enter the wound and be sealed off from the air before it can multiply. It then produces a powerful toxin that targets the nerves responsible for muscle movements.

Clinical Signs;



The incubation period varies from one to several weeks but usually averages 10–14 days.

1. Localized stiffness often involving the masseter muscles and muscles of the neck, the hind limbs, and the region of the infected wound is seen first, general stiffness becomes pronounced 1 day later and tonic spasms and hyperesthesia become evident because of their high resistance to tetanus toxin, dogs and cats often have a long incubation period and frequently develop localized tetanus; however, generalized tetanus does develop in these species.
2. The reflexes increase in intensity, and the animal is easily excited into more violent, general spasms by sudden movement or noise. Spasms of head muscles cause difficulty in prehension and mastication of food hence the common name, (lockjaw).

3. The temperature remains slightly above normal, but it may rise to (40–41°C) toward the end of a fatal attack. In mild attacks the pulse and temperature remain nearly normal. Mortality averages 80%. In animals that recover, there is a convalescent period of 2–6 wk; protective immunity usually does not develop after recovery.

Many signs will alert the farmer to the existence of tetanus in cows. Abnormalities in movement begin to show first such as reluctance to move stiffness and an unsteady gait. The tail can go stiff, and lockjaw is a big danger sign.

4. The cattle may become restless and excitable. Their muscles may begin twitching and the cattle could become bloated. In later stages, the cattle fall over and begin to spasm with their legs held out stiffly; death usually follows soon after over-reaction to sudden sounds or movements as illustrated in (figure 36).

Diagnosis

The clinical signs and history of recent trauma are usually adequate for a diagnosis of tetanus. It may be possible to confirm the diagnosis by demonstrating the presence of tetanus toxin in serum from the affected animal. In cases in which the wound is apparent, demonstration of the bacterium in gram-stained smears and by anaerobic culture may be attempted.

Treatment

➤ Immediately

- Tranquilizers or barbiturate sedatives drug for animal to avoid asphyxia.
- Animal administered with 300,000 IU of tetanus antitoxin.
- Draining and cleaning the wounds and administering penicillin or broad-spectrum antibiotics.
- Good nursing is invaluable during the acute period of spasms.
- A combination of chlorpromazine and Phenobarbital may be used to reduce hyper esthetic reactions and convulsions.
- Cattle with early tetanus probably respond to treatment better than most other livestock.
- High doses of penicillin may be helpful.
- Local treatment of the infected site.

Prevention

Vaccines are available to protect against bovine tetanus which can last as long as three years if given in the

correct doses at the correct times. These are usually used in areas of high risk. In these cases, vaccines are seen to prove economically worthwhile to farmers when compared to if the cattle were to actually become infected. Symptoms of the disease are generally milder in cattle that have vaccinated as opposed to those that have not.

In other cases, good hygiene is needed especially during and following operations such as castrations and calving. The spread of the disease can be reduced by ensuring that all surgical equipment is sterilized and the operation is undertaken in a clean area. Barbed wire which has rusted should be replaced as these can also act as a source of infection and any open wounds cleaned immediately.

Antitoxin can be useful as short-acting (up to 21 days).

Mastitis

Mastitis is the inflammation of the mammary gland and udder tissue, and is a major endemic disease of dairy cattle. It usually occurs as an immune response to bacterial invasion of the teat canal by variety of bacterial sources present on the farm, and can also occur as a result of chemical, mechanical, or thermal injury to the cow's udder.

The inflammatory response consists of an increase in blood protein and white blood cells in the mammary tissue and the milk. The purpose of the response to destroy the irritant repair the damaged tissue and return the udder to normal function.

Milk-secreting tissues and various ducts throughout the udder can be damaged by bacterial toxins, and sometimes permanent damage to the udder occurs. Severe acute cases can be fatal, but even in cows that recover there may be consequences for the rest of the lactation and subsequent lactations.

Etiological causes.

Infectious agent;

Chief pathogens include, streptococcus agalactia, staphylococcus aureus. Yeast can also cause mastitis over use of antibiotics and poor sanitation contributes to yeast mastitis.

Non Infectious Agent

- Improper use of milking machine may be lead to irritations and incidence of mastitis.
- Incidence of mastitis increase with age.

Types of mastitis

Mastitis may be classified according two different criteria: either according to the clinical symptoms or depending on the mode of transmission.

1. Clinical symptoms;

- Clinical mastitis
- Sub-Clinical mastitis

2. Mode of transmission;

- Contagious mastitis
- Environmental mastitis.

Risk factors for mastitis;

Infectious organism, characteristic of the cow, milking methods cow and environmental condition.

Nutrition (↓ of vitamin E and selenium associated with ↑ rate of new mammary infection).

The clinical signs;

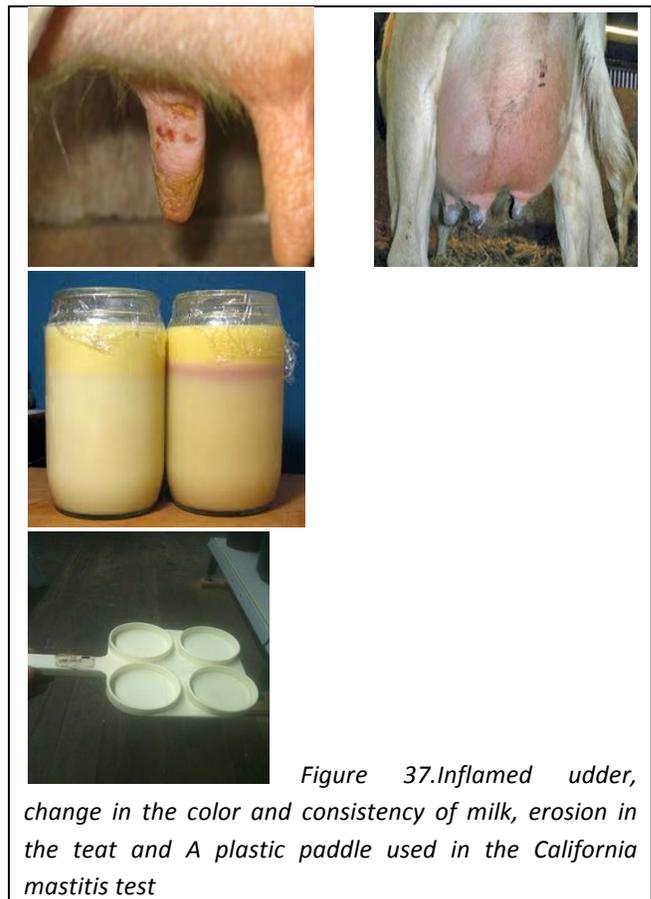


Figure 37. Inflamed udder, change in the color and consistency of milk, erosion in the teat and A plastic paddle used in the California mastitis test

Clinical signs illustrated in (figure 37);

- Gross abnormalities in the udder (swelling, redness and pain).
- Persisting inflammation lead to tissue damage and replacement of secretory tissues with of the udder with nonproductive connective tissue.
- Change in composition and appearance of milk. Abnormalities in milk may includes flakes, clots or a watery appearance .The flakes in the milk are congealed, leucocytes, secretory cells and protein.
- A lower milk yield.

Type of mastitis

Clinical Mastitis

Clinical mastitis is an inflammatory response to infection causing visibly abnormal milk (e.g. color .fibrin clots).The extent of the inflammation increase, changes in the udder (swelling,hotness,pain and redness) may also be apparent .If the inflammatory response includes systemic involvement (fever,anorexia and shock).

Any number of quarters can be infected, Gangrenous mastitis can also occur).

Subclinical mastitis

The presence of an infection without apparent signs of local or systemic inflammation.Although transient episodes of abnormal milk or udder inflammation may appear.

Diagnosis

Diagnosis is best done by examination of milk somatic cell counts (predominantly neutrophils) using either the California mastitis. Causative agents must be identified by bacterial culture of milk.

Controlling the amount of mastitis in the herd

The amount of mastitis in herd at any time depends on the new infection rate and the duration of infection. Prevention of new infection or decreasing the new infection rate is always the most economical approach to mastitis.

Treatment

The treatment protocols should be described the condition to be treated, the drug to use, its dosage and route of administration and the milk and meat withdrawal.

- **Immediately**
 - Wash hand with soap and water, wash teats and udder with sanitation solution (alcohol), thoroughly

dry teats and udder with individual towels prior to intramammary infusion.

- **For milking cow**
 - Use commercial antibiotic products in single dose containers formulated for intramammary infusion.
- **For dry cow therapy**
 - Use commercial antibiotic products specifically formulated for dry cow therapy in single dose.
 - Oxytocin treatment

Injection of oxytocin to stimulate an effective milk letdown, clearly removal of the primary growth medium of the bacteria.

➤ **Non-Responding Cases**

- Antibiotic treatment to help fight bacterial infection, and other methods such as frequently stripping out the milk, chronically infected cows sometimes is the only way to effectively control spread of mastitis in the herd.

Prevention

The 6 Point Plan for mastitis prevention

1. Hygienic teat management: which includes good housing management, effective teat preparation and disinfection for good milk hygiene, teat health and disease control.
2. Prompt identification and treatment of clinical mastitis cases: including the use of the most appropriate treatment for the symptoms.
3. Dry cow management and therapy: where cows are dried off abruptly and teats are cleaned scrupulously before dry cow antibiotics are administered, including the use of teat-end sealants if appropriate.
4. Culling chronically affected cows: cows that becomes impossible to cure and represent a reservoir of infection for the whole herd.
5. Regular testing and maintenance of the milking machine: with regular, recommended teacup liner replacement and milking machine servicing and attention paid to items which must be checked on a daily, weekly or monthly basis.
6. Good record keeping: of all aspects of mastitis treatment, dry cow therapy, milking machine servicing, Somatic Cell Counts and clinical mastitis cases.

PARASITIC DISEASE

Verminous Pneumonia (Lungworm Infestation)

An infection of the lower respiratory tract usually resulting in bronchitis or pneumonia can be caused by any of several parasitic nematodes including *Dictyocaulus viviparus* in cattle. Adult females in the bronchi lay larvae eggs that hatch in the bronchi. The infective third-stage larvae can develop on pasture within 5–7 days in warm, moist conditions but typically in summer in temperate northern climates will require 2–3 wk, mild to severe dyspnea and fever in domestic animals due to the presence in the bronchioles or lungs of nematode lungworms, usually of species of *Dictyocaulus*, *Filaroides*, *Metastrongylus*, *Muellerius*, or *Protostrongylus*. Acute episodes may be fatal. In sheep, cattle, goats, and pigs it is called (*hoose*).

Incidence

The infestation occurs particularly on moist pasture. Since such pastures offer favorable conditions for other parasites. The cattle lungworm *D viviparousis* the cause of “husk” or “hoose” in young (and more recently, older) grazing cattle.

Pathogenesis

The pathogenic effect of lungworms depends on their location within the respiratory tract, the number of infective larvae ingested, and the animal's immune state.

Pre-patent Phase

The main lesion is blockage of bronchioles by an infiltrate of eosinophils in response to the developing larvae; this results in obstruction of the airways and collapse of alveoli distal to the block. Clinical signs are moderate unless large numbers of larvae are ingested, in which case the animal may die in the prepatent phase with severe interstitial emphysema and pulmonary edema.

Patent Phase

The adults in the segmental and lobar bronchi cause a bronchitis, a cellular exudates, frothy mucus, and adult nematodes are found in the lumen where the bronchial irritation causes marked coughing. A major component of the patent stage is development of a chronic, nonsuppurative, eosinophilic, granulomatous pneumonia in response to eggs and first-stage larvae aspirated into alveoli and bronchioles.

Death may result where. Interstitial emphysema, pulmonary edema, and secondary bacterial infection are

complications that increase the likelihood of death. Survivors may suffer considerable weight loss.

Clinical Signs;

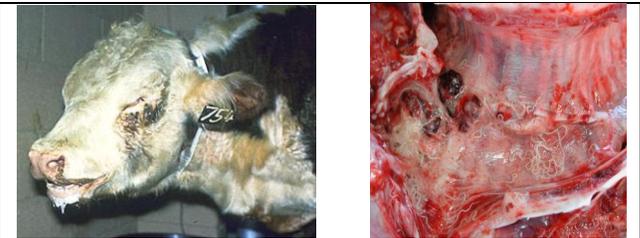


Figure 38. Lungworm (*Dictyocaulus viviparus*) infestation of the trachea Calf with extended neck, hunger air, respiratory distress.

- The first clinical signs of lung worm infestation usually appear 4 week after the animals have taken the infection (figure 38).
- Signs range from moderate coughing with slightly increased respiratory rates to severe persistent coughing and respiratory distress.
- Reduced weight gains, reduced milk yields, losses of appetite, dullness, nasal discharge and high temperature up to 41c may occur.
- Heavily infected animals stand with their heads stretched forward and mouths open and drool. Lung sounds are particularly prominent at the bronchial bifurcation.

Diagnosis;

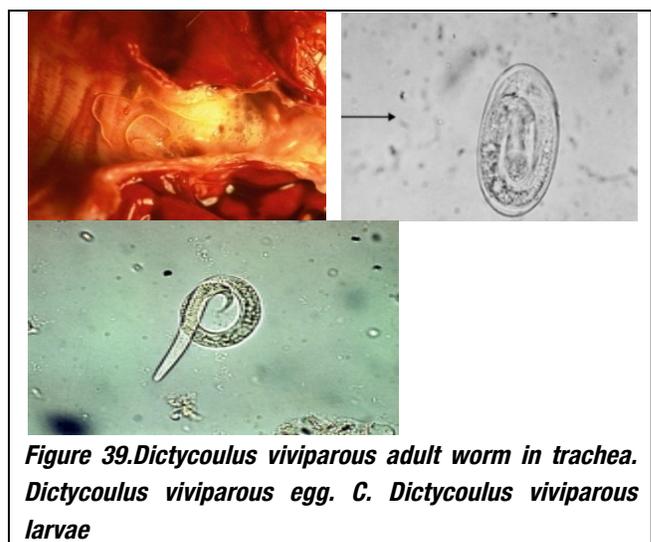


Figure 39. *Dictyocaulus viviparus* adult worm in trachea. *Dictyocaulus viviparus* egg. C. *Dictyocaulus viviparus* larvae

- **Clinical Signs and Case History**

- **Laboratory examination:** The fecal sample from animals that cough and show reduced weight gains should be examined for lung worm larvae according to Baermann/Wetzel is used, in which large fecal samples (25–30 g) are wrapped in tissue paper or cheese cloth and suspended or placed in water contained in a beaker. The water at the bottom of the beaker is examined for larvae after 4 hr; in heavy infections, larvae may be present within 30 min. where the larvae can then be observed under the microscope (figure 39).
- Post-mortem examination; as in figure (38).

Treatment;

Immediately;

- De-worming the infected animals by injection of anti-parasitic drug as. The benzimidazoles (fenbendazole, oxfendazole, and albendazole) and macrocyclic lactones (ivermectin, doramectin, eprinomectin, and moxidectin) are frequently used in cattle and are effective against all stages of *D. viviparus*.
- Levamisole is used in cattle but treatment may need to be repeated 2 wk later because it is less effective against larvae during the early stages.

Note

- Not more than 10ml per injection site and not more than 20 ml per animals.

Prevention and Control

- All contact animals carried out of parasitological examination.
- Anthelmintic prophylaxis has become feasible with the advent of anthelmintics with prolonged activity (e.g., ivermectin, doramectin, moxidectin, eprinomectin). With persistent anthelmintics, two or three treatments during the grazing season.

Fascioliasis(Liver Fluke Infestation)

Fasciolosis is a common parasitic disease of cattle in many countries worldwide caused by *Fasciola hepatica*. Infection is more commonly encountered in beef cows grazing poor wet pasture but disease can be seen in dairy cattle. Liver flukes live as a parasite in the bile duct of the liver where they damage the liver itself and cause changes in the bile ducts.

Incidence

Liver fluke disease is a problem in areas where the cattle are kept on moist pasture so the disease occurs especially in low-lying regions. The animals are particularly at risk during the first grazing period.

Cause

Fasciola hepatica has the liver as its site of infection in cattle. During the spring/early summer months the intermediate stages infect snails (*Limnaea truncatula*), which need moisture and an environmental temperature above 10°C. In the late summer/autumn, challenge to cattle causes disease several months later with the severity depending upon the level of challenge.

The life cycle of the large liver fluke comprises two stages (figure 40).

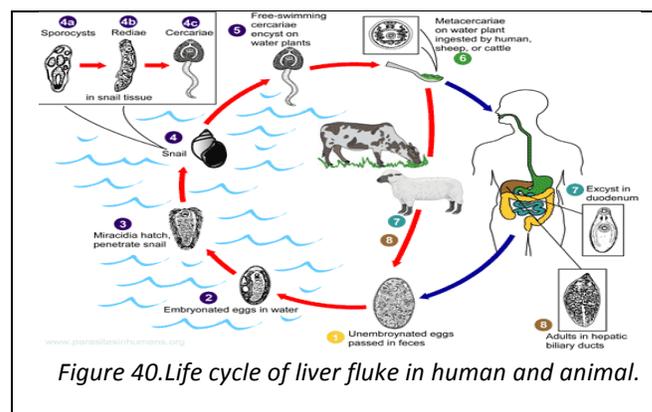


Figure 40. Life cycle of liver fluke in human and animal.

A. Development in the environment and in the intermediated host.

- In a moist environment, the fluke eggs excreted in the faces develop into miracidia within 3–4 weeks. The larvae penetrated into the snails where they multiply asexually, one miracidium produce about 250 caudate larvae (cercaria) which leave the snail and attach themselves to wet grass and formed encapsulated larvae.

B. Development in cattle.

The cattle ingested the encapsulated larvae with the grass and the capsule is dissolve in small intestine and metacercaria are released and penetrated the intestinal wall into the abdominal cavity and migrated the liver. They penetrate the liver capsules and invade the liver where young liver flukes move about the liver for 6–8 weeks and then invade the bile ducts where they become sexually mature.

Clinical Signs;



Figure 41. Tarry appearance, persistent diarrhea, bottle jaw, and dullness, rough coat, and emaciation.

During a wet summer, grazing cattle ingest the intermediate stages of the fluke from contaminated pasture with invasion of the liver causing disease during the winter months. The major presenting clinical findings are persistent diarrhea and chronic weight loss with resultant poor condition as illustrated in (figure 41).

The resulting bile engorgement may cause jaundice which manifested itself by yellow discoloration of the mucosa.

Bile acids are necessary for fat digestion, so disturbed bile flow leads to intermediated diarrhea, changing appetite, dullness, rough coat, and emaciation.

Diagnosis;

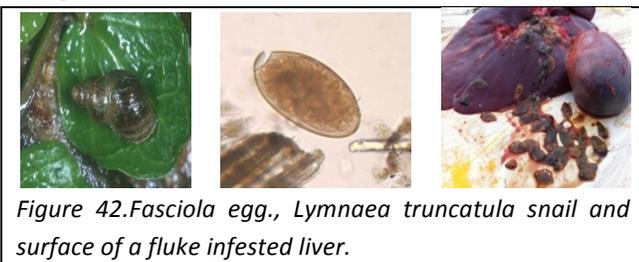


Figure 42. Fasciola egg, Lymnaea truncatula snail, and surface of a fluke-infested liver.

- Accurate clinical signs and case history
- Parasitological examination carried on fecal samples from diseased animals and the oval, operculated, golden brown eggs (130–150 × 65–90 μm, figure 41), must be distinguished from paramphistomes egg (rumen flukes), which are larger and clear.
- Eggs of Fasciola hepatica cannot be demonstrated in feces during acute fasciolosis but in sub-acute or chronic disease cattle, the number varies from day to day, and repeated fecal sedimentation may be required

- At necropsy, adult flukes are readily seen in the bile ducts, and immature stages may be squeezed or teased from the cut surface (figure 42).

Treatment

A number of products are available for treating fluke in cattle where flukicides are effective against immature and adult fluke.

1. Triclabendazole;

Effective at killing all stages of triclabendazole susceptible flukes from two weeks old.

2. Nitroxylin;

Licensed for the treatment of fascioliasis (infestation of mature and immature stage).

3. Clorsulon

Effective against adult flukes.

➤ N.B

- The interval between Nitroxylin treatments must not be less than 60 days. Cattle may be slaughtered for human consumption only after 60 days from last treatment. Do not use in cattle producing milk for human consumption.
- Cattle may be slaughtered for human consumption only after 56 days from last treatment, do not administer to cows producing milk for human consumption.
- Intensive use or misuse of preparations such as Triclabendazole can give rise to drug resistance with reduced efficacy of the preparation.
- The recovery of chronically infected cattle is slow following treatment with a flukicide. Improved nutrition of affected cattle is essential to restore body condition and production. Treated cattle should be moved to clean pastures wherever possible. All withhold times etc. can change so always check the data sheets for the latest information.

Prevention/Control Measures

- In areas with endemic fasciolosis, control is founded upon strategic flukicide treatments outlined in the veterinary herd health plan.
- Where cattle are in-wintered a single dose of a flukicide, effective against appropriate stages, should be given around housing time in accordance with the farm's veterinary herd health plan – this will highlight the most appropriate treatment for that specific farm. All purchased cattle

should be treated with a flukicide, the choice of which depends upon time of the year and local veterinary advice, before joining the herd. During a low risk year, treatment to kill mature flukes is given to at-risk cattle from January. In years when epidemiological data indicate a high risk of fasciolosis, a treatment with a flukicide effective against immature should be given at housing and if necessary more than 8 weeks after the first treatment.

- A third treatment in January/February with a different drug may be required to remove adults which have subsequently developed. From any early stages, which were not susceptible to the previous flukicide treatments?
- Fecal samples may be taken to help confirm if such treatments are necessary.
- Milk withdrawal periods in dairy cattle may determine the timing for treatments and drug used, with treatment given during the dry period in dairy herds – consult your veterinary surgeon for the best advice.

Gastrointestinal Parasitic Infestation

The common stomach worms of cattle are *Haemonchus placei* (barber's pole worm, large stomach worm, wire worm), *Ostertagia ostertagi* (medium or brown stomach worm), *Trichostrongylus axei* (small stomach worm), *Cooperia Spp* and *Nematodirus Spp*.

Incidence

Grazing cattle virtually always show slight infestation with gastrointestinal worms due to the delayed development (hypobiosis) of the worm in the winter while animal suddenly start to excrete many worm eggs in the spring but young cattle are particularly affected.

- Conditions when gastrointestinal worm infections are likely to occur. High rainfall regions with more than 500–600 mm annual rainfall or on irrigation, high stocking rates or set stock grazing situations, Livestock grazing short pastures.
- Young animals, especially after weaning (cattle up to 15 months of age should be considered higher risk). Rising 2–3 year-old cows (are at higher risk of developing type 2 ostertagiasis in summer and

autumn, especially in association with calving or nutritional stress).

- Bulls and bull beef production systems (are higher risk because bulls have lower immunity to gastrointestinal parasites).
- Hot and humid conditions for Barber's pole worm (Barber's pole worm is more common in summer rainfall region, while black scour worm and brown stomach worm larvae are more of a problem in winter rainfall regions).

Pathogenesis and Life Cycle;

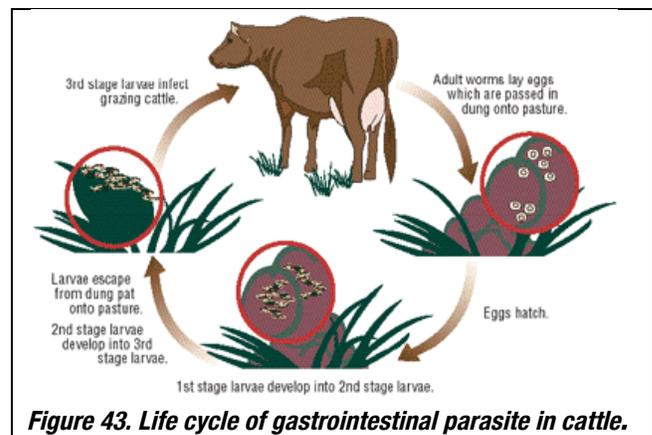


Figure 43. Life cycle of gastrointestinal parasite in cattle.

life cycles of the all parasites are generally similar where larvae hatch shortly after the eggs are passed in the feces and reach the infective stage within 2–4 week under optimal temperatures (24°C), as in figure (43).

Development to the infective stage is delayed during cold weather. In areas with narrow diurnal temperature variations, those months with a mean maximum temperature of (18°C) and with rainfall are favorable for development of the free-living stages, but where wide fluctuations occur a mean minimum temperature of (10°C) may effectively limit development. The infective larvae ingested by the grazing cattle and mature via two further skins in the rumen, the abomasums and small intestine. The larvae of the species *Ostertagia*, *Trichostrongylus* and *cooperia* lie dormant in the intestinal mucosa from the autumn to the spring. This carry their further development and the worm eggs are not excreted in large number until the animals are put out to graze. Young cattle being put to postures for the first time are particularly susceptible to infection with larvae

hatched from these eggs and with larvae that have hibernated on the pasture. For this reason young cattle excrete large number of gastrointestinal worm eggs when the worms have reached sexual maturity.

Clinical signs

- Worm infestation of cattle is most pronounced.
- Rough coat despite excellent fodder.
- Retarded growth, emaciation and diarrhea.
- Debility is the most clinic syndrome.

Diagnosis;

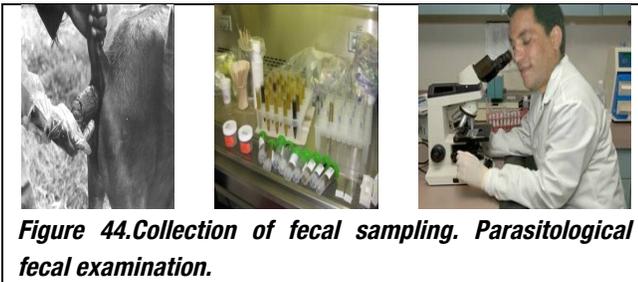


Figure 44. Collection of fecal sampling. Parasitological fecal examination.

- Accurate clinical signs and case history.
- Parasitological examination carried on fecal sample indicated eggs of parasites as in (figure 44).

Treatment and Prevention

- Immediately affected animal given 5 gm (Thiabendazole, piperazine citrate) /10gm body weight by oral administration. All affected animals injected by 5 ml levamisole 10%/100kg body weight. Not more 10 ml per injection site.
- An approach to gastrointestinal worm control should include the following:
- Cattle develop greater immunity to worms than goats.
- Developing a property worm control plan.
- Knowing the types of worm that occur on your farm and the seasons of highest risk.
- Monitoring the worm status of livestock regularly, especially higher risk stock during high risk seasons.
- Preventing the introduction of new or resistant worms onto the property by quarantining all incoming stock and treating them with a quarantine drench.
- Requesting an animal health statement when purchasing stock so you are aware of the disease

status level of assurance that is being provided by the stock vendor.

- Improving nutrition.
- Grazing management to provide young animals that are most vulnerable to worms with pastures with lowest contamination.

Ringworm(Dermatophytosis)

Ringworm is an infection of the outer layer of the skin and hair shafts by one of several types of fungus. All domestic animals and people are susceptible to ringworm. In rural areas, 80% of ringworm cases in people are caught from animals and human cases may be especially severe, possibly resulting in scarring.

Etiological Causes

Parasitic fungus, usually *Trichophyton* or *Microsporum*. (*T. verrucosum*) which penetrate into the outer skin layers where they multiply. The fungi develop highly resistant permanent stages (spores) which remain infective for years in dry places.

Occurrence in animals: Ringworm is also a common infection in domestic animals, especially farm animals, in cattle it is seen mainly in young animals.

Source of Infection in animals: Skin lesions, contaminated skin flakes (dander), hairs, or fungal spores in environment.

Transmission and predisposing agents:

Ringworm can also be spread by contact with animals, clipping, brush, cards, and blankets, fence posts, wire, and the hands of handlers.

Once an infected animal comes in contact with equipment or the environment, the equipment or environment may serve as a source of infection for up to 4 years.

Direct contact with lesions on animals or other humans, contact with contaminated skin flakes (dander), or indirectly through spores in the environment. The fungus takes advantage of skin of people and animals with reduced immune capacity. This puts young animals and children, elderly people and pets, those who are AIDS/HIV+, people on chemotherapy or taking medication after transfusion or organ transplant, and highly stressed people and animals at high risk.

1. Young animals are more easily infected.
2. Poor nutrition increases the likelihood of ringworm infection.

3. Animals that have not had ringworm before are more likely to catch it.
4. Animals kept in the same house with infected animals are more likely to catch ringworm.
5. Animals kept in dark, damp, warm, and poorly ventilated places become infected more easily.
6. The use of clippers, brushes, and blankets on different animals without disinfection will spread this disease.
7. It is likely that the increase in ringworm that we have experienced can be associated with extremely close shearing which causes skin irritation allowing the fungus a way to get into the skin.
8. Flies may spread ringworm.

Clinical Signs;



Figure 45. A patch lesion of skin and round lesion with varies in size.

Ringworm appears as a patch of thick, scaly skin, causing a loss of hair. Oozing or redness may occur at the site. The disease typically, but not always, appears as a round lesion that varies in size. The disease derives its name from the circular pattern it creates that was once thought to be caused by insect larvae.

Lesions appear 10 days to four weeks after exposure to the fungus without treatment, the patches disappear in four weeks to eight weeks. Hair may regrown within eight weeks to 16 weeks. Once the skin is no longer thickened or scaly and lost hair regrown the lesion is virtually cleared of infection (figure 45).

In calves, symptoms are most likely found around the eyes, on the ears and back.

In adult cows, ringworm is most common on the chest, and legs. Sometimes the lesions are extremely itchy. Itching and irritation generally starts on the chest or hindquarters and spreads to other parts of the body as grayish or ashy areas of skin with a slightly raised

circular lesion. These areas are susceptible to secondary skin infections so it is important to treat Ringworm symptoms proactively to prevent complications. The skins lesions that appear with Ringworm are variable, and do not necessarily form a ring. There will be hair loss, usually in small patches at first, with time goes on the patches may disappear or appear at other locations on the skin. There might be scratching due to itchiness.

Diagnosis

Clinical Signs

Accurate clinical signs where the skin under the scab may ooze clear fluid or be bloody. Itching is not usually present.

Laboratory Diagnosis

Microscopic test: The vet takes hairs from around the infected area and places them in a staining solution to view under the microscope. Fungal spores may be viewed directly on hair shafts. This technique identifies a fungal infection in about 40%–70% of the infections but cannot identify the species of dermatophyte.

Laboratory Test: This is the most effective but also the most time-consuming way to determine if there is ringworm on a pet. In this test, the veterinarian collects hairs from the pet, or else collects fungal spores from the pet's hair with a toothbrush, or other instrument, and inoculates fungal media for culture. These cultures can be brushed with transparent tape and then read by the vet using a microscope, or can be sent to a pathological lab. The three common types of fungi which commonly cause pet ringworm can be identified by their characteristic spores.

Differential diagnosis

The condition may possibly be confused with ;
(Squamous eczema, skin allergies and mange)

Treatment

Always wear rubber gloves when handling infected or suspect calf.

Immediately

- Treatment should begin with removing the hair for about 2 inches around the ringworm spot. Next a brush and soapy water should be used to remove the scab down to the skin.
- A mixture of 1 part 7% iodine and 3 parts of baby oil used for 4 days only is said to dry up lesions effectively.

- 1% povidol iodine (tamed iodine) can be used but may stain.
- 5% lime–sulfur solution is effective but smells like rotten eggs and stains wool yellow. Affected animals should be treated daily for 5 days, then weekly for 3 treatments.
- Vitamin A injection may speed healing.
- Treatment may shorten the healing time but is important mainly to reduce the spread of the disease to other animals. Several disinfectants can be used to treat ringworm,

Precautions

Good Personal Hygiene

1. wash hands after handling animals
 2. No eating, drinking, smoking, etc., around animals or their environments
- Wear gloves and protective, long sleeved clothing when handling affected animals or their contaminated environment
 - Spores are very resistant and survive for months in cool, dry, shaded areas.
 - Halters and grooming equipment can be disinfected with bleach or a 4% solution of formaldehyde.
 - Reducing the density of animals and direct contact in addition to increased exposure to sunlight and being maintained on dry lots help prevent the spread between animals.

Babesiosis

(Tick fever, Cattle fever, Red water fever);

Babesiosis is caused by intraerythrocytic protozoan parasites of the genus *Babesia Spp*, that infect wide range of domestic and wild animals and occasionally man.

Most of the 1–2 billion cattle in the world are exposed to babesiosis but figure is not a true reflection of the number at risk to disease.

Etiological Causes

Red water is caused by protozoan parasites of the genus *Babesia spp*. The disease is spread between cattle by ticks and then invades the red blood cells and begins dividing, eventually rupturing the cell. Clinical signs begin around 2 week after infection.

Clinical Signs;



Figure 46. Urine is dark red in color (Hemoglobinuria), reflecting one of the initial names for the disease, "red water".

The first sign is usually a high fever with temperatures reaching 41.5 °C for several days before other signs become obvious. There is anorexia, ruminal atone, increased respiratory rate and reluctance to move. There is hemoglobinemia and hemoglobinuria (red urine) the conjunctiva and mucous membranes are first congested and reddened, but as erythrocytic lysis occur, the color changes to the pallor of anemia as in (figure 46) .

- In the terminal stages, there is severe jaundice, the urine is dark red to brown in color and produces a very stable froth. Either constipation or diarrhea may be present.
- Babesiosis should be suspected in cattle with fever, jaundice and hemoglobinuria.
- The parasitemia and clinical appearance of, *B. divergens* infections are somewhat similar to *B. bigemina* infections; *B. divergens* causes spasm of the anal sphincter, causing the passage of "pipe stem" feces. The feces are evacuated with great force in a long thin stream, even in the absence of diarrhea as illustrated in (figure 46).
- Death occurs within days of the onset of fever and severely affected animals can die after an illness of only 24 hours.
- Mortality is extremely variable and may reach 50 percent or higher, but in the absence of too much stress, most animals will survive
- Increased pulse rate, Abortion in pregnant cows may occur where the clinical signs vary with the age of the animal and the species and strain of parasite.

Diagnosis

Accurate clinical signs and case history;

The disease is typical of a haemolytic anemia disease process but vary according to agent (i.e. species of parasite) and host factors (i.e. age, immune status).

Babesiosis is predominantly observed in adult cattle with *B. bovis* generally being more pathogenic than *B. bigemina* or *B. divergens*. Infected animals develop a life-long immunity against re-infection with the same species and some cross-protection is evident in *B. bigemina*-immune animals against subsequent *B. bovis*.

High fever, ataxia, in coordination, anorexia, production of dark red or brown colored urine (figure 46). Signs of general circulatory shock, sometimes nervous signs associated with sequestration of infected erythrocytes in cerebral capillaries.

Anemia and Hemoglobinuria may appear later in the course of the disease but in acute cases maximum parasitaemia in circulating blood is often less than 1% *Babesia bigemina*

Case history provided that area is enzootic areas where *Boophilus* ticks occur.

Laboratory tests;

- Babesiosis can be diagnosed by identification of the parasites in blood smear.
- These parasites are found within RBCs and all divisional stages—ring (annular) stages, pear-shaped (pyriform) as illustrated in figure (46).
- Polymerase chain reaction (PCR) assay is very good in diagnosis of infection.

Differential Diagnosis

The disease must be differentiated from the following disease. Anaplasmosis, Trypanosomiasis, Theileriasis, Bacillary hemoglobinuria, Leptospirosis, Chronic copper poisoning.

Treatment

Treatment is most likely to be successful if the disease is diagnosed early. It may fail if the animal has been weaker by anemia. Recovery is the rule if treatment is given early in the course of the infection. However, if treatment is delayed, supportive therapy and blood transfusion may be essential if the animal is to survive. Generally, the small *Babesia* spp are more resistant to chemotherapy.

Specific Treatment

- **Imidocarb dipropionate salt (Imizol®)** is effective at a dose 1mg/ kg BW, (Imizol 12 % – 1 ml /100 kg by s/c injection). Imidocarb has been

successfully used as a chemoprophylactic at a high dose rate of 3mg/ kg BW (Imizol 12 % – 2.5 ml /100 kg by s/c injection) that will prevent clinical infection for as long as 2 months but allow mild subclinical infection to occur as the drug level was resulting in premonition and immunity.

- **Diminazene aceturate (Berenil®)** is widely used and given in a 7 % aqueous solution by deep IM injection; 1.05 gm vial is dissolved in 12.5 ml water, and 7.35 gm vial is dissolved in 87.5 ml then 0.5 ml/10 kg (3.5 mg/kg). It does not sterilize the diseased animal completely. There is a high safety margin; the dose does not exceed 4 gm/ animal. Do not inject more than 10 ml at a single site. The prepared solution can be stored for 14 days in a cool place.
- **Quinuronium derivatives (Acaprin, Babesan, Pirevan, Piroparv, Piroplasmin)** are effective against larger babesias especially *B. bigemina*. Quinuronium sulfate, (Acaprin) 5 % solution, at a dose rate of 1 ml/50 kg (1 mg/kg) with a maximum dose 6 ml, by strictly SC injection in the tail fold or dewlap. It has a low safety margin and sporadic side effects may occur, such as muscle tremors, salivation, and restlessness. Side effects are controlled by SC injection of atropine sulfate in cattle (4–5 ml/100 kg) or adrenaline injections.
- **In severely affected animals**, anemia will be severe, blood transfusion and anti-shock preparations are indicated. Supportive therapy is important in chronic cases and convalescent animals, vitamins, electrolytes, hematinic drugs (as Iron dextran 7 ml /kg b.w. by i/m), good nutrition and shade are indicated.
 - In acute *B. bovis* infections, antioxidants, e.g. Vitamin E and high doses of corticosteroids may prove helpful.
- **Blood transfusion:** Blood transfusion to partially restore the PCV greatly improves the survival rate of more severely affected cattle. The amount of RBCs to treat anemia is based on the volume necessary to increase the PCV or HB concentration to the desired value where all domestic animals have blood volumes of 7 % of the body weight except the cat (4 %). Venepuncture into the jugular vein is the method

of choice. Inject a small amount of blood (50–200 ml) and wait for 10 minutes. If no transfusion reaction occurs (restlessness, sweating, tachycardia, increased respiratory rate and dyspnea), the remainder of the blood can then be injected with limited risk.

- **Epinephrine:** 1:1000 (4– 5 ml/400 kg s/c or i/m) and Dexamethazone (0.05 – 0.1 mg/kg, I/V).
- **Long –acting ox tetracycline:** Commonly used treatment consists of two doses of long–acting ox tetracycline, I/M injected at 20mg/kg of body weight at 48hours apart or 6–10mg/kg body weight daily for 5 days.

Prevention and Control

Sanitary Prophylaxis

Eradication of bovine *Babesiosis* has been accomplished by elimination of tick vector, reducing exposure of cattle to ticks, repellents, acaricides and regular inspection of animals, control and eradication of tick vector and cattle develop durable long–lasting immunity after a single infection with *Babesia species*.

- Endemic environments should be monitored carefully.
- Introduction of immune–native animals and new species or strains of diseased agent.

Theileriosis; (East Coast Fever, Egyptian Fever, Tropical Theileriosis).

Theileriosis is a tick–borne disease of cattle, characterized by high fever, swelling of the lymph nodes, dyspnea, and high mortality. Caused by intracellular blood parasites belonging to the *Theileria. Spp* (*Theileria parva*) and transmitted by the tick vector (*Rhipicephalus appendiculatus*), it is a serious problem in east and southern Africa. A large number of *Theileria spp* are found in domestic and wild animals in tick–infested areas of the old World. The most important species affecting cattle are *T parva* and *T annulata* which cause widespread death in tropical and subtropical areas of the World. Both *Theileria* and *Babesia* are members of the suborder *Piroplasmorina*. Although *Babesia* is primarily parasites of RBCs, *Theileria* use successively WBCs and RBCs for completion of their life cycle in mammalian hosts. The infective sporozoite stage of the parasite is transmitted in

the saliva of infected ticks as they feed. Sporozoites invade leukocytes and within a few days, develop to schizonts. In the most pathogenic species of *Theileria* (e.g. *T parva* and *T annulata*) parasite multiplication occurs predominantly within the host WBCs, whereas less pathogenic species multiply mainly in RBCs. Development of the schizont stage of pathogenic *Theileria* causes the host WBC to divide; at each cell division, the parasite also divides. Thus, the parasitized cell population expands and, through migration, becomes disseminated throughout the lymphoid system. Later in the infection, some of the schizonts undergo merogony, releasing merozoites that infect RBCs, giving rise to piroplasms.

Etiology

The African buffalo is an important wildlife reservoir of *T. parva*, but infection is asymptomatic in buffalo. *T. parva* transmitted by ticks from either cattle or buffalo cause severe disease in cattle, but buffalo–derived parasites differentiate poorly to merozoites in cattle and generally are not transmitted by ticks. Hence, buffalo *T parva* are maintained as a separate population. Buffalo *T. parva* were previously considered a separate subspecies (*T. parva. lawrencei*), but DNA typing indicate that the cattle and buffalo parasites are a single species. *T parva* is usually highly pathogenic, causing high levels of mortality, although some less pathogenic isolates have been identified. There are two most important species in cattle are *Theileria. Parva*, which causes East coast fever and *Theileria Annulata* which causes Tropical Theileriosis.

Transmission

Theileria spp are transmitted by tick acting as biological vectors. Cattle that recover from theileria infection usually become carriers for months or year. Latrogenic transmission can also occur via blood (e.g. on reused needles).

Incubation Period

Incubation period for East coast fever is 8–12 days, it might be as long as three weeks in naturally infected animals while incubation period for tropical theileriasis is thought to be approximately 1–3 weeks.

Clinical signs;

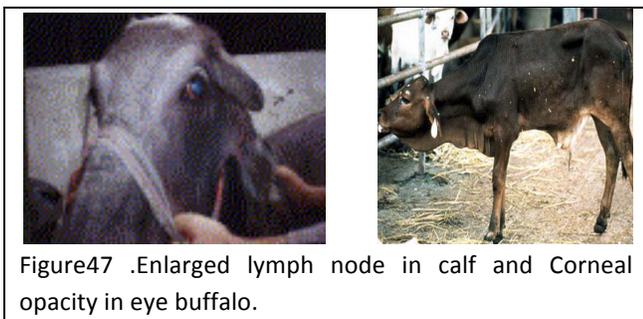


Figure 47 .Enlarged lymph node in calf and Corneal opacity in eye buffalo.

Clinical signs vary according to the level of challenge and they range from in apparent or mild to severe and fatal.

- Typically, fever occurs 7–10 days after parasites are introduced by feeding ticks, continues throughout the course of infection, and may be $>106^{\circ}\text{F}$ (41°C). Lymph node swelling becomes pronounced and generalized. Anorexia develops, and the animal rapidly loses condition; lacrimation and nasal discharge may occur (figure 47). –Terminally, dyspnea is common. Just before death, a sharp decrease in body temperature is usual and pulmonary exudates pours from the nostrils. Death usually occurs 18–24 days after infection.
- Anemia is not a major diagnostic sign (as it is in babesiosis) because there is minimal division of the parasites in RBCs and thus no massive destruction of them

Diagnosis

- A tentative diagnosis of acute theileriosis in endemic area can be justified from the accurate clinical signs (fever, anemia, jaundice and enlargement of superficial lymph nodes) and case history (presence of external parasite on animal).

Laboratory diagnosis

- The tentative diagnosis can be confirmed by detection of the intraerythrocytic piroplasms (round or oval) in Giemsa–stained blood smears, Lymphoblasts in Giemsa–stained smears of needle aspirates from lymph nodes contain multinuclear schizonts. (Koch's blue bodies) .
- Serological tests.
- Hematological features of anemia.

Differential Diagnosis

Tropical theileriosis may be confused with diseased and condition that result in anemia and jaundice such as anaplasmosis and leptospirosis.

Immunity

There is no cross–immunity to other *Theileria spp*, cattle immunized against *theileria annulata* are fully susceptible to *theileria.parva*.

Effective immunity may be developing after recovery or induced by immunization. Cell–mediated immunity may be accompanied by carrier status and last for at least one year.

Treatment

Treatment options for theileriosis are limited to supportive care and symptomatic treatment. In other countries veterinarians have sometimes reported a good response to treatment with ox tetracycline and/or imidocarb of mildly affected animals when administered early in the course of the disease. Response to treatment in severely affected animals is poor. Apart from the obvious unreliability of this treatment, the issue of milk and tissue residues must be borne in mind when considering such medications...

- **Bupavquane (Butalex)** is at present the best therapy at dose of 2.5mg/kg (1ml/20kg) by I/M injection .some time two doses may be required in severe cases at 48–72 hours apart.
- **Oxytetracycline** by injection have been claimed to give relief if given early in prep taint period but have no therapeutic effect when administrated during acute stage. Commonly used treatment consists of two doses of long–acting ox tetracycline, I/M injected at 20mg/kg of body weight at 48hours apart or 6–10mg/kg body weight daily for 5 days.
- **Trace Element:** Treating any underlying subclinical trace element deficiency (i.e. selenium, copper or cobalt) has anecdotally improved recovery rates in some instances. The role of trace element status in the *Theileria* / anaemia syndrome is currently unknown
- **Hematopoiesis:** stimulating drugs must be applied to avoid the deleterious effects of severe anemia.
- **Blood transfusion:** Blood transfusion has been performed occasionally on valuable animals. Animals improve following transfusion but it is expensive and not practical if multiple animals are involved

- **Note:** Most importantly, stress and movement of affected cattle should be minimized. They should be rested, nursed and given high quality feed. Handling of affected cattle should be avoided where possible; if movement is necessary, move animals slowly.
- **Contraindication** to be used fluid therapy in treatment of blood parasite due to low PCV.

Anaplasmosis

Anaplasmosis, formerly known as (gall sickness), traditionally refers to a disease of ruminants caused by obligate intraerythrocytic organism of the order Rickettsiales, family Anaplasmataceae, genus *Anaplasma*. Cattle can be infected with the erythrocytic *Anaplasma*. Anaplasmosis occurs in tropical and subtropical regions worldwide (~40° N to 32° S), is of economic significance in the cattle industry.

Etiology

Anaplasmosis is an infectious, transmissible disease caused by reckettsia organism (*Anaplasma marginals*). Anaplasmosis is called "yellow bag" or yellow fever as affected animals can develop jaundice appearance. The organism invades (infects) and resides in blood cells.

Transmission

Anaplasma marginals can be transmitted by two different ways.

- *First*, it can be transmitted mechanically when red blood cells infected with *Anaplasma marginals* are inoculated into susceptible cattle. This can occur through needles, ear taggers, Castrating knives or other surgical instruments.
- *Second*, mechanical transmission can also occur through the mouth parts of biting insects, such as biting flies, face flies and house flies.

Incubation

The incubation or prep taint period is to 3–8 weeks. The developmental stage is about 4–9 days and is the time when most of the characteristic signs of anaplasmosis appear.

Clinical Signs

Clinical signs of anaplasmosis are related to the age of animals. Calves less than 6 month of age can become infected, rarely show clinical signs and remain carrier.

Carrier animals seldom exhibit clinical signs when challenged with anaplasma.

Yearling to 18 month old cattle may develop clinical signs of varying degree of severity,

Clinical signs in adult animals

- Increase in temperature up to 40–41c.
- Depression
- Loss of appetite
- Anemia or paleness and/or yellow mucus membrane
- Rapid respiration
- Urine may be yellowish in color. Yellowish coloration of the tongue
- Dehydration and constipation and death.

Diagnosis

1. Acute stage of the disease usually based on clinical signs (presence of anemia).
2. Microscopically examinations of stained blood smear where in cattle anaplasma marginal's organisms are located on the periphery of the RBCs.
3. Serological examination in some infection where the organism is not visible on blood smear because all infected cells were removed from circulation and immature erythrocytes are usually not infected.

Treatment

Tetracycline is often used for clinical anaplasmosis. However it cannot be used in every country. General supportive care is also important for anemic animals. Blood transfusions are of limited benefit. The incubation time for the disease to develop varies from two weeks to over three months, but averages three to four weeks. Adult cattle are more susceptible to infection than calves. The disease is generally mild in calves under a year of age, rarely fatal in cattle up to two years of age, sometimes fatal in animals up to three years of age, and often fatal in older cattle. Once an animal recovers from infection, either naturally or with normal therapy, it will usually remain a carrier of the disease for life. Carriers show no sign of the disease but act as sources of infection for other susceptible cattle.

Chlortetracycline can reduce the risk of anaplasmosis and use at the rat of 0.5mg/kg body weight daily during tick season will help to prevent anaplasmosis.

Tetracycline is the drug of choice for clinical anaplasmosis .A single dose of long acting ox-tetracycline (200mg/ml).

- General supportive care such as blood products and fluid therapy may be necessary for anemic animals.
- Blood transfusion is of limited benefit.

Control

Typically cases of anaplasmosis increase in late summer and fall as insect vectors increase; therefore control of vectors is key to preventing.

- Herd treatment with ox tetracycline injection every 3–4 weeks.
- Oral administration of chlortetracycline at 1.1mg/kg body weight.
- Cattle need to be treated with long-acting ox tetracycline every 3–4 days for treatment or 5mg chlortetracycline/bodyweight orally/day for 30 days.

Mange

Mange is a contagious skin disease characterized by crusty, purity dermatitis and hair loss, and caused by a variety of parasitic mites burrowing in or living on the skin.

In humans, these two types of mite infections which would otherwise be known as "mange" in furry mammals are instead known respectively as scabies and demodicosis. However the mites that cause these diseases in humans are closely related to those that cause the mange in other mammals.

Type of mange;

There are five main types of mange in animals.

1. Sarcoptic mange is also known as scabies and is highly contagious.
2. Psoroptic mange is common in the central and western United States.
3. Chorioptic mange, also known as leg mange, affected the pastern area.
4. Demodectic mange is a type of mange that is transferred from cow to calf during nursing.
5. Psoregratic mange is very mild and usually is not treated.

Clinical Signs;

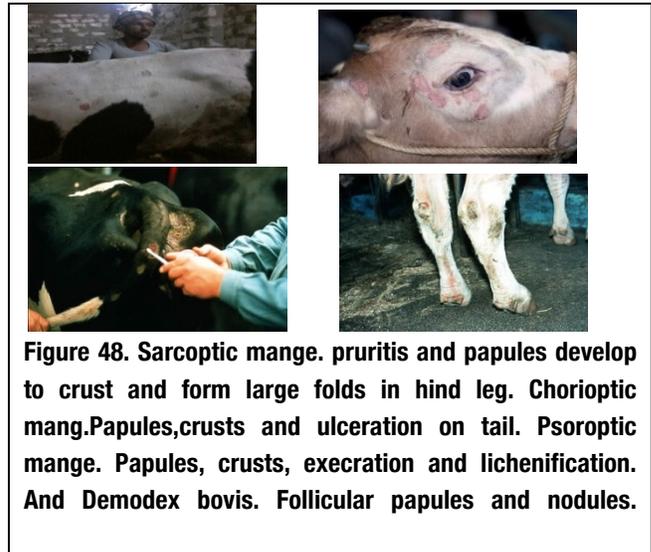


Figure 48. Sarcoptic mange. pruritis and papules develop to crust and form large folds in hind leg. Chorioptic mang. Papules, crusts and ulceration on tail. Psoroptic mange. Papules, crusts, excretion and lichenification. And Demodex bovis. Follicular papules and nodules.

Mange mites cause irritation, thickened scaly skin, hair loss, hide damage and reduced productivity. Different type of mite infection is commonly found in different body areas as illustrated in (figure 48):

Surface mite (*Chorioptes bovis*): usually found on the neck, legs, and tail head causing areas of hair loss which increase slowly in size and cause irritation. Hide damage can occur as cattle rub the affected areas.

Burrowing mite (*Sarcoptes scabiei*): Usually found on the neck and the loin area next to the tail. Burrowing throughout the skin, these mites produce intense irritation and severe skin damage, with large areas of skin becoming thick, crusted and eventually infected. This can result in reduced productivity.

Psoroptic mange (*Psoroptes sp*): Usually found along the back, shoulders and tail head of cattle, causing severe dermatitis (inflammation of the skin), scabs and intense itching.

Bacterial infections of affected areas is common, leading to bleeding and crusting. Production losses and even death can occur, if skin lesions are extensive.

Diagnosis

1. Clinical appearance of the lesions on the skin and hair.
 - The lesions are often first noticed as a circular area on the head, ears, neck and sides, oozing a clear or bloody fluid and the hair will be lost.
2. Cattle with signs of previous infection which appear to have healed with new hair growth.

- Veterinarians usually attempt diagnosis with skin scrapings from multiple areas, which are then examined under a microscope for mites.

Differential diagnosis from other diseases with similar signs skin disease such as lice or photosensitization.

Confirming the diagnosis by microscopic examination of skin scrapings can be used to identify mange mites.

Treatment

The treatment is similar for various types of mange, so discuss the options with your veterinarian. Pour-on medications usually take effect faster than injections. Injections, however, are used in severe mange cases where the infection is deep under the skin. Discuss the treatment options with your veterinarian. In severe cases, crusting can make it difficult for products to make contact with the mites and scabs may have to be removed before treatment.

Immediately

- Thick crusts should be removed gently with Bruch and disinfected with hydrochloric alcohol solution.
- Treatment depends on the use of some agents (4% lime sulphur sprays, 0.5% sodium hypochlorite, 1% povidone-iodine and natomycine).
- Other authors recorded that, the crusts should be removed by scraping or brushing and the lesion should be treated at least twice, three to five days apart by topical application of 2% solution of iodine.
- Thiabendazole paste is suitable.
- Treat all incoming animals against mites, especially during the winter months. Two injections with a macrocyclic lactone (e.g. doramectin, ivermectin, moxidectin) with 7 to 10 days interval should do the

job but keep the animals isolated until 10 days after the second injection. Remember that cattle may be infected with mites without showing clinical signs! Topical products (sprays, dips, pour-ons, see below) can be used to treat incoming animals as well: they are cheaper, but less convenient and less reliable than injectables.

Prevention

- Several strategies are available that can be used to protect cattle from getting scabies:
 - Clean stalls used to house infected cattle, and add fresh bedding before reusing stalls for new animals.
 - Disinfect grooming tools and other instruments used on infected animals.
 - Isolate infected animals from the rest of the herd, and then treat them.
 - Examine replacement cattle for mites before putting them with the rest of the herd avoid overcrowding.
 - Ensure animals are well-nourished; cattle in poor condition are more susceptible to infection than healthy, well-fed animals

VIRAL DISEASE

Influenza-Type Disease Complex

The influenza-type disease complex in cattle comprises disease affecting particularly the respiratory organs but also the gastrointestinal tract. In a narrower sense 'cattle influenza' is the virus influenza type condition affecting the entire stock or crowding disease and includes two further conditions: IBR and mucosal disease which present a similar clinical picture and may be associated with crowding disease.

Since IBR and mucosal disease—in contrast to crowding disease—are caused by known pathogens this can be well demonstrated.

Disease	Crowding disease	IBR/IPV	Mucosal disease
Organs affected	Nose and lung	IBR. Larynx, muzzle, later lung. IPV/External sexual organs	Muzzle, throat, oral cavity, stomach, intestine and claws
Pathogens	Several type virus and bacteria.	Herpes virus.	Toga virus.
Signs	–Aqueous mucopurulent discharge from nose and eyes, pneumonia and pulmonary emphysema	IBR/ respiratory form. Red nose; pneumonia, respiratory distress. Formation of foam at the mouth. IPV/Gential form. Redding and small pustule on the vulva or penis	Erosion on the muzzle and inflammation of the coronal edge of the claws

Treatment	Severely affected. Injection of antibiotic or sulfonamide. mucolytic agent and drug supporting the circulation. Less severely affect. injection of sulafamethazine	Severely affected. Injection of antibiotic or sulfonamide. mucolytic agent and drug supporting the circulation. Less severely affect. injection of sulafamethazine	Severely affected. Treatment useless in severe diarrhea stock. Emergency vaccination
Prevention	Vaccination	Vaccination	Vaccination

Infectious Bovine Rhinotracheitis 'IBR' (red-nose)

The Infectious Bovine Rhinotracheitis /Infectious Pustular Vulvovaginitis (IBR/IPV), sometimes called Red Nose, is an infectious disease of cattle due to Bovine Herpes virus 1. The virus can infect the upper respiratory tract or the reproductive tract. Mortality is low but the economic loss can be considerable. The latent infection allows the virus to persist in the infected hosts for indefinite periods. Reactivation may occur either spontaneously or induced by natural or artificial immunosuppressive stimuli (parturition, transport). It leads to virus replication and re-excretion with its spread in the environment

Etiology

Bovine Herpesvirus-1 (BHV-1) belongs to the Herpes family of viruses. BHV-1 is highly contagious and can quickly spread through a group of calves where the secretions of affected calves are extremely infectious and appear to be highly attractive to other animals. All ages of animals are potentially at risk. With regard to pneumonia, two other viruses are commonly involved: bovine respiratory syncytial virus and parainfluenza 3 viruses. This pathogen virus also causes infectious pustular vulvovaginitis (IPV), a vesicular eczema

Clinical Signs;

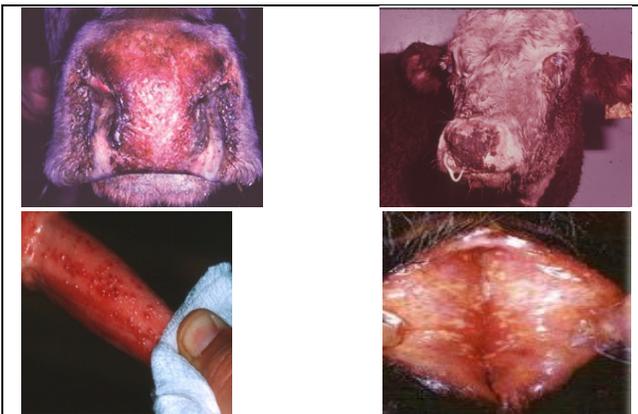


Figure 49. Signs of rhinitis; red nose, nasal lesions, abundant nasal discharge, red and watery eyes. Small pustule in the vulva of cow and small pustule in the glans penis of bull

The severity of symptoms very much depends on the strain of the virus and the susceptibility of the cattle.

In the Respiratory form the Symptoms Include fever (up to 42°C), general depression, drop in milk production, anorexia and emaciation, severe hyperemia of the nasal mucosa (Red nose) with numerous clusters of grayish foci of necrosis on the mucous membranes, abundant serous discharge from nose and eyes, conjunctivitis, hyper salivation, tachypnea and tachycardia, sometimes mastitis, short explosive cough and rarely, death due to obstructive bronchiolitis or bronchopneumonia from secondary bacterial infection (figure 49). An abortion form can complicate the respiratory form with late abortion (between the fifth and eighth month of pregnancy) and placenta retention. It can be the only manifestation.

In the Genital form of the Disease

It lasts for two to three weeks and symptoms include:
In Cows: The pustules are seen in the vulva, particularly at the point of transition from the outer skin to the vaginal mucosa and in the clitoral fossa. There is vaginal discharge which may range from aqueous/mucoid to mucoid/purulent in consistency (pollakiuria) and reduction in milk yield (figure 49).

In Bull: The pustules are observed in the region of the urethral orifice and in the glans penis. Following rupture of these pustules, mucosal defects develop which may ulcerate as a result of bacterial infection and adhesion of the penis to the sheath may occur, totally or partially preventing the protrusion of the penis (figure 49).

In the Young Calf Less Than Six Months Old

The disease is more severe: meningoencephalitis (lack of coordination, hyperexcitation and depression), salivation, blindness, and a high mortality rate.

In newborn calves the disease causes: fever and lack of appetite, salivation, inflammation of the nasal mucosa, conjunctivitis, erosions of the mouth mucous membranes covered with mucopurulent exudates, and respiratory distress is common due to swelling of the larynx and pneumonia while some calves may develop diarrhea. Pathological manifestations have been observed mainly in bovines, while the pathogen role in buffaloes is less clear. Lesions are usually restricted to the upper respiratory tract and include: swelling and congestion of mucosa, sometimes with necrotic foci, petechiae, profuse and fibrin purulent exudates in severe cases.

Diagnosis

Accurate clinical signs (fever, nasal discharge and conjunctivitis), pustules in vulva of cow and penis of bull. In dairy cattle, prolonged drop in milk production, abortion and reduced fertility are also precursory signs.

Laboratory examinations;

Laboratory examination are often required for a definitive diagnosis. The virus can be isolated from blood on EDTA, nasal, pharyngeal, and conjunctiva swabs, aborted fetus, placenta, vaginal swab, prepuce washing fluid and semen. Techniques include neutralization or antigen detection methods using mono-specific antiserum or monoclonal antibodies; PCR is also used for detection on semen. Samples should be stored in a transport medium (cell culture medium containing antibiotics and two to ten percent fetal bovine serum to protect the virus from inactivation), cooled at 4°C, and rapidly submitted to the laboratory. Serum can be submitted for virus neutralization test and ELISA. The ELISA test allows the detection of antibodies in milk. A delayed cutaneous hypersensitivity test has also been proposed for IBR/IPV diagnosis.

Differential diagnosis

- The respiratory form: enzootic bronchopneumonia, Bovine Virus Diarrhea/Mucosal Disease, gangrenous coryza, rinderpest, theileriosis;
- The abortion form: Bovine Virus Diarrhoea/Mucosal Disease, brucellosis, listeriosis, leptospirosis, coxiellosis.

Treatment

No specific treatment but Broad spectrum antibiotics can prevent bronchopneumonia from secondary bacterial infection.

➤ 1st day

- 1ml sulfamethoxypyrazine/10kg bodyweight
- 1ml Biosolvon/10kg bodyweight.
- 10 ml Vitamin AD3E/100kg bodyweight.
- 0.3 ml caffeine-sodium salicylate 50%/10kg bodyweight.\
- Antipyretic drug for all visible affected animals with fever.

➤ 2nd – 3rd day

- 1ml Biosolvon/10kg bodyweight.
- 0.3 ml caffeine-sodium salicylate 50%/10 kg if necessary.
- 0.2 ml of dexamethasone/10kg once one second day.

➤ 1st – 21 days

- 10g (cholortetracycline, sulphadimidine and vitamin A and B1)/animal per day for at least 3 weeks.

➤ N.B

- Management practices designed to reduce stress, isolate infected animals, and provide adequate food and water will limit disease transmission and severity.

Prevention

The prevention of IBR is based either on preventing the virus from entering the herd or vaccination. Vaccination with modified live virus (MLV) vaccines generally provides adequate IBR protection by helping prevent disease or reducing its severity. Parenteral (intramuscular or subcutaneous) and intranasal administered IBR vaccines are available, with intranasal (IN) products producing a rapid local immune response.

Intestinal Diseases in Cattle (Bovine viral diarrhea and Mucosal disease complex)

Bovine Viral Diarrhea

Bovine viral diarrhea (BVD) is most common in young cattle (6–24 mo old). The clinical signs can range from subclinical infection to acute severe enteric disease to the highly fatal mucosal disease complex characterized by profuse enteritis in association with typical mucosal

lesions. BVD must be distinguished from other viral diseases that produce diarrhea and mucosal lesions. These include malignant catarrhal fever which usually is a sporadic disease in more mature cattle, bluetongue and rinderpest which is currently considered to be eradicated worldwide.

Etiology and Epidemiology

Bovine viral diarrhea virus (BVDV), the causal agent of BVD and mucosal disease complex and belongs to the Pestivirus genus (Flaviviridae family) and is closely related to the classical swine fever virus, the border disease of sheep and hepatitis C these viruses are related antigenic ally as well as genetically. The bovine viral diarrhea (BVDV) infection is a major worldwide problem affecting different species of ruminants, two biotypes of the virus, cytopathic and non–cytopathic, are identifiable based on their activity in vitro cultures.

Clinical Findings and Lesions

- BVDV infection can be described as an acute, chronic, persistent, mucosal disease and hemorrhagic syndrome.
- Subclinical infection without any clinical signs that is followed by seroconversion is the most common form of infection in the field.
- Acute clinical disease may range from mild disease of high morbidity and low mortality to severe enteric disease with considerable mortality.
- Biphasic fever [40°C], depression, decreased milk production, transient in appetite, rapid respiration; excessive nasal secretion, excessive lacrimation and diarrhea are typical signs of acute clinical BVD.
- Clinical signs of disease usually are seen 6–12 days after infection and last 1–3 days. Transient leukopenia may be seen with onset of signs of disease. Recovery is rapid and coincides with production of viral neutralizing antibody.
- In thrombocytopenic cattle, petechial hemorrhages may be seen in the conjunctiva, sclera, nictitating membrane of the eyes and on mucosal surfaces of the mouth and vulva. Prolonged bleeding from injection sites also occurs. Swollen lymph nodes, erosions and ulcerations of the GI tract,
- In pregnant cattle, BVDV may cross the placental barrier and infect the fetus. The consequences of fetal infection usually are seen several weeks to

months after infection of the dam and depend on the stage of fetal development and on the strain of BVDV. Infection of the dam near the time of fertilization may result in reduced conception rates. Infection during the first 4 month of fetal development may lead to embryonic resorption, abortion, growth retardation or persistent infection. Congenital malformations of the eye and CNS result from fetal infections that occur between months 4–6 of development. Fetal mummification, premature birth, stillbirth, and birth of weak calves also are seen after fetal infection.

Mucosal Disease

Mucosal disease is an uncommon but highly fatal form of BVD occurring in persistently infected cattle and can have an acute or chronic presentation.

Causative agent

Cytopathic BVDV; The origin of the cytopathic BVDV is usually internal, resulting from a mutation of the resident persistent, noncytopathic BVDV. In those cases, the cytopathic virus is antigenic ally similar to the resident noncytopathic virus. External origins for cytopathic BVDV include other cattle and modified–live virus vaccines. Cattle that develop mucosal disease due to exposure to a cytopathic virus of external origin often produce antiviral antibody. Prevalence of persistent infection usually is low, and many persistently infected cattle do not develop mucosal disease, regardless of exposure.

Clinical Signs

Acute mucosal disease is characterized by fever, leukopenia, dysenteric diarrhea, in appetite, dehydration, erosive lesions of the nose and mouth, and death within a few days of onset.

Chronic mucosal disease may last several weeks to months and are less severe than those of acute mucosal disease. Intermittent diarrhea and gradual wasting are common.

Coronitis and eruptive lesions on the skin of the interdigital cleft cause lameness in some cattle. Lesions found at necropsy are less pronounced than, but similar to, those seen in acute mucosal disease. Often, the only gross lesions seen are focal ulcerations in the mucosa of the cecum, proximal colon, or rectum, and the mucosa over Peyer's patches of the small intestine may appear sunken.

Diagnosis

Accurate Clinical Signs and Case History

The primary step for veterinary clinicians and diagnosticians is diagnosed tentatively from disease history, clinical signs and detection of the virus in samples obtained from clinically ill, as well as sub clinically infected cattle.

Although acute infections with BVDV are often asymptomatic or produce only mild clinical symptoms, there is evidence that they result in immunosuppression. The problems associated with the BVDV infections during pregnancy are complex. Pregnant, immunologically naive cattle are at risk of acquiring BVDV infections early (<100 days gestation), resulting in abortion; or mid-gestation (100–125 days), resulting in calves that are congenitally infected and are born persistently infected (PI); or later in gestation (>125 days), resulting in weak calves.

There are at least two factors associated with the virus and two associated with the pregnant animal that contribute to the vulnerability of the host to infection and disease. The two viral factors are immunosuppression and strain variation while the two host factors are the immune status of the heifer entering pregnancy and the physiologic immunosuppression which occurs during pregnancy.

Laboratory examination also is required in some outbreaks of mucosal disease or clinically severe acute BVD, because either disease may appear similar to rinderpest or malignant catarrhal fever. Laboratory tests can confirm clinical diagnosis and detect persistent infections. The samples are represented by oral swabs, fecal samples and blood with EDTA; spleen, brain, lung and kidney from fetuses and spleen, lymph nodes, gut and abomasums from adult animals. The samples are submitted to cell cultures with labeled antibodies. Stereological diagnosis can be performed with serum neutralization and ELISA.

Necropsy examination, erosions and ulcerations may be found throughout the GI tract. The mucosa over Peyer's patches may be hemorrhagic and necrotic. Extensive necrosis of lymphoid tissues, especially gut-associated lymphoid tissue is seen on microscopic examination

Treatment and Control

- **Immediately**

- All animals at risk emergency vaccinated with BVD/MD vaccine.
- Revaccinated calves which were younger than 3 months at first vaccination not earlier than 4 weeks after first vaccination.
- **1st – 21st day**
- All animal at risk given 10g (chlortetracycline, sulfadimidine, vitamin A, B1) but animals feed on milk given 10g (chlortetracycline, sulfadimidine, vitamin A and citric acid) /per animals and per day.
- Severely affected cattle with diarrhea, treatment usually hopeless and advice by early slaughter of sick animals.

Rabies disease

Etiology

The Rhabdovirus (genus Lyssavirus) responsible for the disease is a truly neurotropic virus that causes lesions only in nervous tissue. It may be eliminated by the use of standard disinfectants and heat treatment.

A bite from an infected wild animal, such as a fox or raccoon, is a common method of infection in cattle.

Epidemiology

The source of infection is always represented by infected animals and it spreads mostly by saliva through contamination of wounds or bites or ingestion. The excretion in milk is low and does not cause the disease. Reports of this disease in buffaloes are not very numerous since they defend themselves well from rabid animals and no cases of rabies have been reported due to transmission by bats as is the case in cattle. However mortality in buffaloes is 100 percent. Rabies occurs in most countries of the world except on islands where rigid quarantine measures are guaranteed. The incubation period is almost three weeks.

Saliva is the main method of transmission of the virus being shed through infected cattle and spreading through the body via the bloodstream. It travels to the spinal cord where it can incubate for months on end, although 3–12 weeks is common. From the spine the virus moves into the brain, at which point clinical signs of rabies often appear in cattle.

It is possible for the virus to enter the body through orifices and lacerations to the skin. This however, is uncommon. Open wounds, mucous membranes, eyes and

the mouth are possible entrance points for the virus. Under normal conditions the virus is not spread through the air, though this method of transmission is possible.

Often presented in a single animal rather than the full herd, rabies can be spread due to the curious nature of cows that will inspect raccoons, dogs and foxes etc. that are exhibiting strange behavior.

Transmission from unpasteurized cow's milk to humans is currently being scrutinized although properly cooked meat and pasteurized milk from infected animals poses no danger to humans.

Clinical Findings;

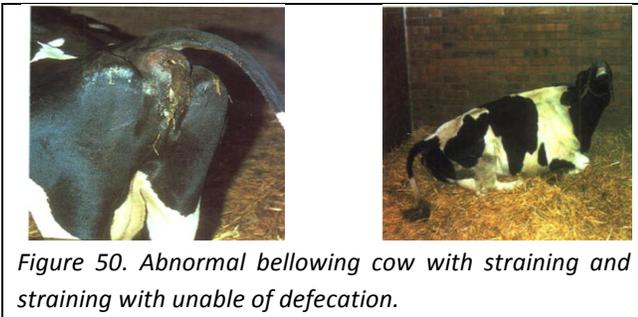


Figure 50. Abnormal bellowing cow with straining and straining with unable of defecation.

The symptoms of disease may present two forms, illustrated in (figure 50).

Paralytic form (drooling of saliva, eructation, grinding of teeth, tail movement, anorexia, stiffness of hind limbs, paralysis and recumbence, death in two to three days)

Furious form;(alert state, hypersensitivity, sexual excitement, inability to swallow, ramming of head on fixed objects, loud bellowing, collapse and death.

Milk production and feed intake may drop gradually before rapidly falling and cows may look very alert, staring at objects.

Aggressive, excitable or exaggerated movements can be signs of rabies. Sexual activity can be increased, including mounting behavior. Bulls can have persistent erections or a prolapsed penis.

Clinical signs of rabies can be varied in cattle and other animals. Some of the more common clinical signs include: Sudden change in behavior, progressive paralysis, Ataxia in addition to decrease milk production in dairy cattle

Abnormal bellowing, paralysis of the throat, drooling, head extension and bloat.

Diagnosis

- Accurate clinical signs and case history (last biting by other animal).
- Laboratory examination for confirmation: Detection of negri bodies by brain microscopic examination, impression smears from brain tested by FAT, CFT and ELISA, direct immunofluorescence test and PCR. The biologic test on mice is also important.

Therapy

- **Immediately**
 - Wounds should be irrigated with a soap solution and water,
 - Post exposure vaccination can be performed.
 - Anti-Viral rabies drugs will be used as soon as possible.
 - –Suspected animals should be kept under close observation avoiding euthanasia.

Prophylaxis;

Destruction of wild fauna around animal holdings and vaccination of cats and Dogs are important. Vaccines from chick embryo origin and tissue culture origin can be used.

Education and vaccination of wild animal populations can control rabies transmission. By limiting the number of wild animals carrying the virus and the chance that those animals will come in contact with pasture and farmland, the likelihood of rabies transmission is reduced.

Cautious handling of cattle with undetermined illnesses is recommended, especially if neurological signs have been observed. Examining a cow's mouth should be done with gloves to avoid exposure of the veterinarian's or stockman's hand to saliva.

Papillomas ' (Warts);

Bovine papillomavirus (BPV) is a group of DNA viruses of the family Papillomaviridae that are common in cattle. Infection causes warts (papillomas and fibropapillomas) of the skin and alimentary tract, and more rarely cancers of the alimentary tract and urinary bladder. They are also thought to cause the skin tumors.

BPV provides an excellent model for studying papillomavirus molecular biology, and also allows the dissection of the processes by which this group of viruses

causes cancer. Warts are growth on the skin and mucus membrane

Etiological Causes

Cattle warts are caused by at least six different types of papilloma virus, these viruses are species-specific for cattle, this they do not cause disease in other livestock or human.

Clinical Signs;



Figure.51. Typical skin warts around the eye and neck

- Wart lesions in cattle are growths which vary in size and sharp, some being fairly flat on the skin and other being raised on stalk-like projection with a cauliflower-like surface (figure 51). They can occur anywhere on the body but are usually noticed on the nose, face, neck, side and teat.
- Young animals are most commonly affected with warts and will eventually develop an immune response against warts. Warts on the penis of bulls are potentially serious because they can physically prevent the bull from breeding females.
- In rare cases animals will have large masses of warts which may create physical problems with eating or other function.
- Esophageal papillomas are known to occur in cattle infected with bovine papillomas virus and may undergo malignant progression in cattle that feed on bracken fern.

Treatment and prevention;

1. Surgical removal is recommended if the warts are sufficiently objectionable, However because surgery in the early growing stage of warts may lead to recurrence and stimulation of growth. The warts should be removed when near their maximum size or when regressing.
2. When the disease is herd problem it can be controlled by vaccination with suspension of ground

wart tissue in which the virus has been killed with formalin.

3. Autogenously vaccines may be more effective than those commercially available. It may be necessary to begin vaccination in calves as early as 4–6 week of age with a dose of 0.4 ml intradermal given at two sites and repeated in 1year of age.
 - **N.B;** If the animal was exposed to virus before vaccination, immunity may develop too late to prevent warts.

Foot and Mouth Disease

Foot-and-mouth disease is an infectious and sometimes fatal viral disease that affects cloven-hoofed animals, including domestic and wild bovid. The virus causes a high fever for two or three days, followed by blisters inside the mouth and on the feet that may rupture and cause lameness.

Foot-and-mouth disease is a severe plague for animal farming, since it is highly infectious and can be spread by infected animals through aerosols, through contact with contaminated farming equipment, vehicles, clothing or feed, and by domestic and wild predators. Its containment demands considerable efforts in vaccination, strict monitoring, trade restrictions and quarantines, and occasionally the elimination of millions of animals.

Etiology

The foot-and-mouth disease virus (FMDV) is a member of the genus *Aphthovirus* in the family Picornaviridae. There are seven immunologically distinct serotypes – O, A, C, SAT 1, SAT 2, SAT 3 and Asia 1 – and over 60 strains within these serotypes. New strains occasionally develop spontaneously. FMDV serotypes and strains vary within each geographic region. Serotype O is the most common serotype worldwide. This serotype is responsible for a pan-Asian epidemic that began in 1990 and has affected many countries throughout the world. Other serotypes also cause serious outbreaks. Immunity to one serotype does not provide any cross-protection to other serotypes. Cross-protection against other strains varies with their antigenic similarity.

Transmission

The foot-and-mouth disease virus can be transmitted in a number of ways, including close contact animal-to-animal spread, long-distance aerosol spread and

inanimate objects, typically fodder and motor vehicles. The clothes and skin of animal handlers, such as farmers, standing water, and feed supplements containing infected animal products can harbor the virus as well. Cows can also catch FMD from the semen of infected bulls. Control measures include quarantine and destruction of infected livestock, and export bans for meat and other animal products to countries not infected with the disease.

Foot and Mouth Disease Infecting Humans

Humans can be infected with foot-and-mouth disease through contact with infected animals, but this is extremely rare. Some cases were caused by laboratory accidents. Because the virus that causes FMD is sensitive to stomach acid, it cannot spread to humans via consumption of infected meat, except in the mouth before the meat is swallowed. Symptoms of FMD in humans include malaise, fever, vomiting, red ulcerative lesions (surface-eroding damaged spots) of the oral tissues, and sometimes vesicular lesions (small blisters) of the skin. According to a newspaper report, FMD killed two children in England in 1884, supposedly due to infected milk.

Another viral disease with similar symptoms, hand, foot and mouth disease, occurs more frequently in humans, especially in young children; the cause, Coxsackie A virus, is different from FMDV. Coxsackie viruses belong to the *Enteroviruses* within the *Picornaviridae*.

Because FMD rarely infects humans, but spreads rapidly among animals, it is a much greater threat to the agriculture industry than to human health. Farmers around the world can lose huge amounts of money during a foot-and-mouth epizootic, when large amounts of animal capital are destroyed, and revenues from milk and meat production go down.

Economic and Ethical Issues

Epidemics of FMD have resulted in the slaughter of millions of animals, despite this being a frequently nonfatal disease for adult animals (2–5% mortality), though young animals can have a high mortality. The destruction of animals is primarily to halt further spread as growth and milk production may be permanently affected even in animals that have recovered. Due to international efforts to eradicate the disease, infection would also lead to trade bans being imposed on affected countries. Critics of current policies to cull infected herds

argue that the financial imperative needs to be balanced against the killing of many animals, especially when a significant proportion of infected animals, most notably those producing milk, would recover from infection and live normal lives, albeit with reduced milk production. On the ethical side, one must also consider that FMD is a painful disease for the affected animals. The vesicles/blisters are painful in themselves, and restrict both eating and movement. Through ruptured blisters, the animal is at risk from secondary bacterial infections and, in some cases, permanent disability.

Clinical Signs;



Figure 52. Indicated vesicular lesion in the gum and tongue, foamy and sticky saliva, no ruptured vesicle in the inter-digital space and the heel bulbs of a steer's foot with no ruptured vesicles

Vesicles (blisters) followed by erosions in the mouth or on the feet and the resulting excessive salivation or lameness are the best known signs of the disease. Often

blisters may not be observed because they easily rupture leading to erosions.

These Signs May Appear in Affected Animals During an FMD Outbreak as Illustrated in (figure 52);

1. Marked rise in body temperature for 2 to 3 days. Vesicles that rupture and discharge clear or cloudy fluid, leaving raw, eroded areas surrounded by ragged fragments of loose tissue. Production of sticky, foamy, stringy saliva caused by the presence of painful vesicles in the skin of the lips, tongue, gums, nostrils.

2. Reduced consumption of feed due to painful tongue and mouth lesions, Lameness with reluctance to move caused by the presence of painful vesicles in the interdigital spaces, Abortions, low milk production (dairy cows) and myocarditis (inflammation of the muscular walls of the heart) and death, especially in newborn

3. In cattle, suspicion should be raised by simultaneous salivation and lameness, particularly when a vesicular lesion has been seen or is suspected to exist.

4. When sudden death is observed in young calf, older animals should also be examined; young animals that die of heart disease may not have vesicular lesions.

Diagnosis

Clinical signs and case History

Tranquilization may be necessary for a thorough examination as vesicles are painful and may be difficult to see.

Laboratory examination;

Laboratory confirmation is necessary by foot-and-mouth virus isolation in cell culture and laboratory animals. Serological methods consist of complement fixation (CFT), plaque reduction assay, virus neutralization, radial immunodiffusion, virus infection associated antigen test and ELISA.

Differential Diagnosis:

FMD cannot be distinguished clinically from other vesicular diseases including vesicular stomatitis, swine vesicular disease and vesicular exanthema.

- In domesticated animals, the symptoms may also resemble foot rot, traumatic stomatitis, and chemical and thermal burns.
- In cattle, oral lesions can resemble rinderpest.

Treatment

Affected animals will recover. However because of the loss of production and the infectious state of the disease infected animals are usually culled.

➤ **Immediately**

- FMD lesions should be disinfected and treated with emollients
- Affected cattle may give symptomatic treatment.

➤ **1st day to 5th day**

- Immediately infected animals injected, I/V injection with supportive fluids therapy to treat dehydration.
- Administration of feverish animal with antipyretic drug (I/V or I/M).
- Antibiotic drug to avoid secondary bacterial infection (ox tetracycline, penicillin).
- Lotion of the mouth lesion with antiseptic drugs as well as washing the foot lesion with copper sulfate.
- You can help the animals to recover: Shade them from the sun and give them plenty of water, give them soft feed such as green soft lush grass, better than hay as the blisters make it painful for the animals to eat and addition of molasses is devised to give the animals energy.
- **Warning:** Do not give antibiotics by mouth to adult cattle. It makes these animal sick by destroying essential micro-organisms in their rumens, bacteria which are there as a vital part of the digestive process.

Control

- FMD vaccine is a killed preparation and at best affords good protection against challenge for 4–6 month. Vaccination can be used to reduce the spread of FMD or protect specific animals.
- Vaccines are also used in endemic regions to protect animals from clinical disease. FMDV vaccines must closely match the serotype and strain of the infecting strain.
- Vaccination with one serotype does not protect the animal against other serotypes, and may not protect the animal completely or at all from other strains of the same serotype. Currently, there is no universal FMD vaccine. In country where foot-and-mouth disease is not endemic, the importation of animals and animal products from FMD-endemic areas is strictly controlled.

- High temperature short time (HTST) pasteurization greatly reduces the amount of viable FMDV in milk, but some studies suggest that residual virus may sometimes persist.
- FMD outbreaks are usually controlled by quarantines and movement restrictions, euthanasia of affected and in-contact animals, and cleansing and disinfection of affected premises, effective disinfectants include sodium hydroxide (2%), sodium carbonate (4%), citric acid (0.2%), while iodophores, quaternary ammonium compounds, hypochlorite and phenols are less effective, especially in the presence of organic matter. Infected carcasses must be disposed of safely by incineration, rendering, burial or other techniques.
- Milk from infected cows can be inactivated by heating to 100°C for more than 20 minutes. Slurry can be heated to 67°C for three.

Bovine Ephemeral fever (Three day sickness)

Bovine ephemeral fever (BEF), commonly known as three day sickness is a disease of cattle and occasionally buffaloes, characterized by short fever, shivering, lameness and muscular stiffness. It causes serious economic losses through deaths, loss of condition, decreased milk production, and lowered fertility of bulls, mis-mothering of calves, delays in marketing and restrictions on the export of live cattle.

Cause

Bovine Ephemeral Fever (BEF) is a viral disease of cattle and buffalo, caused by an arthropod-borne rhabdovirus that features four serovirus (Sharma and Kumar, 2003). It is also referred to as an arbovirus in so much as biting insects spread it. The BEF virus is transmitted between cattle by flying insects where the transmitting insects (vectors) have not been definitely identified but it is thought that mosquitoes and biting midges (especially sand flies) are responsible for that. Spread of the disease depends on the many factors as season, weather conditions – rain, prevailing easterly and southerly winds are necessary for the survival and dispersal of the vectors which spread the virus.

Clinical Signs;



Figure 53. Recumbence cow with dullness appearance.

Typically, three stages of the disease are recognized as illustrated in (figure 53).

The acute febrile stage: A sudden onset of fever (41°C) can be observed. The first sign in milking cows is a sudden and severe drop in milk production. Cattle in advanced stages of pregnancy may sometimes abort; this is probably due to the fever, rather than to a specific effect of the virus. Animals stop eating and drinking and become depressed, start drooling saliva and often there is a discharge from the eyes and nostrils. Feeding and cud chewing stop and milk production, especially in dairy cows, is reduced.

The Second Stage: Lameness may not appear prior to the second day of illness but may cause the typical posture of laminitis. Muscular areas over the shoulder, back and neck regions show swelling. Shivering, stiffness and colonic muscular movements are also manifest. Animals go down with degree of secondary bloat may also occur due to general inflammation of the abdominal cavity and ruminal stasis. The lameness may shift between limbs. Joints may be visibly swollen. Affected cattle may become recumbent and paralyzed for 8 hr to >1 wk. After recovery, milk production often fails to return to normal levels until the next lactation. Abortion, with total loss of the season's lactation occurs in 5% of cows pregnant for 8–9 month. The virus does not appear to cross the placenta or affect the fertility of the cow. Bulls, heavy cattle, and high-lactating dairy cows are the most severely affected, but spontaneous recovery usually occurs within a few days. More insidious losses may result from decreased muscle mass and lowered fertility in bulls.

The recovery stage: By day three the affected animal is usually standing again and will begin to eat. However, lameness and weakness may last for another two or three days. The disease can vary in severity. Some animals may show only slight symptoms for about 24 hours, while a

small number may be affected for many weeks. The disease is usually milder in calves below 12 months of age. Milk production should return nearly to normal after about three weeks, but cows which are affected late in lactation, often become dry mastitis sometimes. Some animals remain down due to muscle damage or damage to the spinal cord. Generally, about 1% die or are destroyed because they cannot get up, although this figure may be as high as 10%. Some animals that recover from ephemeral fever will have the staggers due to spinal cord damage.

Diagnosis

Accurate clinical signs and case history;

When an outbreak occurs in unvaccinated cattle not previously exposed to Virus, a diagnosis of BEF can often be accomplished based on clinical signs and the brevity of the illness.

Laboratory examination;

Laboratory confirmation is by serology, rarely by virus isolation. Whole blood should be collected from sick and apparently healthy cattle in affected herds. Samples must be sufficient to provide 2 air-dried blood smears, 5 ml in anticoagulant (not EDTA), and 20 ml for serum. Blood analysis evidences leukocytosis, neutrophilia, lymphopenia and increased fibrinogen. BEF virus can often be identified from a blood sample taken from animals in the fever stage of the disease.

Alternatively, two blood samples, the first obtained during the fever stage and the second fourteen days later can be examined for development of antibodies to ephemeral fever virus.

Isolated viruses are identified by neutralization tests using specific ephemeral fever virus anti-sera and by ELISA using specific monoclonal antibodies. The neutralization test and the blocking ELISA are recommended for antibody detection and give similar results. A 4-fold rise in antibody titer between paired sera collected 2-3 wk apart confirms infection.

Necropsy examination; Cattle that die is important to rule out other acute febrile diseases that often occur under the same conditions as ephemeral fever and present in a similar manner, such as tick fever.

Treatment and Prevention

- **Complete rest** is the most effective treatment, and recovering animals should not be stressed or worked because relapse is likely.
- **Medical treatment** is often unnecessary for non-lactating stock. However, bulls and high-producing cows, in early to peak production, should have supportive treatment. It consists in relieving temperature and muscular stiffness with paracetamol and phenylbutazone. Broad spectrum antibiotics (streptopenicillin, tetracycline's) prevent secondary bacterial complications.
 - The intravenous or subcutaneous administration of calcium borogluconate has been found to be beneficial to cattle with signs of hypocalcaemia.
- **Antibiotic treatment** to control secondary bacterial infection.
- **Supportive fluids therapy** has been used to treat dehydration.
- **N.B.** Affected animals should not be drenched or force fed as BEF can impair the swallowing reflex, so this may result in the inhalation of food or water and pneumonia.

Prevention

Both live and inactivated vaccines against BEF are available: In cattle the live vaccine gives at least 12 months' protection following two doses, the killed one only gives about six months' protection. Cattle can be vaccinated beginning from six months of age and should then be revaccinated each year to ensure continued protection.

The efficacy of vector control remains uncertain because the insect vectors have not been fully identified.

Lumpy Skin Disease

Lumpy Skin Disease (Pseudourticaria. Nethling virus disease, Exanthema) Lumpy skin disease (LSD) is an acute to chronic a viral disease of cattle that is characterized by fever, multiple firm, well-defined nodules in the skin, lesions left by erosion on the teats, nose, mouth and pharynx, enlarged lymph nodes and swelling of one of more legs. Lumpy skin disease has a low mortality, but because of the prolonged detrimental effect on the body of the animal, it is of large economic importance, where this in turn leads to significant losses in production as a result of emaciation, temporary or permanent loss of milk

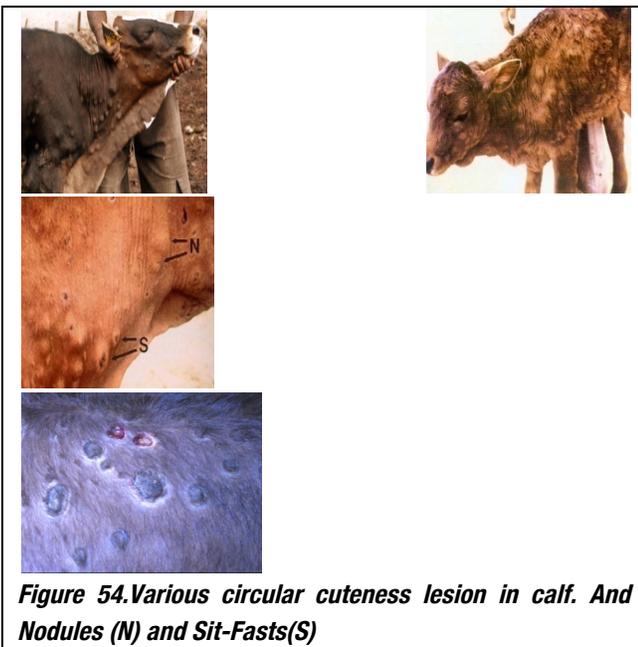
production, infertility in bulls as well as cows, abortions and permanent damage to the skins.

Causes

Virus in the family poxviridae, genus capripoxvirus .It is closely related antigenic ally to sheep and goat poxviruses, the morbidity rat in cattle can vary from 3–85% depending on the presence of insect vectors and host susceptibility while the mortality is low in most cases (1–3%) but can be as high as 20 to 85% usually high mortality rate(75–85%) in some outbreaks have not been explained.

- Animal transmission;
- *Primary route*; biting insects (particularly mesquites).
- *Minor route*; direct contact to coetaneous lesion, saliva, nasal discharge, milk and semen
- No carrier state and the spread related to movement of cattle.

Clinical signs;



Clinical signs illustrated in figure (54);Painful swelling and then fever, lacrimation, nasal discharge and hyper salivation, followed by the characteristic eruptions on the skin and other parts of the body in 50% of susceptible cattle. The incubation period is 4–14 days. Morbidity is 5%–50%; mortality is usually low. The nodules are well circumscribed, round, slightly raised, firm, and painful and involve the entire cutis and the mucosa of the GI,

respiratory, and genital tracts. Nodules may develop on the muzzle and within the nasal and buccal mucous membranes.

The skin nodules contain a firm, creamy–gray or yellow mass of tissue. Regional lymph nodes are swollen and edema develops in the udder, brisket, and legs with late stage and secondary infection sometimes occurs and causes extensive suppuration and sloughing; as a result, the animal may become extremely emaciated, and euthanasia may be warranted. In time, the nodules either regress, or necrosis of the skin results in hard, raised areas (“sit-fasts”) clearly separated from the surrounding skin. These areas slough to leave ulcers, which heal and scar. The greatest loss is due to reduced milk yield, loss of condition, and rejection or reduced value of the hide.

Diagnosis

Accurate clinical signs: Characteristic skin nodules, enlarged superficial lymph nodes are common and fever where the clinical diagnosis of LSD is not difficult for those familiar with the disease but those who are not so experienced can readily confuse the lesions with many other conditions: for instance, bovine herpes virus 2 (Allerton) infections; delayed hypersensitivity reactions following foot–and–mouth disease vaccinations; insect bites; streptothricosis, globidiosis, or demodicosis.

Laboratory diagnosis:

- Virus isolation and identification
- Histopathological sections of the skin lesions show changes peculiar to LSD.
- Serology.

Necropsy examination;

Characteristic skin nodules, lesions in the mucous membranes throughout the gastrointestinal tract, nodules in lungs and Bronchopneumonia may be present.

- Hemorrhage in spleen, liver and rumen. Synovitis and tenosynovitis may be seen with fibrin in the synovial joint.

Treatment

There is no specific treatment available for LSD infected cattle.

- Sick animals may be removed from the herd and given supportive treatment.
- **Initially**

- There is no treatment for LSD. In areas where the disease is common such as South Africa and Egypt, vaccination is used to prevent its occurrence.
- **Supportive treatment**
- **Secondary infection** by bacteria can be controlled by administration of broad-spectrum antibiotics (e.g. tetracycline) and sulfa drugs (sulphonamides). When you see your animals showing the clinical signs described above, immediately contact your veterinary agent so that you can be assisted.
- Local wound dressing to discourage fly worry and prevent secondary infections.
- Good nursing care should always be exercised for the sick animals and food and water should be made readily available
- Infected cattle immediately injected I/V with antipyretic drug (Novalgin ,Novacid or Analgen) to relief high temperature and supportive fluid therapy.
- Animals injected by antihistaminic drug, I/M, for 3–5 day.
- Local applications of insecticides to infected cattle have been made in an attempt to reduce further transmission but to no apparent benefit.
- Animals generally recover with good nursing care.

Control and Eradication

A better method is however preventative immunizing by means of vaccination. Calves of mothers that have not been vaccinated or cows that have never been exposed to the disease could be vaccinated at any age. Calves of cows that have been vaccinated should only be vaccinated after 6 months of age. After that vaccination should be done annually.

Vaccination is the method of choice for the control of LSD in your cattle post. Both live (attenuated) and dead (inactivated) LSD vaccines are commercially available. The live vaccine confers a life-long immunity after a single injection and is therefore the most preferred. Booster vaccination will be required should you opt to use the inactivated LSD vaccine. Contact your veterinary doctor or veterinary office closest to your cattle post for more advice on control of LSD.

DISEASE DUE TO DIFFERENT CAUSES

Disturbed Urination

Disturbance of urine flow is observed almost exclusively in male cattle where the main cause is urinary stones or injures causing an obstructive of the urethra and congestion of the bladder and the kidneys with urine, resulting in uremia.

Causes;

Formation of urinary stones:

In cattle urinary stone is mainly induced by inappropriate composition of the fodder. The condition is rarely due to inflammatory causes.

A change in the equilibrium of the body salts excreted in the urine may be regarded as the starting point of stone formation where shift in the solubility ratio and the degree of acidity of the urine lead to the precipitation of small crystals(nidus) to which further crystals attach themselves and in the course of weeks or months, these crystals grow to the size of visible urinary stones, these stones may differ in color (white, yellow, brown, grey) and hardness according to their composition. In the majority of cases the stone consist of silicate, carbonate, phosphate or oxalate or mixture of salts. They irritated the mucosa of the bladder lead to inflammation and thus promote the formation of further stones. If unduly large or sharp-edged stones pass into the urethra, they may become wedged in the S-shaped curve of the penis or in the tip of the penis. Local irritation may then lead to spasms of the urethral muscles. The stones become even more firmly lodged and complete blockage of the urethra may result. In such cases, urination decreases initially to a trickle and then stop where congestion of the urinary tract with urine leads to over distention or rupture of the bladder and to uremia.

Injures due to kicks

Sometimes injures to the penis caused by kicks, which may be followed by disturbed urination. Such injures may occur if some bulls suddenly jump up while others remain lying down. This is frequently the case with fatigue animals after prolonged transport where the prepuce and the tip of the penis are particularly at risk from injures.

Clinical signs;



Figure 55. Penile urethral dilatation in a Calf. Calf with abdominal distension due to bladder rupture. Frog belly abdomen due to urethra rupture and Perineal urethrostomy.

Note: The Line of Separation between Non-Infiltrated and Infiltrated Tissues with Urine.

- Animals afflicted with urinary calculi may appear restless with frequent straining in unsuccessful attempts to urinate. Responses to extreme pain include tail wringing, stamping the feet, and kicking at the abdomen. In cases where urinary blockage is not complete, urine may dribble slowly from the sheath. Upon failure to pass the stone and after complete blockage of urine flow, the bladder or urethra finally ruptures, releasing urine into the body cavity and surrounding tissues.
- Animals with urethral rupture accumulate urine with swelling of the lower abdomen. Urinary bladder rupture results in urine accumulation in the abdominal cavity with fullness of the abdomen gradually developing. At these stages, animals may show loss of appetite and stand quietly or lie down, being reluctant to rise and death follows as illustrated in (figure 55).

Diagnosis

Accurate clinical signs and case history

- Both conditions are characterized by congestion of the bladder with urine.
- Rectal examination reveals the tightly filled bladder.
- With urethral injuries as a result of kicking, pronounced swelling on the lower abdomen and bluish black discoloration of the skin are observed.

- The condition appears suddenly where the animal suffers attack of severe colic and are restless, they refuse to feed but still drink water. The animal dies rapidly within few days due to progressive uremia.
- Post-mortem examination of animals with a ruptured bladder generally reveals blood-tinged fluid in the body cavity, severe infection in the abdominal cavity, inflammation of the urinary tract, and a hemorrhagic condition at the point of rupture. One or more stones can often be located at the point of blockage.

Treatment

Treatments designed to facilitate passing or dissolving the deposits have generally met with limited success. Surgery represents the most effective treatment, with the stone(s) being removed at the point of blockage. In rams, the point of blockage is often the filamentous urethral process at the end of the penis. In this instance, the urethral process and the accompanying stone may be surgically removed. In steers, the urethra and penis may be bisected and brought to the outside of the body to bypass the constricted portion of the tract. Steers treated in this manner often make acceptable weight gains for the remainder of the feeding period. Other surgical interventions, such as catheterization, have also been used. These operations require the skill of an experienced veterinarian.

Prevention Programs:

- Programs designed for prevention of urinary calculi have yielded disappointing results when initiated after the problem has already developed. While a calculi-prevention program may reduce the formation of additional deposits under these conditions, some losses may continue to occur from stones previously formed. In some cases stone analysis may be beneficial for prevention and treatment. Grazing heifers rather than steers may be a logical option in high incidence areas.
- In instances of urinary calculi outbreaks, attention is often focused upon the source of drinking water. However, assuming an adequate supply of potable water, there is no reason to suspect that the water may be a contributing factor. Further, the minerals (calcium and magnesium) contributing to water "hardness" are among the factors found to be protective against phosphate urinary calculi.

Diaphragmatic Hernia;

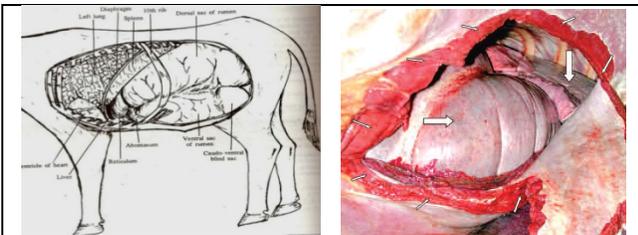


Figure.56.A *A photograph showing visceral content of thoracic and abdominal cavity of buffalo's .B. Postmortem photograph showing diaphragmatic hernia in cow.*

A break in the continuity of the diaphragm allows protrusion of abdominal viscera into the thorax. Diaphragmatic hernia (DH) is one of the thoraco-abdominal disorders inducing mortality in buffaloes. It has been described as a congenital or acquired condition in bovine. Generally, violent trauma and increase of intra-abdominal pressure constitute main causes of diaphragmatic hernia in animal,(figure 56).

Etiology Cause

- Trauma is a common cause of diaphragmatic hernia, although congenital defects of the diaphragm may also result in herniation (e.g., peritoneopericardial hernia).
- In cattle (especially in female buffaloes), there is rarely a history of trauma and hernias are reportedly associated with traumatic reticuloses, weak diaphragm.
- Increase intrabdominal pressure and musculotendinous junction (less ton and thickness).

Clinical signs:

- The clinical signs of diaphragmatic hernia are dullness, depression, in appetite, tympani, scanty black feces and regurgitation of food from mouth, these findings can be attributed to impaired process of eructation and rumination as a complication of reticular herniation.
- Respiratory distress and abnormal lung sound in cases of diaphragmatic hernia. Moreover signs of cardiac involvement. This might be due to lung irritation or heart displacement by herniated reticulum.

- Additionally, positive pain tests referred to high DH prevalence following traumatic reticuloperitonitis and if the stomach is herniated it may bloat and the animal may deteriorate rapidly.

Diagnosis:

An accurate clinical signs and case history

Decreased milk production, decreased appetite, exercise intolerance, scant feces, signs of impaction, coughing may be present and abduction of front limb is present.

- Reticular sound cranial to 6th rib is present,
- Scant black feces are more prominent signs.
- History of recent parturition.

Auscultation:

Careful physical examination, including auscultation and percussion, usually suggests the presence of thoracic disease.

- Intestinal sound on thoracic part is heard and muffled heart sound.
- The reticulum was first examined from area just behind xiphoid cartilage at ventral midline then both sides of chest from 3rd to 7th intercostals space (ICS).Reticular shape, contour and motility per 4 minutes as well as relation to adjacent organs were assessed.
- The definitive diagnosis is most frequently made from radiographs. Loss of diaphragmatic contour, abdominal viscera in the thorax and displacement of viscera from the abdomen may be apparent.

Radiographic contrast studies may be necessary to make the diagnosis.

- Barium may be given by mouth (GI series), or water-soluble contrast may be injected intraperitoneally (celiogram). Radiographs may be difficult to obtain in horses and cows; ultrasound is useful.
- Samples from abdominocentesis and thoracocentesis, electrocardiographs, and blood work may be obtained.
- Surgical exploration of the abdominal cavity may be necessary for definitive diagnosis in these species.

Treatment

- Surgical repair of the hernia is the only treatment. Other areas of trauma may be present. Optimally, the animal should be stabilized before surgery but from my

experience point it is bad prognosis and advice to slaughter.

Abortion

Abortion is the termination of pregnancy after organogenesis is complete but before the expelled fetus can survive. If pregnancy ends before organogenesis, it is called early embryonic death while dead full-term fetus is stillbirth.

Etiological Causes:

The etiological diagnosis in livestock is difficult and often frustrating task. Abortion often follows initial infection by week or month so that the causative agent is no longer apparent by the time abortion occurs.

Miscellaneous Causes: Drug-induced abortion (prostaglandins), Insemination/intra-uterine infusion, Hypothyroidism, Trauma/stress (transport, noise, veterinary treatment etc.), High fever and end-toxins (toxic plants, nitrate/nitrite, fungal toxins, other disease, Nutritional (malnutrition, vitamin A/selenium/vitamin E deficiency, and goiter), Twin pregnancy and genetic (malformation).

Both non-specific and specific infectious causes of abortion can lead to "abortion storms" in a herd, whereas the miscellaneous causes often result in sporadic, individual cases. Expulsion may follow fetal death by hours or days with lesions obscured by autolysis, fetal membrane and the aborted fetus.

Many sporadic abortion are likely the result of non infectious (i.e., toxic or genetic) causes about which much less is known in comparison to infectious causes.

Infectious Agent: Infectious agents are perhaps the most frequently though the common causes of animal abortion.

A. Bacterial causes.

1. *Actinomyces*, *Pyogenes* Bacillus, *Streptococcus* spp and other common bacteria found in the environment can be the cause of sporadic abortion in dairy herd.
2. *Brucella abortus* (Brucellosis, Bang's disease), where this bacteria once caused very significant reproductive problems on dairy farms.
3. *Leptospira* spp. Is usually associated with abortion outbreaks in the last trimester of gestation.
4. *Listera monocytogenes*. *Listera* can causes abortion in addition to some of the common disease seen due

to infection with bacteria. *Listeria* can be found in many places in the environment of dairy farms.

B. Viruses Causes:

- Bovine viral diarrhea virus (BVD). Infectious Bovine Rhinotracheitis virus (IBR).

C. Fungi can causes abortion

- Fungi can also cause abortion in dairy cattle; most often occur in the last 2 months of gestation.

D. Parasite can causes abortion

1. *Neospora caninum* is protozoa parasite that does not appear to cause any disease, immature cattle abortion usually occurs in herd in the middle of gestation (4–5 month).
2. *Trichomonas fetus* (Genital trichomoniasis) and *Campylobacter fetus* subspecies *venerealis* (commonly *C. venerealis*) called (vibrio) are the two organisms that are most often associated with venereal disease in cattle.

Clinical Signs;



Figure 57. Late period cow abortion and aborted fetus with placenta

The first abortion symptom is vaginal bleeding, which can range from spotting to being heavier than a period. A little spotting may be an early sign of abortion although fortunately this may amount to no more than a threatened abortion and the pregnancy continues. The second abortion symptom is pelvic pain. The third abortion symptom is cessation of pregnancy symptoms including udder tenderness, morning sickness and having to pass urine more frequently than usual as illustrated in (figure 57).

Sometimes there may be no sign or symptom to suggest abortion and pregnancy symptoms continue, and the abortion is only discovered in a routine scan, this is a missed abortion.

Sr. No.	Infectious Disease.	Samples for Diagnosis	Usual Stage of Gestation	Control Methods.
1	BVD	Fetus	Two months after Exposure	MLV vaccine to heifers.
2	Brucellosis	Fetus, placenta Serum of dam	Last half	Regulatory program; heifer vaccination; test / cull.
3	Campylobacter (Vibrio)	Cervical mucus, fetus, placenta preputial scraping.	early embryonic death or abortion	Vaccine for <i>C. venerealis</i> . Antibiotic treatment
4	Chlamydia	Placenta (& fetus)	Last trimester	Separation, sanitation
5	IBR	Fetus, placenta, Serum.	Last half	Vaccination program
6	Lepto	Urine of dam, fetus, Serum of dam,	Any stage	Vaccine, antibiotic
7	Neospora	Serum of dam; fetus (brain)	4–6 months	Dog control – fetal tissues and out of feed areas.
8	Sarcocystis	Caruncle from Uterus	Last trimester	Canine feces away from Feed.
9	Trich	Fetus, placenta cervical mucus, preputial scraping	Early embryonic death or under 5 months	cull infected bulls; control breeding; vaccine

Prevention of abortions

1. Proper hygienic and biosecurity measures in the cow's environment and feed storage,
2. Isolation of aborting cows and immediate removal of aborted materials,
3. Systematic evaluation of the feed for mycotoxins and other phytotoxins,
4. Adequate immunization against infectious diseases causing abortion
5. Maintenance of adequate breeding and treatment records to avoid insemination of pregnant cows and administration of drugs that may cause abortion to pregnant cows.

Foot Problems in Cattle

Foot health and lameness are major issues facing dairy producers because of their common occurrence and the tremendous economic losses incurred but early detection and prompt treatment can minimize the loss, improve recovery, and reduce animal suffering. Economic loss is mostly due to the foot problems not the treatment costs. Losses are often depending on the body weight loss, decreased milk production, dry matter intake and reproductive efficiency.

Disease of limbs and claws occur with all three forms of housing as the following.

1. Deep litter house which may be lead to.
 - Dislocation, sprain, contortion, foul in the foot, hematoma and abscess.
2. 2– Slotted floor house which may be lead to.
 - Cuts from rough or badly fixed planks.
 - Sores and callosities from lying down.
 - Ulcer of soles and balls.
 - Laceration of horn and claw bone fractures.
3. Stanchion stable which may be lead to.
 - Articular changes and postural anomalies especially in the foreleg.
 - Inflammation of the ankle joint.

The Bovine Hoof anatomy;

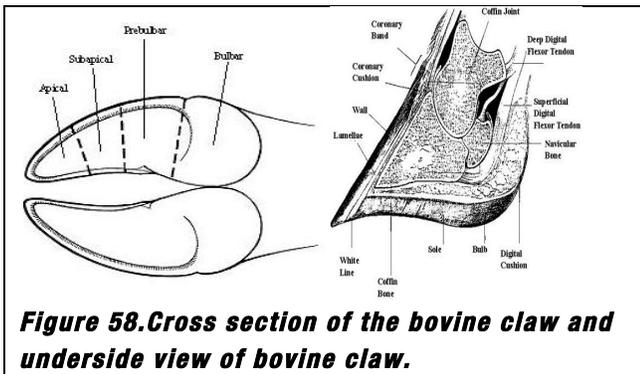


Figure 58. Cross section of the bovine claw and underside view of bovine claw.

The bovine hoof structure illustrated in (figure 58), the bovine hoof consists of a hard outer casing or hoof horn, the corium, which contains the blood vessels and horn forming cells and the skeletal portion of the foot. The coffin bone is the large, terminal, weight-bearing bone around which the hoof is formed and to which the tendons are attached. The hoof wall, sole and heel are made of keratin (like hair and the cow's horn) and water. They are not very thick and cover tissues which hold nerves and blood vessels. The junction between the horn forming tissues of the hoof wall and sole called the white line is located around the circumference of the bottom of the hoof. This area is susceptible to physical damage and bacterial invasion. The hoof grows from the corium at a rate of about 2 inches per year. The rate of growth depends on the genetics of the cow as well as the environment and nutrition of the cow. The rate of hoof growth is greater in the rear feet compared to the front feet. Weight distribution over the cow's feet is an important factor which will influence how her feet grow. The major weight bearing area of the foot is the outside part of the outside claw. This area absorbs the highest pressures during midstance. The cow's weight then moves towards the toes as she pushes forward. At this point there will be stretching of the white line. A hoof responds to heavier weight bearing by depositing greater amounts of keratin. In young animals, the weight is pretty evenly placed around the hooves. As the cow matures more weight is put on the outside walls of the rear feet. This is where they have more overgrowth. The inner walls of the front feet bear more weight as the cow matures.

The bulbs of the heels are not normally weight-bearing surfaces.

With exaggerated overgrowth of the hoof horn, the body weight shifts and the bulbs of the heel come into contact with the ground. The bulbs are like skin and are loaded with nerves and blood vessels, which make them more sensitive. Some cows with excessively long toes develop lameness because of bruising of the bulbs of the heel.

General causes of foot problem.

Mechanical injury or softening and thinning of the interdigital (between the toes) skin by puncture wounds or continuous exposure to wet conditions can provide entrance points for infectious agents.

A. Environment

1. Flooring

Free stall barns are more common on dairy farms and a problem which could lead to foot problems where the cows are on either concrete or brick surfaces for the majority of the day. Concrete surfaces generally cause more concussion to the foot than earthen surfaces. Whereas earthen surfaces absorb some of the shock associated with walking, concrete or stone transmits shock back to the foot. Extended exposure to concrete results in increased trauma to the foot such as excessive hoof wear caused by abrasive floor surfaces. Wet environments, such as wet free stalls, tend to create soft feet. Whereas very dry environments can result in brittle hoofs predisposing feet to cracking;

2. Stall comfort and type

A good indication of stall comfort in free stall barns (or pasture comfort on pastures) is the proportion of cows recumbent and chewing their cud, which should be about 90% plus of cows (this percentage is called the Stall Comfort Index – SCI).

3. Stage of lactation

The first 100 days after calving is the most stressful time during the cow's lactation cycle. This is also the most likely time for metabolic disturbances with hoof problems being no exception. Almost double the expected incidence of lameness will appear during this period which could directly be related to the high concentrates received in this period, but there are many more other reasons.

4. Age of animals

The incidence of lameness increases with age. For example: cows 3–4 years of age will have a 3% incidence of lameness and cows 9–10 years old will have an incidence of 13%.

5. Managing the cow's

If the flooring is too rough it must be smoothed out. If possible allow access to a well–drained, debris free earth lot daily. Try preventing cows from having to stand on concrete surfaces for extended periods, such as lengthy waits in holding areas before milking.

Types of Foot Problems

Foot infections, abscesses or sole ulcers may stem from cracks that result when feet are too soft or hard. Excessively soft feet are more suitable to occur in free stall systems from standing in manure and urine. This may result in heel and sole cracks allowing ulcers, abscesses or infections to occur. Excessively hard feet usually occur in stall–barns especially when kiln–dried shavings or sawdust are used for bedding. This may result in cracks at the top of the foot which may extend down from the hairline and allow infections relatively high in the foot.

Foot Rot

A smelly infection of the foot, which generally occurs high between the claws or toes, is referred to as foot rot. This results mainly from an infection caused by the bacterium, *Fusiformis necrophorus*. The organism may build–up in barnyards, exercise lots, mud–holes, and pastures.

Clinical Signs;



Figure 59. case of foot rot in a steer showing the swelling crack and dead tissue between the toes. Foot rot in a calf (seen from the bottom of the foot. Note how the swelling

is so severe that it pushes the toes apart). Foot rot in a calf has swelling of the entire foot from above the hoof to above the dewclaws and sole ulceration due to nail injuries

1. Cattle with foot rot show lameness, usually on one leg only but may be affected more than one leg.
2. The foot swells above the coronet and the toes spread.
3. Cracks and fissures develop in the interdigital space.
4. There are characteristic foul–smelling exudates at these fissures.
5. If left untreated, the infection can progress into the joint space or tendon sheath producing permanent damage as illustrated in (figure 59).

Treatment;

- **Immediately**
 - Affected animals should be separated from the herd and confined to prevent the spread of the organism.
 - The interdigital area should be washed and any loose necrotic tissue removed.
 - Topical dressings of antibiotics, sulfas or antiseptics have been used with success and if the infection has spread to deeper tissues a drawing ointment may be beneficial –Treatment consists of administration of systemic antibiotics and/or plus local therapy..
- **If feet are too soft** avoid having cows stand in moist sod for extended periods of time and allow cows to stand in dry soil or sand.
 - Routinely use a dry mineral mixture in a walk–through foot box. A formula of 80% hydrated lime, 15% copper sulfate, and 5% flowers of sulfur (acts as an antiseptic) can be used. Barn lime or superphosphate can be used on walkways.
- **If feet are too hard**, avoid having animals stand in soil or muddy areas for extended periods of time and allow cows to stand or graze on sod especially when it is moist or dew–laden but not soft or muddy. Barn lime or superphosphate should not be used on walkways.
- **For herd problems**, moist clay can be used in a foot box but slats may be necessary in the container to prevent slipping. Plain water can be used if it is drained and replaced frequently.

- **For individual problems**, a hoof ointment can be rubbed into the coronary band at the hairline of the foot.

Control and Prevention

Control of foot rot is important to minimize the economic impact of this contagious disease to the herd. Isolating individual cows, rigid sanitation in high-density areas and use of a foot bath has proven helpful in controlling the spread of foot rot. The foot bath should contain 5% copper sulfate. The depth of the solution should be at least four inches. The feet bath should be located where cattle must pass through it several times a day. An alternative to the foot bath is a dry bath containing 1 part copper sulfate to 9 parts hydrated lime.

Heel Erosions

Heel erosions or under run heels begin at the bulb of the heel. They start out as pits on the surface that can develop into parallel grooves that get filled in with black material and bacteria. The horn can separate at the grooves to form a “flap”. A new sole develops underneath and material becomes packed in between the layers. This condition is usually seen in confined cattle in wet, dirty lots. Overgrown hooves shift the weight toward the heels, exposing the heels to erosion, mostly in the hind claws.

Treatment

➤ Immediately

Treatment should be first directed toward removal of all the unsound horn. After cleaning the exposed area may be treated with a disinfectant liquid. The cow should be confined for several days until the newly exposed sole hardens.

- **In more severe cases in which sensitive tissue is exposed;**
 - A protective bandage applied over an astringent medication may be necessary in addition to confinement. Herd control involves genetic selection for strong feet and legs without excessive slope to the pastern.
 - Feet should be trimmed regularly and excessive exposure to wet environments should be avoided.
 - A dry foot bath may help toughen hoof sole as well as reducing the spread of infection.

Sole Ulcers

Sole ulcers are raw sores usually occurring on the inner side of the outside claw. It is a bulge of granular-like tissue sticking through the sole. Sole ulcers are usually associated with clinical manifestations of laminitis. A general rule of thumb is that if 10 percent of a herd has documented sole ulcers, the herd should be suspected for laminitis. However, there are other factors that can predispose cows to sole ulcers such as moisture and manure, excessive wear, and poor hoof trimming. Sole ulcers usually occur in both hind legs (figure 59).

Treatment

➤ Sole Ulcers

- The initial treatment of sole ulcers is to relieve the pressure on the ulcer.
- Therapeutic trimming consists of paring out the affected area around the ulcerated sole or lesion, which helps to relieve pressure and allows healing to occur.
- Topical application of copper sulfate and/or astringents followed by bandaging is necessary to control infection and prevent re-growth of the granulation tissue.
- Often times the ulcerated area has expanded to the point where no wall structure on the affected digit can be maintained. Therefore, it is often necessary to block or elevate the unaffected toe such that the pressure can be reduced on the affected toe.

Laminitis

Founder or laminitis can result in long overgrown and deformed feet or toes. Animals may appear quite lame or stiff and have difficulty in getting up and down. Hemorrhages can be found in the soles and walls of the feet. Infections, abscesses, or ulcers may occur when foreign material enters places where the wall and sole have separated. The highest incidence of laminitis often occurs during the first 100 days postpartum. Laminitis is an aseptic inflammation of the dermal layers inside the foot. There is usually some inflammation and sensitivity above the hoof and around the coronary band.

Clinical signs of an animal contracting laminitis;

1. Animal moving very stiffly and “crampy”. Standing on toes on the edge of stalls is very typical of a stance to alleviate pain.

2. Solar characteristics include sole hemorrhages and yellowish discoloration. Often, a white line separation (juncture between the sole and the outer keratinized wall) may be apparent.

3. Double soles and heel cracks may be present. However an animal may exhibit pain with no visible or apparent reason for lameness within a given foot.

Causes of laminitis

There is no one specific cause and laminitis may be associated with several, largely interdependent factors. Nutritional management is normally considered a key component in the development of laminitis, especially the feeding of increased fermentable carbohydrates, which leads to acidosis. Metabolic and digestive disorders can be predisposing factors. Hormonal changes associated with parturition and the lactation cycle can impact certain physiological changes. Infectious diseases, such as mastitis, metritis, and foot rot can impose specific endotoxic insults. Environmental aspects, such as hard surfaces, lack of or little use of bedding, and lack of or excessive exercise on undesirable surfaces can predispose animals to mechanical damage.

Acute Laminitis

A cow is systematically ill during acute laminitis. Inflammation of the corium is evident. The cow is prone to recurrences if the metabolic insults persist. The major local clinical signs in addition to intense pain include some swelling and temperatures that are slightly warmer than normal above the coronary band in the soft tissue area.

Subclinical Laminitis

This can be a long and slow process that is dependent upon persistency of low-grade insults. The inflammation that takes place ultimately results in internal hemorrhaging. As the horn tissue grows, the hemorrhagic area moves to the surface. The interval between the occurrence and appearance of the hemorrhage is related to the growth rate, which is about 0.20 inches per month. The thickness of the normal sole is about 0.40 inches. Therefore, the hemorrhage is seen about two months after the internal insult occurred. The occurrence of sole hemorrhages and yellow discolorations are signs that subclinical laminitis may be a herd problem. Sole hemorrhages can affect up to 50 to 60 percent of first calf heifers.

Chronic Laminitis

Several changes are associated with the localized area of the digit. The growth pattern of the keratinized horn is disrupted and the shape of the digit is altered. It becomes more elongated, flattened, and broadened. The surface of the claw is deeply grooved giving a rippled appearance. A dish-like appearance to the front of the hoof wall and sole are also characteristic. Internally, the coffin bone has separated from the front of the wall. Double soles with yellowish discoloration continue to be a major clinical sign. In severe situations, the bottom portion of the coffin bone can protrude through the corium and hard-horned tissue of the sole. Once the disease process has reached this point, the damage has been done and no therapy can return the foot to a normal configuration. The degree of chronic laminitis depends on the intensity and frequency of each acute episode and the degree of damage each preceding episode has caused as a result of the initial insult.

Digital Dermatitis

There are several scientific and common names to characterize the disease. They are heel warts, hairy foot warts, strawberry foot disease, raspberry heel, digital papillomatosis, and Mortellaro disease. Affected animals have pronounced lameness and spend excessive time lying down. First-calf heifers are often affected, and to a greater degree in the hind feet. There is little to no digital swelling with this disease.

Foot wart lesions look like raised, red and yellow patches and are usually located at the back of the foot above the heel. They are particularly painful and prone to bleeding when manipulated. Mature lesions are larger— up to two inches across, and usually raised with long, brown or grayish-black tufts of hair like projections along the surface. They have a hairy wart appearance. The hairs along the lesions are usually “true hairs”. The lesions can persist for many months. They may regress with dry weather. This disease is probably caused by a spirochete bacterium and it appears to be very contagious. The high morbidity of herds contracting this disease, as well as observations that greater than 90 percent of the lesions are highly responsive to antibiotics suggests an infectious agent. Environment may predispose animals to the foot wart agent. Examples would be wet free stalls, poorly drained lots etc. Spirochetes have been found in

digits of healthy cows, in affected herds, and in herds without incidence of digital dermatitis. It appears possible that many animals can be infected with the organism but show no evidence of lameness or lesions. When a specific stress or environmental component triggers the disease, it can then spread very rapidly.

Treatment

There are several treatment protocols that can be used, since the specific cause of foot warts is unknown, a specific treatment that always works is questionable. There are no treatments labeled for this condition thus the use of drugs requires a label and instructions from a veterinarian.

In the initial stage of the disease because of the pain, allowing animals to walk normally is critical. This means treatment of the infected area by removing debris from the specific lesion plus using a topical application of caustic chemicals and/or antibiotics.

- Topical oxtetracycline (soluble powder) and/or the injectable solution can be applied directly to the lesion. LS (Lincomycin /Spectinomycin)–50 powders or a solution has been used successfully. Treatments also have been sprayed on lesion areas.
- Topical sprays of iodine or iodized copper have proven to be effective for treatment and control. Caustic chemicals should be used with extreme caution. If they are overused on the lesion, they can cause serious lameness because of chemical burns on the skin.
- A concentrated foot bath along with topical antibiotic applications can be effective in controlling foot warts. A foot bath containing 9–10% solution of copper sulfate can help control foot warts and other infections.
- If feet get too hard, use foot baths less frequently or use 5% copper sulfate solution. –For large (greater than two inches in diameter), persistent, mature foot warts, surgical removal may be elected. The normal skin peripheral to the base of the wart–like structure must be excised around the entire circumference for the surgical procedure to be successful otherwise re–growth is common.

Toe Abscesses

Young cattle coming from lush pastures are prone to toe abscesses. These abscesses are caused by a sole penetration that leads to an infection under the hoof wall. The hoof is soft and easily worn down into the sensitive tissues, especially in the toe area.

Fall weaned calves, cattle with high moist pasture and cattle with abrasive surfaces and rough handling of the cattle often combine to create this problem.

Clinical Signs

The early symptoms of toe abscesses are very subtle. The cattle will appear sore and short–stride. The foot is not swollen in the early stages. Nearly all animals treated properly at this stage will recover, illustrated in (figure 59).

If the disease is allowed to progress, the animal will become noticeably lame. The animal may hold the most severely affected foot up and if the disease continues to progress, a slight swelling may be noticed at the top of the hoof.

When the animals are examined, the feet must be picked up and pressing your thumb on the side at the end of the toe, you should feel a soft area. You may also notice a crack between the hoof wall and the sole.

There should be no swelling between the toes. Swelling between the toes is a cardinal sign of foot rot and is totally unrelated to toe abscesses. Beef producers often make the mistake of treating all lame cattle for foot rot or upper leg injuries when toe abscesses may be part of the problem. If toe abscesses are not treated in time the toe will have to be amputated or the animal sold for salvage.

Treatment

➤ Immediately

- Toe abscesses are treated by trimming the end of the hoof just enough to relieve the pressure inside the hoof caused by the infection. If the animal bleeds when you trim the end of the hoof, you have trimmed too much.

➤ Medical treatment;

- Animals should be treated with a long–acting tetracycline. Antibiotics alone will not benefit the animal.
- The hoof must be trimmed.

Mechanical Injury to the Hoof

Hoof injuries are another cause of lameness. They are most often caused by poorly designed facilities or poorly maintained facilities. An animal's toe can be caught in the space between the ground and the wall in crowding facilities. If the animal steps forward with a toe caught, the hoof can be injured.

Equipment should be inspected every day before it is used. Loose metal can cause mechanical injuries to dozens of animals before it is detected. Mechanical injuries should not be left to heal on their own. A minor injury can become a severe local infection which can cause loss of animal performance.

Swollen Joints

Swollen joints usually fall into three categories; an infection that settles in the joint after an animal has a generalized infection; an injury to a joint; or an infection that develops in the joint after an infection in the foot was improperly treated. The most common joints involved are the front fetlock, the hock and the elbow. Stifle, hip and shoulder lameness is very rare in cattle. Regardless of the cause of the swollen joint, the three most common isolated bacteria are *Hemophilus somnus*, *Pasteurella multocida*, and *E. coli*.

Fracture Bones

If the animal is not under medication withdrawal time restrictions, it is best to salvage the animal as soon as possible. If time restrictions exist, and the fracture does not break the skin, the animal may get along quite well if kept in a small pen. The fracture will not heal, but it will allow the animal to clear the medication before marketing

Clinical Signs

The most obvious clinical sign is lameness (figure 59). There is inflammation and tissue death resulting in swelling and pain. There is usually a bad smell associated with foot rot. Cattle may stand with the foot raised, be reluctant to move, lose their appetite, lose weight, and have a low-grade fever and reduction in milk yield. Hind feet are affected most often and cattle tend to stand and walk on their toes. If left untreated, lameness becomes increasingly severe, with infection extending to the joints and other deeper structures of the foot.

Diagnosis

Accurate clinical symptoms, looking at the characteristic signs of sudden onset of lameness (usually one foot), elevated body temperature, interdigital swelling, and separation of the interdigital skin.

- Clinic examination of the feet.
- The conditions causing lameness must be identified if it is foot rot, interdigital dermatitis, sole ulcers, sole abscesses, sole abrasions, infected corns or fractures.
- Additionally, cattle grazing endophyte infected fescue pastures that develop fescue toxicity, causing loss of blood circulation to the feet and subsequent lameness, are sometimes mistaken as having foot rot.
- Cattle producers often diagnose any lameness associated with foot swelling as foot rot, but a more careful examination may reveal other causes of the swelling and lameness, such as injury or foreign bodies.

Treatment

The best way to help a lame cow, bull, or calf is often based on a preliminary diagnosis of the cause of lameness.

➤ Immediately

- In order to help animals with hoof-related lameness, it's often necessary to access the hoof for trimming or other procedures.
- The first lameness treatment idea depends on an antibiotic injection. However, of all the causes of cattle lameness only a couple of them respond to antibiotics. One of these conditions is foot rot. Lameness that originates from the foot with symmetrical swelling above the hoof and a foul-smelling draining sore between the toes can be expected to respond well to injections of long-acting antibiotics such as tetracycline.

➤ In some cases

- Antibiotics are also appropriate for joint infections. Not all swollen, painful joints are due to infections, though. In addition, systemic antibiotic injections veterinarians may choose procedures such as intra-articular (into the joint space) injections or flushing.

➤ In some cases of lameness

- Systemic antibiotics are ineffective. Therefore, an accurate diagnosis will prevent antibiotic treatments

that result in unnecessary expense and extended slaughter withdrawal times. One of these non-responsive conditions is hairy heel warts. For this condition, surface treatment with topical compounds such as tetracycline with or without bandaging is indicated.

➤ **For lameness cases caused by trauma (joint sprains, etc.)**

- The only feasible treatment may be time. Lame animals should ideally be isolated from other animals and provided deep bedding (straw or sand) until the time at which they are better able to move and bear weight on the affected limb.
- Anti-inflammatory medications can be administered for pain relief but they do not last for long periods of time.

➤ **Fractures and severe joint dislocations**

- In cattle are generally not conditions that can be successfully treated and timely salvage.
- Baby calves with fractures or dislocations low on the limb may be successfully treated with casts or heavy bandages.

➤ **Hoof-Care Treatments**

- Treatments can consist of hoof trimming, foot baths, and/or topical applications. Depending on the problem, a veterinarian and hoof trimmer should be consulted as to the best method of treatment. A combination of several treatment protocols may be necessary to correct individual and herd problems.

Restoring feet to proper hardness;

Prevention

Preventative measures include removing sources of injury and keeping feet dry and clean and feedlots should be well drained and manure removed frequently. In areas where cattle walk frequently such as in lanes or gateways grading or filling in low areas to provide a well-drained pathway for walking may help to prevent foot rot cases. Pouring a concrete pad or establishing solid pads around feed bunks and water troughs will help keep feet dry. In dairy cows, beef cows and bulls, regular foot care, including claw trimming as needed helps prevent foot diseases and injuries. Animals may also be walked through a foot bath containing copper sulfate, zinc sulfate or formalin. Footbaths are more commonly used and may be impractical for most beef herds.

Lameness is usually a multifactorial problem even though nutrition receives attention as being the main cause, other areas should be evaluated.

Nutrition

There are several areas in nutrition that can help reduce the risk of foot problems. They include carbohydrates, protein, trace minerals, and vitamins. Formulating the ideal ration to maintain good hoof health is not always enough. Nutrition should be weighed along with other factors in preventing bovine lameness from being a herd problem.

Carbohydrates

A major challenge regarding nutrition is a lack of information to specify threshold levels of carbohydrate that initiate nutritional insults such as acidosis. Carbohydrates constitute about 70 – 80 % of the dairy ration. The level and availability in various rations can have a substantial impact on ruminal metabolism. The amount of carbohydrates necessary to induce ruminal acidosis depends on the type of feed processing, the adaptation period, the nutritional status of the cow and the volume and frequency with which the carbohydrate is fed. Lactating cows need a minimum amount of forage in the ration. Forages should be included in the diet at no less than 1.40 % of body weight. In most situations, forage should make up no less than 40 –45% of the total ration dry matter.

Example: The average cow bodyweight is 1300 pounds and the total NDF in the ration is 32% on a dry matter basis.

A cow consuming 50 pounds of dry matter would be getting 16 pounds of total NDF (50 x .32) or 1.23% of bodyweight as total NDF.

A cow consuming 42 pounds of dry matter would be getting 13.4 pounds of total NDF (42 x .32) or 1.03% of body weight as total NDF.

Protein

The amount of protein in the ration has been suggested to influence the incidence of laminitis. Several studies have shown that high percentages of ruminally degraded protein have been identified in association with lameness and laminitis. However, the role of protein is still unclear.

Little information is available to identify what role protein might play in the development of lameness. Several postulations involve allergic histaminic reactions

to certain types of proteins or a link between high protein supplementation and protein degradation end products.

Trace Minerals

Copper is essential for the production of healthy claw horn. A copper deficiency can interfere with the synthesis of keratin, inhibiting development of the horn tissue.

Zinc is essential for horn production and plays an important role in immunity. The effect of zinc on bovine lameness is normally related to wound healing, epithelial tissue repair, hoof hardness, and maintenance of cellular integrity.

Many nutritionists formulate rations with higher levels of trace minerals than what NRC recommends to take into account stress related problems due to increased milk production and/or disease.

Vitamins

Vitamin A, β -carotene, vitamin E and biotin are of concern when studying factors related to cattle lameness. Vitamin A is important in the maintenance of epithelial tissue and cell replication. β -carotene is thought to play a role in epithelial tissue repair, integrity and immune function.

Vitamin E is involved in helping cells maintain integrity and in the immune process. Its major role is that of an antioxidant.

Biotin is associated with the formation of the hoof horn. It is important in claw hardness. If rations are high in concentrates, the synthesis of biotin in the rumen is reduced. The current recommendation is to supplement 20 mg per day of biotin through lactation and 10 mg per day for dry cows.

Stall Comfort

Adequate stall space should be provided to allow reclining and ruminating for about 10 – 14 hours per day. The dimensions of the stall must be proper for the size of the animal that is being housed. Large cows need a stall length of seven to eight feet. The width can range from 42 to 50 inches depending on the animal size (heifer versus cow). The lower the curb height (not less than six inches), the less chance a cow has of standing in the passageway.

Soft bedding is essential. Sand is optimal stall bedding, providing cows comfort and traction. In addition, sand must be free of small stones, which can penetrate the sole horn. An earthen base with shredded tires covered with polyethylene sheets also works in providing

a cushioned base. However, material must not be of an abrasive nature and must not scrape hocks or knees as cows rise and lie down. The use of sawdust with wood chips on these polyethylene surfaces can be abrasive and cause hock lesions.

Hoof Trimming

Regular hoof trimming may increase the functional life of a dairy cow by lactation. Correctly trimming cow's feet can give the claw stability and enable the cow to distribute weight equally between the claws.

Routine trimming, which removes even small amounts of the horn from the sole, can stimulate horn producing tissues. This can accelerate production of a new healthy horn. It is recommended to trim feet at least once or twice a year. The ideal times would be once at dry-off and again around 100 days in milk.

A professional hoof trimmer who uses correct equipment and procedures should be employed. Good record keeping is key to monitoring a cow's condition.

Torsion of Uterus;

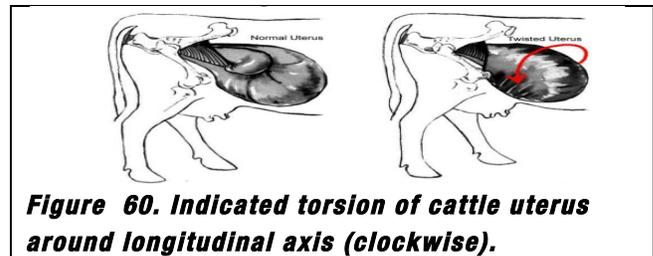


Figure 60. Indicated torsion of cattle uterus around longitudinal axis (clockwise).

Torsion of uterus is the rotation of pregnant uterus on its longitudinal axis. Uterine torsion was diagnosed in domesticated species like cattle, buffalo, doe, ewe, llama, camel, mare, bitch and queen, and even in laboratory species like rabbit and guinea pig. In bitch and queen, torsion is limited to a uterine horn or a part of the horn, whereas in the remaining domestic species especially buffaloes, torsion is of the uterine body. The importance of this condition in buffaloes can be judged from the fact that about 67–83 per cent of difficult parturition affected cattle presented at referral hospitals suffer from uterine torsion. The striking feature of torsion of uterus in cattle is its association with advanced pregnancy and process of parturition. Usually, torsion of uterus occurs before the onset or during the late first stage of parturition.

Occasionally, torsion of uterus can occur around 5th to 8th month of pregnancy.

Predisposition of Cattle Uterus to Torsion

Reassessment of various speculations made for justifying the higher incidence of uterine torsion in cattle in comparison to other species has produced some realistic explanations. Small quantity of fetal fluids and associated decrease in size of uterus at the end of pregnancy seems to be a realistic justification for the occurrence of uterine torsion. Destabilizing factors such as weak broad ligament musculature, lower tone of uterine muscles along with sudden movements of dam and fetus can further add up to probability of occurrence of torsion of uterus.

Maternal Destabilizing Factors

Attachment of broad ligaments (figure 60):

Uterus is held in position by two folds of peritoneum called broad ligaments. Cattle uterus is conducive to torsion during last trimester of pregnancy because of its relatively unstable anatomical arrangement. On one side, broad ligaments are attached at sub-iliac region and on the other side along the lesser (ventral) curvature of uterus which leaves greater (dorsal) curvature free. Also, uterine horns are not fixed by broad ligaments but are lying free. As pregnancy advances, there is a relatively small increase in the length of broad ligaments but the pregnant horn extends massively beyond the area of attachment. In addition, poor musculature of broad ligaments makes the pregnant uterus less stable in cattle.

Unfilled Rumen

Role of rumen in preventing torsion of uterus is evident from the fact that presence of rumen on left side increases the incidence of right side uterine torsion. If rumen is unfilled, space in the abdominal cavity is increased and relatively unstable pregnant uterus gets predisposed to torsion.

Body frame

Capacious and pendulous abdomen of buffaloes facilitates easy rotation of pregnant uterus in buffaloes compared to cattle.

Age

About 70–77% torsions occur in pluriparous and 23–30% in primiparous cattle due to larger abdominal cavity, stretching of pelvic ligaments, loose and long broad

ligaments together with loosening of uterine tissue and decreased uterine tone in old age.

Sudden Movements

In case of sudden fall, sudden push from other animal and bumpy movements during transportation, the fetus in advanced pregnant uterus may respond with violent movements, and thereafter the heavy uterus may take time to return to its original position, while dam may change their position quickly to expose unstable pregnant uterus to torsion. In addition, while lying down, cattle go down on fore legs first and while getting up, the hindquarters are relevant first; the pregnant uterus is temporarily suspended in the abdominal cavity and is prone to torsion. However, other contributory factors must be present in addition to instability that occurs during sudden movements of dam; otherwise uterine torsion would have been frequent in advanced pregnant buffaloes compared to during the first stage of parturition.

Fetal contributory factors

- i. ***Calf birth weight:*** About 90% animals with uterine torsion deliver calves which have birth weight above breed average. During normal parturition, average size fetus is able to rotate and flex its limbs within the boundaries of uterine wall, however, when fetus is oversized, fetal limbs may get entangled in the uterine wall and the continued vigorous movements of fetus may lead to rotation of uterus.
- ii. ***Reduced amount of amniotic fluid:*** About 90% animals with uterine torsion deliver calves which have birth weight above breed average. During normal parturition, average size fetus is able to rotate and flex its limbs within the boundaries of uterine wall, however, when fetus is oversized, fetal limbs may get entangled in the uterine wall and the continued vigorous movements of fetus may lead to rotation of uterus.
- iii. ***Fetal movements and uterine tone:*** About 90% uterine torsions are encountered during the late first stage of parturition process. At this stage, conditions favorable for torsion are created because cervix has started to dilate and uterus has begun to contract and gets molded on fetus. The forces impulsive for the rotation of unstable uterus are strong intrauterine movements of fetus that are invoked by myometrial

contractions, changes in intra-uterine pressure as well as changes in fetal blood flow. In addition, at this stage, uterine muscles are not in much tone, thus uterus is not able to restrict the movement of upper portion of fetus and the relaxed and unstable uterus may be a cause for torsion of uterus. In fact, uterine instability may induce torsion only up to 180° , whereas torsions of $\geq 360^\circ$ require active fetal movements.

Clinical Signs

1. Cow around the parturition or pregnancy above 5–6 months,
2. Off food cow and signs of colic/distress animal (cow up and down).
3. Cow suffering from dysuria (dripping urine drop by drop)

Diagnosis;

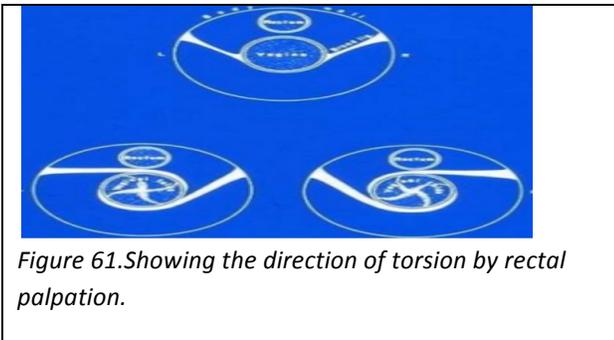


Figure 61. Showing the direction of torsion by rectal palpation.

1. Accurate clinical signs and case history:
2. Rectal and pelvic examination. It must be carried with more care to detect the direction of torsion to help you to choose the side of dam rolling as illustrated in (figure 61).

In Early Cases

Typical history of a case of torsion of uterus will suggest that cattle was about to calve (as exhibited by letdown of milk and relaxation of pelvic ligaments), but adequate time has passed and there is no appearance of water bags or fetus. On the contrary, dam has become restless (frequently gets up and down) with severe abdominal pain (due to stretching of the broad ligament) as manifested by kicking of the abdomen with her hind legs.

In Delayed Cases

If the uterus is not detorted during this period, then the history will suggest that continuous straining initially

exhibited by cattle to deliver the fetus has ceased followed by tightening of pelvic ligaments and reabsorption of milk. If the condition remains undetected for several days, then appetite diminishes and rumination ceases. Based upon the symptoms of abdominal pain and discomfort of dam, farmers are usually misguided by the unqualified practitioners by treating the case of torsion of uterus as a simple case of digestive disorder and are usually treated for the same. Later on, when there is no improvement, animal is diagnosed with uterine torsion and is referred. In cattle with uterine torsion of <36 h and 36–72 h, pelvic ligament relaxation and mammary gland engorgement are usually evident in 90 and 37% cases, respectively. Beyond 72 h, milk usually gets reabsorbed and pelvic ligament are tightened in 80% torsions.

Treatment;

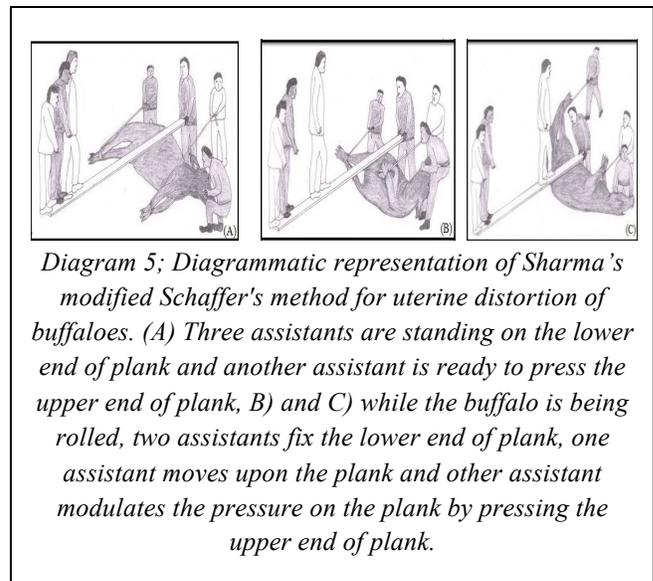


Diagram 5; Diagrammatic representation of Sharma's modified Schaffer's method for uterine distortion of buffaloes. (A) Three assistants are standing on the lower end of plank and another assistant is ready to press the upper end of plank, (B) and (C) while the buffalo is being rolled, two assistants fix the lower end of plank, one assistant moves upon the plank and other assistant modulates the pressure on the plank by pressing the upper end of plank.

- **Immediately**
 - At first must be detected the direction and degree and amount of cervical dilatation by pelvic examination.
- **Per-Vaginal Rotation of Fetus**
 - Degree of torsion and the amount of cervical dilatation are critical factors for the success of this method. With rotations of $\leq 90^\circ$, the fetus is easily rocked manually into a normal position. Success rate is high if dam is standing, cervix is sufficiently dilated to grasp the fetus and the fetus is live.

➤ **Rolling of Dam**

- The method used for distortion of uterus in buffaloes is Sharma's modified Schaffer's method (Diagram 5). This method was designed for cattle due to their thick skin which causes skidding of plank during distortion of uterus and their pendulous abdomen which warrants greater pressure for fixation of pregnant uterus. Theory is to rotate the dam to the same degree and direction to which the uterus has rotated, keeping the fetus fixed by fixing uterus with a plank (length: 11.9 feet, width: 9 inch and thickness: 2 inch). In brief, after ascertaining the side of torsion, animal is casted carefully in lateral recumbence on the side of direction of torsion and the front and hind legs are secured separately. The plank is placed on the upper paralumbar fossa of dam in an inclined manner with lower end on ground. Next step is to quickly roll over the dam on to its back. For this, the front and hind legs are pulled up and over the recumbent dam. While rolling, plank is anchored by 1–2 medium weight assistants who

stand still upon the lower end of plank and another assistant moves on the plank. An additional assistant modulates the pressure on the plank by pressing the upper end of plank (Diagram 5). Sharma's modified Schaffer's method of distortion was developed based upon the principle of lever (fulcrum, load and effort). In this, fulcrum is lower end of plank that does not move, load is the weight of assistants standing and moving on the plank and effort is the force used by the assistant on the upper end of plank. After each roll, effectiveness of roll is judged. If the roll is successful, disappearance of the vaginal spirals or rectal pouch can be immediately palpated. If the roll is not successful, then whole procedure needs to be repeated.

➤ **CaesareanSection**

- Caesarean is usually attempted in 11–26% torsions, in which all other methods of distortion had failed or there is failure of complete cervical dilatation subsequent to successful distortion.

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