Ethiopian Veterinary Association

Cattle (Dairy, Beef) Health Management
Continuous Professional Development (CPD) Training Manual

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Module Introduction

Module title: Animal Health Management Module (Dairy, Beef, Small Ruminants, Poultry)

Module code:

Credit points:

Target trainees:

- Veterinarians with DVM degree and
- Other Animal Health Professionals with BVSc or equivalent degree

Module Format

Module Description

Animal Health Management module contains three parts: Dairy and Beef Cattle Health Management Module; Small Ruminant Health Management Module and Poultry Health Management Module.

The module begins with introduction of the meaning and concept of herd health management and its role in early detection and management of animal health problems. It deals with monitoring herds/flocks for collection of important information that, upon analysis, would help early identification of health and production problems, and associated factors. The module describes about how the veterinarian would design and implement a particular health management scheme, analyse the findings and devise methods to counter the problems. The topics covered in the module include: the concept of herd/flock health management, monitoring for certain health problems, young stock health management, reproductive health management, common diseases and their diagnosis and control/prevention, and immunization.

***Module Duration*:**

- 40 hours (16 hours face-to-face or on-line theory divided over 4 weeks- 4 hours a day per week, and 24 hrs face-to-face practical session- 3 hrs a day for 8 days).

Learning Objectives

The CPD training will

- Discuss the principles, components and importance of herd/flock health management
- Explain the need for routine monitoring of animal health for early detection and management of animal health problems

- Enable trainees to design and implement herd/flock health monitoring for some important animal health problems
- Discuss common diseases of cattle, small ruminants and chickens (Etiology, Clinical presentation, Diagnosis, Prevention/Control)
- Explain the importance and application of herd health monitoring in improving reproductive performance, young stock health and survival, and foot and claw health (in dairy cattle)
- Enable candidates to design and implement relevant vaccination protocol for a herd
- Discuss the principles and importance of biosecurity in disease prevention/control

Learning outcomes

By the end of this CPD training, candidates should be able to:

- Demonstrate an understanding of the basic components of herd/ flock health management programmes
- Understand the importance of herd/flock health management for profitable, environmentally sound and quality/safe animal production
- Appreciate the importance and design and implement health monitoring/management in dairy cattle, beef cattle, sheep and goat, and chicken flocks/ herds/ (routine monitoring/ collection of information, analysis of the problem and design appropriate corrective measures)
    - Identify health parameters for monitoring herd/flock health /disease problems
    - Design and implement postpartum health monitoring
    - Design and implement young stock health monitoring for effective rearing
    - Monitor and manage diseases affecting reproductive performance
- Recognize, diagnose and control important diseases
- Recommend/develop relevant vaccination protocols
- Demonstrate sound knowledge of biosecurity and hygiene
- Perform post-mortem dissection of birds and diagnosis of some diseases with this technique
- Convince farmers to engage in herd health programmes

Content

For dairy cattle, beef cattle, sheep and goats, and chicken

- The concept of herd/flock health management

- Monitoring for certain health problems
- Young stock health management
- Reproductive health management
- Common diseases and their diagnosis, control/prevention
    - Viral diseases
    - Bacterial diseases
    - Parasitic diseases
    - Metabolic
- Immunity and vaccination

Learning Approach

The learning approaches for this module are:

- Theoretical/lecture classes (face-to-face or virtual, using power point presentation, Soft copies, Pictures)
- Group discussions
- Individual and group exercises
- Farm visits
- Clinic visits
- Case studies
- Demonstrations
- Pictures
- Hands-on training

Measurement of learning

- Summary and daily recaps for key learning points
- Assignment (individual or group)
- Practical class report
- Farm visit report
- Case report/book (after clinic visit)
- Presentation of the reports

- Pre and post training evaluation- written exam at the beginning and at the end of the training - to assess the learning objectives compared to the entry knowledge

CPD module assessment

- The module will be assessed by feedbacks from trainees
- It will also be assessed by the performance of the trainees

Required reading materials

- da Silva, J.C. and Noordhuizen, J., 2013. Dairy herd health management: rationale and tools -*Veterinary Skills*-, Conference Paper
- da Silva, J.C., Noordhuizen, J.P.T.M., Vagneur, M., Bexiga, R., Gelfert, C.C. and Baumgartner, W., 2006. Veterinary dairy herd health management in Europe constraints and perspectives. *Veterinary Quarterly*, *28*(1), 23-32.
- De Kruif, A., G. Opsomer, and J. P. Noordhuizen. 2007. Dairy herd health management: Current state and perspectives. Pages 337– 350 in Proc. Production Diseases in Farm Animals. Merkur druck und. Kopier, Leipzig, Germany.
- Grove-White, D., 2004. Health care in the modern dairy herd. In Practice, July/August 2004, 368-376.
- LeBlanc, S.J., Lissemore, K.D., Kelton, D.F., Duffield, T.F., Leslie, K.E., 2006. Major advances in disease prevention in dairy cattle. J. Dairy Sci. 89, 1267–1279.
- Noordhuizen, J.P., 2006. Assessing dairy cattle health worldwide. XXIV World Buiatrics Congress, Nice, France.
- Radostits, O.M., 2001. Herd Health. WB Saunders Company, Philadelphia.
- Radostits et al., 2007. A Textbook of the Diseases of Cattle, Sheep, Goats, Pigs and Horses, 10th edition
- Risco, C.A. and Melendez, P., 2011. Dairy Production Medicine. John Wiley & Sons Ltd, Chichester, West Sussex, UK. 363p.Moran, J. 2002. Calf Rearing: A Practical Guide. 2nd Edn., Landlinks Press, Collingwood Vic., Australia. 211p.
- Swayne, D.E., 2013. *Diseases of poultry*. 13th Edn., John Wiley & Sons Inc., 1394p.
- Sibley, R., 2006. Developing health plans for the dairy herd. *In Practice*, *28*(3), 114-121.
- The Merck Veterinary Manual

I. Cattle Health Management Module

1. Introduction

Ethiopia has huge cattle population estimated at 60.4 million, however only a small fraction (about 1.8%) are improved (exotic or crossbred dairy) animals (CSA, 2018). There is unsatisfied demand for milk and dairy products in Ethiopia, which will only grow in the future unless substantial efforts are made to improve productivity of the sector (Shapiro et al., 2017). Dairy production in Ethiopia is classified into urban, peri-urban and rural production systems (Redda, 2001). Rural dairy system corresponds to the traditional system and includes pastoralist/agropastoralist and mixed crop-livestock systems. The sector is largely dependent on low producing indigenous cattle breeds. The urban and peri-urban systems are located in or in the proximity of Addis Ababa and other regional towns. The sector includes small to large, mostly intensively managed, commercial dairy farms based on the use of grade and crossbred animals (Redda, 2001; Ahmed et al., 2004). Improving milk production in Ethiopia largely entails significant increase in the number of crossbred dairy animals and intensification of production, among other measures (Shapiro et al., 2017).

There are no improved beef cattle breeds in the country. As a result, meat from cattle in Ethiopia is almost exclusively derived from indigenous cattle breeds. These indigenous cattle are kept in the mixed crop-livestock production system practiced in the highlands of Ethiopia and in the pastoral/agro-pastoral production system in the lowlands of the country. Cattle in these areas are generally raised for multiple purposes; including milk and meat production among others. There is no practice of specialized beef cattle production with specialized beef breeds in Ethiopia. Cattle can go directly from pasture to the slaughterhouse (particularly from the pastoral areas), or may find their way to backyard fattening practiced in many places in the country, or may pass through feedlots before they are sent to the slaughterhouse. Commercial feedlots are not many in Ethiopia and are geographically limited to certain parts of the country (E.g. Adama area), where agricultural by-products are available in relative abundance.

Animal health plays a crucial role in the dairy farm, as sick cows show poor feed conversion, loss of production, and often have a very low body condition score. Changes in animal health management, in the developed world, have led to a shift in focus from disease treatment to prevention and from the individual animal to herds (LeBlanc et al., 2006). Because prevention of diseases is better for the profitability of the farm and the welfare of the animals than treating animals as diseases occur. Each diseased cow or group of cows should be detected early enough to avoid financial losses or such losses should be prevented altogether by detecting and managing risk factors contributing to disease occurrence. Herd health management (HHM) in dairy cattle may be defined as a combination of advice on animal health, milk production, and disease prevention, placed in a framework of farm economics, welfare, food safety, and environment (Derks et al., 2013). It has three components: routine monitoring, recording and problem analysis and prevention (da Silva

and Noordhuizen, 2013).

A conventional HHM program is based on regular scheduled farm visits, the monitoring of animals and their environment, the recording and analysis of animal health and production data, and the provision and coordination of advice by the veterinarian (De Kruif et al., 2007). For example, farmers participating in HHM in the Netherlands are visited every 4 to 6 wk by their veterinarian, who checks the animals and herd management to intervene in a proactive way with problems regarding animal health and animal welfare (Derks et al., 2013). In a HHP the veterinarian plays that of an advisory role. The veterinarian trains farm personnel, employees, and owners about important health indicator parameters to be observed and recorded regularly. Good record keeping is a prerequisite to the execution of HHM program.

Ideally a dairy herd health program related to prevention consists of the following parts: fertility, udder health and milk quality, nutrition and metabolic diseases, control of infectious diseases, lameness, housing, health of calves and heifers, animal welfare, the use of medicines and food safety (Radostits, 2001).

2. Herd Health Monitoring in Cattle

(Adapted from Risco and Melendez, 2011)

Learning outcomes
By the end of this chapter, candidates should be able to:

- Understand the importance of monitoring in cattle health management
- Plan and implement health monitoring in cattle herds
- Identify important parameters for health monitoring in cattle herds

2.1. Health Monitoring in Dairy Cattle

An important concept in dairy herd health is the early diagnosis and treatment of sick cows, which needs routine monitoring of the herds for collection of data. Monitoring of health refers to a practical methodology to routinely, rapidly, cheaply and effectively gather sufficiently reliable information about certain issues in both dairy cattle and their direct environment with the ultimate goal of interpreting the findings properly and make inferences about the question whether there are (pending or prevalent) health problems in the herd. This will subsequently be the basis to design a plan of actions for the shorter and the longer term (Noordhuizen, 2006).

Health records enable producers to track diseases, treatments and vaccination history. Health records also can be used as an indicator of where to focus efforts and improve production status. Records from health examination enable producers to identify the signs of disease early. Early detection of disease symptoms allows them to make treatment decision early, stop disease progression and even reduce the disease incidence

rate on the farm (Sen, 2021). HHP in dairy cattle is implemented at farm/herd level. A committed farmer which actively participates in setting the targets of the program and pays for the work is an essential requirement of a successful program. On the other hand, it is impossible to implement a similar programme in traditional herds in Ethiopia, as it is highly unlikely that the subsistence farmers would be willing to pay for the work. The main purpose of veterinary service for such herds is still epidemic control, and diagnosis and treatment of sick animals.

2.2. Health Monitoring in Traditional Cattle Herds

Disease monitoring in the traditional livestock production systems should involve selected (sample) herds representative of exposure to various risk factors such as climatic conditions at various implementation levels e.g., district level. Monitoring for diseases in these herds should be the responsibility of the public veterinary services, as smallholder farmers and pastoralists in these systems are highly unlikely to be willing to pay for the service. However, they may be responsible and pay for the interventions (e.g. vaccination, deworming etc.) resulting thereof, if they decide to use the service.

Animal health monitoring in the traditional herds can be designed by veterinarians working at district level and may be implemented mainly by animal health paraprofessionals (AHP) working at the *kebele* level, with possible periodic visits by district veterinarians. The information gathered by the *kebele* AHP is then channeled to the District Veterinary Office for analysis and interpretation, and designing a plan of action. Collection of data on husbandry practices and the environment should be encouraged because they may have association with disease occurrence. The monitoring should not be limited to cattle herds and should include small ruminants for the sake of economic efficiency. Owners of the herds selected for the monitoring can be trained on identification/recognition of certain manifestations of diseases (to be recalled or recorded).

As it is not feasible to include all the herds in the monitoring scheme, reasonable number of representative herds should be selected for the monitoring. The selected herds are visited periodically (say every month by *kebele* AHP and every 6 months by district veterinarian) for collection and recording of some health, production, reproduction and management parameters.

The record may include herd size, births, deaths, sales, slaughters, new introductions, and general and specific signs of diseases. Signs of diseases of interest may include: demeanor/attitude, appetite, coughing, lacrimation, salivation, nasal discharge, difficultly breathing, diarrhea, skin and coat condition and body condition. Information on abortions, retained fetal membranes and vaginal discharge, and external parasite infestation should be recorded. Age at first calving and calving interval would help to assess the reproductive performance of herds. These data are collected by interviewing the owners and direct observation by the AHP and district veterinarians during the periodic visits. Routine examination of fecal samples, at least twice a year, would help to known the status of gastrointestinal parasitosis in the area. Other types of samples, as the

clinical signs suggest, may be collected to establish diagnosis.

A dairy herd health program, for which a lot of literature exists, is recommended to consist: fertility, udder health and milk quality, nutrition and metabolic diseases, control of infectious diseases, lameness, and health of calves and heifers (de Kruif *et al.* 1998 ; Radostits, 2001). Herd health management of traditional herds in Ethiopia, form which the vast majority of slaughter animals come, should include health of calves, control of infectious diseases, and control of internal and external parasites.

2.3. Postpartum Health Monitoring in Dairy Cows

Postpartum health monitoring programs have become popular on dairy farms. The premise for monitoring postpartum health in cows is to identify any change that occurs from what is considered a normal state. Monitoring health early postpartum assures that all cows on farms are examined during the time when they are most susceptible to disease, allowing the opportunity for early identification and management of cows that are sick. Additionally, they are implemented to prevent diseases; a cow diagnosed with metritis and treated can help prevent the development of ketosis and displaced abomasum. Monitoring postpartum health involves the evaluation of cows by trained farm personnel during the early postpartum period (first 3 weeks after calving) to identify sick cows followed by a physical examination to make a disease (metritis, ketosis, displaced abomasum, and mastitis) diagnosis and provide treatment. Food animal veterinarians play a major role in these programs, and their primary role on many dairy farms is no longer to identify and treat sick cows but to develop, implement, and supervise the application of these programs (Risco, 2011).

There are different opinions on health monitoring strategies, which health parameters to use, and how to implement them. Health parameters that can be used include rectal temperature, attitude, milk production, uterine discharge, and presence of ketone bodies in blood, milk, or urine.

Look for sick cows beyond the postpartum period: It is important to recognize that sick cow monitoring must be performed in all cows in lactation. Farm personnel involved in moving, feeding, milking, or breeding cows should be cognizant of the fact that they play a major role in the identification of sick cows. Consequently, they too should be trained in how to look for sick cows. Milkers should also be well trained in how to identify cows with mastitis, as it is an important component in good milking procedures.

3. Management of Calves from Birth to Weaning

(Adapted from Moran, 2002 and McGuirk, 2011)

Learning outcomes
After completing this chapter trainees should:

- Appreciate the importance of perinatal care for calves
- Be able to assess the health status of calves
- Be able to care for weak calves
- Understand the importance of colostrum feeding for the health and future productivity of calves
- Identify, manage and control major heath problems of calves

The perinatal period is one of the most vulnerable times in the life of the calf, and attention to the details of calving and calf viability immediately after birth can prevent the losses commonly encountered in the first 48 h of life. It is apparent that early life management has long-term affects on the health, productivity, and profitability of replacement dairy heifers. For effective health management of calves, the importance of implementing a comprehensive calf record system cannot be overemphasized. Management practices based on calf data consistently gathered, entered, analyzed, and monitored is used to make informed decisions, identify trends, analyze problems, test solutions, and improve profit.

3.1. Perinatal Care

The perinatal period, defined as the first 48 h of a calf ’s life, is one of the most vulnerable times in the life of the calf. Careful attention to the details of calving and calf viability immediately after birth can prevent death. Defined protocols and consistently executed actions that remove the calf from the calving environment, administer quality colostrum, attend to navel care, and identify the high-risk calves will have an immediate and future impact on health, growth, productivity, profitability, and longevity in the herd.

Supervision of newborn calves by persons knowledgeable, capable, and caring must be a priority to improve survival and the health of newborn dairy calves. Knowledge of a timeline for expected newborn calf behaviors and vital sign parameters as shown in Table 1 (Mee, 2008) can provide basis for assessment of calf vitality.

Table 1. Timeline for newborn calf behavior and vital signs (Mee, 2008)

Time	Expectations of a normal dairy calf
Birth	Haircoat covered with placenta but not discolored
Within minutes	Responds to stimulation with vigorous head shake
Less than 5 min	Head righting
5 min	Sitting in sternal recumbency
Within 15 min	Making attempts to stand
Within 1 h	Standing
Within 2 h	Suckling
Within 1 h	Rectal temperature stable at 102–103°F (39–39.5°C)
Within 1 h	Heart rate 100–150 bpm
Within 1 h	Respiratory rate 50–75 breaths/min primarily from chest movements and no open-mouth gasping

A modified APGAR (appearance, pulse, grimace, activity, respiration) scoring system shown in Table 2 that correlated well with blood lactate levels (Sorge et al., 2009) can be used to assess the vitality of newborn calves. Low-scoring calves by APGAR (Sorge et al., 2009) are considered high risk. The high-risk calf requires immediate action and, later, should be readily identified and subjected to heightened observation and regular screening for disease.

Table 2. A modified APGAR scoring system to assess newborn calves (Sorge et al., 2009)

Reaction tested	Score 0	Score 1	Score 2
Cold water on the head	No reaction	Reduced or delayed reaction	Lifting, shaking of head
Squeezing between claws	No reaction	Withdraws foot slowly, weakly	Immediate and strong withdrawal
Mucous membrane color	White or blue color	Pale, grayish, or blue-tinged	Pink, moist
Respiratory pattern	Not breathing	Irregular frequency and pattern of breathing	Regular frequency and pattern of breathing

Procedures for resuscitation of newborn calves must be clearly established, the methods thoroughly understood, and equipment readily available in the calving area. Identified by flaccid muscles, lack of responsiveness to stimulation, or bluish discoloration of the mucous membranes, these calves are placed onto a low platform table. The head and neck should be placed over the edge of the table to allow 10-15 s for drainage of fluid from the mouth and nose. The calf is pulled back onto the table and placed in a sitting position. The topline of the calf is vigorously rubbed with a dry towel, beginning at the tail and moving forward toward the head. At the head of the calf, dry the nose, clearing additional mucus or fluid from the nares. Towel stimulation is then directed to the ears and eyelids.

Newborn calves should be removed from the maternity area or calving pen within 15min of birth or before standing attempts are initiated so as to minimize picking up infection from this highly likely contaminated area. Navel disinfection is recommended if navel infection is a problem in the herd (>5%).

3.2. Importance and feeding of colostrum

Calves are born with no immunity against disease and depend entirely on the passive immunity acquired by drinking colostrum from their dam, until they can develop their own ability to resist disease (Moran, 2002). Colostrum is essentially milk reinforced with blood proteins and vitamins. It has more than twice the level of total solids than in whole milk through boosted levels of protein and electrolytes.

The concentrations of protein, vitamins A, D and E in colostrum are initially about five times those of whole

milk, with a protein content of 17-18% compared to 2.5-3.5%. 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 blood stream (Moran, 2002). Beyond the immediate benefit to calf survival and preweaning health, colostrum also has long-term implications on growth rate and feed efficiency (Robison et al., 1988), reproductive efficiency (Faber et al., 2005), milk production (Faber et al., 2005), and longevity in the herd (Robison et al., 1988).

Current advice to farmers is to ensure calves drink enough colostrum from their dam within the first 3 to 6 hours of life. Newborn calves need to ingest at least 100 g of Ig within their first 3 to 6 hours of life, and ideally the same amount 12 hours later. Although it is not possible to estimate the amount of colostrum ingested by a newborn calf during natural suckling, as practiced in traditional systems in Ethiopia, it is imperative to encourage the calf to suckle as much colostrum as possible within the first few hours of life to prevent failure of passive transfer of immunity. Every half hour after birth that colostrum feeding is delayed, antibody transfer decreases by 5%. The cells in the intestinal wall mature in first 12 hours, eventually shutting down their absorption mechanism. Furthermore, after 24 hours, when the abomasum starts to secrete acids to make the milk-digestive enzymes more effective, these degrade the Ig proteins, which reduces their effectiveness. The first drink is also the most important as the ability of calves to absorb further Ig through the gut wall drops off markedly thereafter. The gut absorbs less Ig following first drink (Moran, 2002). IgG1 transfer from the colostrum to the newborn calf is considered adequate when calves less than 1 week of age reach a minimum serum IgG1 concentration of 1000mg/dL (Wells et al., 1996; Weaver et al., 2000).

Vaccinating pregnant cows can enhance the immune properties of colostrum. In the developed parts of the world there are vaccines to improve calf immunity against *E. coli*, clostridia, leptospirosis, salmonella, rotavirus, cryptosporidia, and also against several respiratory infections. Selection of the most appropriate vaccines should be based on the prevalence of particular calf diseases in the area. Such pregnant cow vaccines are not available in Ethiopia.

Calves can be fed with fresh, refrigerated or frozen maternal colostrum or with colostrum replacers (CR). Colostrum is believed to be preserved for 7 days under effective refrigeration, and for up to 1 year frozen. With clean colostrum that has at least 50 g/L IgG concentration (high quality colostrum), 3-4 L of colostrum will deliver the desired 150–200g mass of immunoglobulin to the newborn calf. When the colostrum immunoglobulin mass is not known, it is recommended that 10%-12% of body weight be fed at the first feeding within the first 4 h of life (Godden, 2008).

3.3. Health Care

Newborn Calf Care

Calves should be closely observed during and immediately after delivery. Calves are removed into a clean, dry, and deeply bedded calf pen or holding area before any standing attempts have been made. Examination of the navel involves attention for persistent bleeding, unusual size, appearance, or structures. Hygiene is the emphasis of navel care, but navel disinfectants, if judged necessary, are applied at this time (McGuirk, 2011).

Colostrum feeding is initiated as soon as the newborn calf demonstrates a suckle reflex but no later than 4h after birth.

Vaccinating Calves

The goals of any vaccination protocol established for calves from birth to weaning should be to provide optimal immunity to the agents or diseases that are most likely to be encountered. Calves need to be protected when they are at most risk for disease challenge (McGuirk, 2011).

The most common infectious problems of preweaned calves are diarrhea and respiratory disease. Important, but less common, are navel infections and neonatal septicemia that can result in infection of the joints, brain, kidney, or any other body organ system. The calves at greatest risk of developing diarrhea are less than 2 weeks of age, with peak onset typically occurring between 5 and 9 days of age The first episode of respiratory disease occurs in calves less than 3 weeks of age (Sivula et al., 1996; Virtala et al., 1996). The ingestion and absorption of immune factors in colostrum obtained from healthy, vaccinated cattle is the primary way to provide young calves with effective immunity to these important diseases.

The conventional view that calves cannot be vaccinated effectively while they have circulating maternal antibodies from colostrum has changed (Woolums, 2007; Chase et al., 2008). New approaches to vaccinating calves from birth to weaning have emerged. Intranasal modified live, temperature-sensitive parainfluenza type 3 (PI3) and infectious bovine rhinotracheitis (IBR) virus combination is utilized for the presumed specific and nonspecific protection against respiratory disease that can affect calves in the first few weeks of life. The intranasal route for vaccinating 1 week and older calves has the benefit of inducing rapid immunity that can circumvent potential interference from circulating maternal antibodies (Kimman et al., 1989; Woolums et al., 2004; Vangeel et al., 2009).

Screening Calves for Health Problems

The gold standard of mortality rate of dairy calves between 48 h of age and weaning is <5% (McGuirk, 2011). Scours and respiratory problems are responsible for the majority of preweaned calf deaths (USDA, 2009). Improved detection and earlier implementation of effective treatment protocols can lower mortality rates but requires that a regular health screening process is implemented. Daily screening to find sick calves or those that require further examination can be an efficient and effective process. Calves exhibiting abnormal

standing or sleeping posture, unresponsive to voices, have abdominal fullness, or have hair standing erect are candidates for further evaluation (McGuirk, 2011).

A calf health scoring chart (Fig. 1) can serve as a basis for the examination details needed to find calves to be treated for diarrhea or respiratory disease. Through quick inspection, abnormal fecal consistency or appearance can be noted and quiet calves can be screened for respiratory disease by looking for abnormal nasal discharge, ocular discharge, ear position, or spontaneous coughing. With moderate to severe ocular or nasal discharge, abnormal ear position or head tilt coupled with repeated spontaneous coughing, respiratory disease is likely. Calves with two or more abnormal signs should be considered for treatment. Then more specific additional examination parameters such as rectal temperature, navel palpation, and tracheal compression to induce coughing can be performed by the examiner (McGuirk, 2011).

Calf Respiratory Scoring Chart

Farm Name: _____

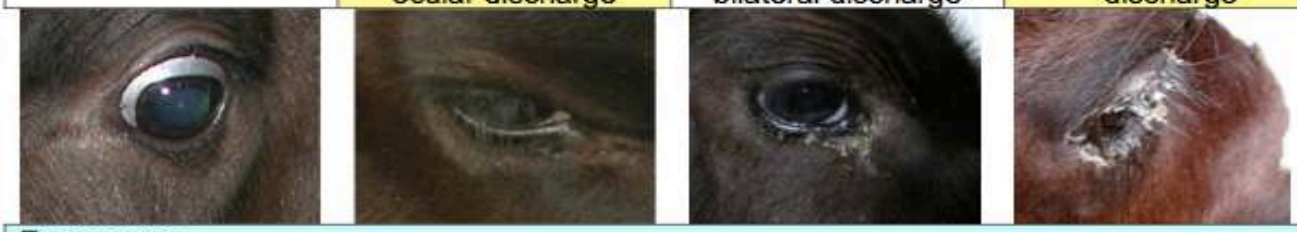
Date: _____

Calf Scores	(Total respiratory score: 4 – watch, 5 or more – treat.)					
Animal ID	Age	Nasal discharge	Eye or ear (highest number)	Cough – spontaneous or induced	Temperature	Total respiratory score

Calf Health Scoring Criteria

0	1	2	3
Rectal temperature			
37.8-38.3	38.3-38.8	38.9-39.4	≥39.4
Cough			
None	Induce single cough	Induced repeated coughs or occasional spontaneous cough	Repeated spontaneous coughs
Nasal discharge			
Normal serous discharge	Small amount of unilateral cloudy discharge	Bilateral, cloudy or excessive mucus discharge	Copious bilateral mucopurulent discharge

Eye scores			
Normal	Small amount of ocular discharge	Moderate amount of bilateral discharge	Heavy ocular discharge



Ear scores			
Normal	Ear flick or head shake	Slight unilateral droop	Head tilt or bilateral droop

Figure 1. Calf health scoring chart and calf health scoring criteria (http://www. vetmed.wisc.edu/dms/fapm/fapmtools/8calf/calf_health _scoring_chart.pdf)

3.4. Major health problems of calves

Diarrhea in the neonatal period and pneumonia in older calves are the major causes of calf morbidity and mortality all over the world (Svensson et al., 2006). These two account for more than 80% of all calf deaths, with diarrhea being the most common (Moran, 2002).

Calf scours or neonatal diarrhea

The causes of scours in calves under 30 days of age are difficult to determine. There is usually not one single cause, but are complex and usually involve an interaction between enteropathogenic bacteria, viruses, and protozoa, the colostral immunity of the animal and the effects of the environment.

The common known causes of diarrhea in newborn calves include enterotoxigenic *E. coli* (ETEC), verocytotoxic *E. coli* (VTEC), necrotoxigenic *E. coli* (NTEC), rotavirus, coronavirus, *Cryptosporidium* spp., *Eimeria* spp., and *Salmonella* spp. Rotavirus, *Cryptosporidium* spp., coronavirus, and enterotoxigenic *E. coli*, collectively are responsible for 75-95% of infections in neonatal calves worldwide (Kodituwakku and Harbour, 1990). Risk factors may also precipitate the disease in calves in which the disease might not normally occur, even though they are infected with a specific enteropathogen (Radostits et al., 2007).

Acute calf scour is characterized clinically by acute profuse watery diarrhea, progressive dehydration and acidosis and death in a few days.

Dietary scours

This mainly results from overfeeding. Affected calves get severe diarrhea but otherwise appear normal. However, they can more easily develop infectious scours. The best control measure for dietary scours is changing from milk to electrolytes for at least 24 hours.

White scours

This generally occurs in the first few days and is usually caused by pathogenic strains of *E. coli* that invade the gut wall. Foul smelling, grey to creamy-white severe diarrhea is seen. Calves quickly become dehydrated and lethargic, will not eat, are 'tucked up' in the abdomen and may die suddenly. In chronic cases that linger on, pneumonia or arthritis can occur. On post-mortem, a calf that died from *E. coli* scours will often show no visible signs of having an infection. Stress factors, such as cold or partial starvation can increase the occurrence and severity of white scours.

Viral and protozoal scours

These are generally caused by rotavirus or coronavirus (viral) or cryptosporidia (protozoal) and constitute most of the scours in calves less than 3 weeks old.

Salmonella scours

This occurs more commonly in older calves, causing bloody, putrid diarrhea containing mucus. Affected calves develop fever, are weak and rapidly become dehydrated and emaciated. They have a high death rate. Less severely affected calves can have rough coats, potbellies and become stunted; they can also become carriers of *Salmonella* and continually infect other animals. Extra personal hygiene is needed when treating *Salmonella*, as the bacteria can infect humans.

Coccidiosis or blood scours

This is caused by *Eimeria* spp. infecting the calf from 3 weeks of age and onwards and can easily be confused with white scours. Affected calves show bloodstained scouring with a lot of mucus and may eventually develop anemia. Coccidiosis is a stress-related disease and usually affects calves that are reared in wet, crowded, unhygienic conditions.

Treating scours

All affected calves should be identified, isolated and treated immediately with oral and parenteral fluid to correct the dehydration and acidosis. Antibacterials may be given orally and parenterally for the treatment of enteric and septicemic colibacillosis. When large numbers of calves are affected at one time all acutely affected calves should be treated (Radostits et al., 2007). Scours accounts for 75% of all deaths under 3 weeks of age (Radostits et al., 1994). The most important pathogens associated with infectious scours at different ages are:

- *E. coli*, 3-5 days
- Rotavirus, 7 - 1 0 days
- Coronavirus, 7 - 1 5 days
- Cryptosporidia, 15-35 days
- Salmonella, several weeks
- Coccidia, older than 3 weeks

Scouring calves can lose up to 20 times more fluid than healthy animals and they will become dehydrated because they are losing considerably more liquid than they can drink. This lost fluid also contains mineral salts and other nutrients. The degree of dehydration can be assessed using the skin fold (pinch) test. Pinch the skin and note how long it takes to return to normal. In healthy calves this is less than half a second. Another indicator is the degree of sunkeness of the eyes. Table 3 provides visual indicators of the degree of dehydration.

Table 3. Measures of dehydration in scouring calves (Moran, 2002)

% Dehydration	Sunken eyes*	Skin fold test (seconds)	Clinical symptoms
4-6	-	1-2	Mild depression, decreased urine output
6-8	+	2-4	Dry mouth and nose, tight skin, still standing
8-10	++	6-10	Cold ears, unable to stand
10-12	+++	20 45	Near death

* The more pluses (+), the more sunken the eyes

Very dehydrated calves (10-15%) will require IV therapy. Calves with less than 8% dehydration and still drinking can be rehydrated orally by electrolyte solutions. Oral fluid therapy is the term used for treating scours with soluble sources of energy and electrolytes by mouth. These supply an energy supplement and replace lost vital minerals and fluids in scouring calves.

The amount of fluid required for daily maintenance requirements and to replace lost fluids can be calculated, based on live weight and the degree of dehydration. For a 40 kg calf with 6% dehydration:

- Replacement: 40 kg x 6% or 2.4 L fluid
- Maintenance: 100 mL/kg/day, or 40 kg x 100 mL or 4.0 L fluid
- Total: 2.4 + 4.0 or 6.4 L fluid
- Feed this quantity in three feeds per day
- Check the degree of rehydration using the skin fold test

Up to 70% of calves will recover with adequate fluid therapy. Electrolyte treatments do not provide sufficient energy to maintain the animal. After 24 hours, reintroduce milk (if it has been withdrawn), but continue electrolytes for a further 48 hours. Separate milk feeding from electrolyte feeding by six hours.

The electrolyte solution can be offered to calves with bucket or teat, but if they do not drink it this way, it can be administered using a drench gun or a stomach tube. Fluids should be given IV to very sick and dehydrated calves.

If examination of the calf with diarrhea reveals a temperature greater than 39.4°C or lower than 37.7°C, if the calf is dull, off feed, drinks slowly, stands with an arched back, or has a significant amount of blood in the feces, a 3-day course of antibiotics is advisable (McGuirk, 2008). The selection of a therapeutic antibiotic is based on fecal culture results or the appropriate gram-negative bacterial spectrum. Nonsteroidal anti-

inflammatory drug administration may be of additional benefit but should be repeated only with persistence of an abnormal temperature (>39.4°C or <37.7°C) or other signs of systemic illness.

Attempt should be made to obtain an accurate diagnosis and give the most appropriate treatment. Sick calves should be isolated from healthy calves and taken care of after feeding other calves to minimize the spread of infection. Water should be made freely available.

Controlling scours through management

Nutritional scours is caused by stresses reducing the production of digestive acids in the abomasum. By removing the initial stress, sufficient abomasal acids are produced, and normal milk digestion will eventually resume.

Sudden changes in milk feeding routines, environmental stress such as sudden changes in weather (a sudden cold and wet spell); cold, damp and draughty or humid conditions inside calf sheds; overcrowding and changes in standards of hygiene can introduce sufficient stress to increase the incidence of nutritional scours in calves.

Calves are particularly susceptible to scour during their second week of life. By careful observation, experienced rearers can anticipate the onset of scours the day before it happens, after which milk feeding can be reduced, with the calf recovering quickly.

The following signs of impending scours should be looked for:

- Dry muzzle
- Thick mucus appearing from the nostrils
- Very firm feces
- Refusal to drink milk
- A tendency to lie down
- A high body temperature (over 39.3°C)

Early identification and treatment of sick calves is the key to their rapid return to health. Most scouring calves that are treated are back to normal after only two days on fluid replacer treatment and then they can be gradually reintroduced to milk over the next three days.

Preventing scours

To ensure healthy and disease-resistant calves, the importance of good colostrum feeding management cannot be overemphasized. Up to 40% of calves do not absorb sufficient antibodies into their bloodstream within the first 12-24 hours of life because of inadequate attention given to their colostrum feeding. Such calves are more likely to succumb to infectious scours.

Prevention of scours centers around good hygiene and minimizing stress. Measures that can be taken include:

- Consider vaccinating cows with calf diarrhea vaccine (*E. coli*, *Salmonella*) 3-6 weeks prior to calving
- Ensure calves are protected from extremes of climate
- Minimize stresses associated with routine management practices
- Maintain strict hygiene by cleaning and sterilizing feeding utensils and facilities
- Quickly respond to early symptoms of scours, isolate sick calves and address the cause
- Keep records of treatment of sick calves to assist in veterinary diagnoses and for withholding periods if the calf is subsequently culled

Pneumonia

Calf pneumonia is a respiratory disease caused by inflammation of the lung tissue, primarily the alveoli. The cause is multifactorial, associated with various species of viruses, bacteria and mycoplasma. Pathogens like mycoplasma and viruses may act as primary pathogens, and certain bacterial species may cause secondary complications (Radostits et al., 2007). It is usually associated with one or more of the following pathogens: bovine respiratory syncytial virus (BRSV); parainfluenza 3 (PI-3) virus; bovine viral diarrhea virus (BVDV); bovine herpes virus-1 (BHV-1); *Mannheimia haemolytica*; *Pasteurella multocida*; *Histophilus somnus*; *Trueperella pyogenes*; and *Mycoplasma bovis* (Barrett, 2000).

Aerosol infection and direct contact are the methods of transmission and both are accentuated in crowded, inadequately ventilated conditions (Radostits et al., 2007). Pneumonia is a problem with housed calves, particularly when stocking density is high and ventilation is poor. In the US, it accounts for 15% of the calf deaths from birth to 6 months of age. The shed temperature and relative humidity are the two most important factors influencing its occurrence. Pneumonia is more common in crowded, cool, damp sheds, although it can also be a problem in hot, dry shed conditions.

Typical signs of pneumonia include lethargy, discharge from the nose and eyes, rapid breathing, and a rise in body temperature and pulse rate. A harsh, hacking cough, easily stimulated by pinching the trachea, is characteristic. Coughing is especially noticeable after exertion (Moran, 2002; Radostits et al., 2007).

The control of pneumonia is mainly through improved housing. Poor ventilation leads to condensation, which results in humid conditions and an increase in the survival and spread of infection through water droplets in the air. Draughts of cold air at animal height in pens will aggravate the condition. High dust and ammonia levels (from urine in poorly drained and ventilated pens) can cause irritations in the lungs making these calves more prone to pneumonia. There is good evidence that the colostral immunological status of the calf has a significant effect on the susceptibility of the calf to pneumonia. There is a clear association between low levels of Ig of calves at 2-3 weeks of age, and subsequent susceptibility to pneumonia at 2-3 months of age (Corbeil et al., 1984; Radostits et al., 2007).

Early recognition and treatment of affected calves, with antibiotics, will minimize losses through deaths and poor calf growth. Sheds should be spacious and adequately ventilated but draught-free. The use of solid walls to at least 2 m high is ideal in cold and windy environments.

Pulpy kidney and other clostridial diseases

Pulpy kidney can occur when calves are first introduced to high concentrate diets. It is caused by one of the clostridia bacteria, which produces a toxin in the gut, eventually killing the calf (hence the name enterotoxaemia). As with all clostridial diseases, the bacteria are a normal part of the environment and are impossible to eradicate. The classical sign of pulpy kidney is that the fattest calves die suddenly and their carcasses rot very quickly. Routine vaccination programs of 'five-in one' vaccines (blackleg, black disease, malignant edema, tetanus and pulpy kidney) can prevent the disease. A combined 'seven-in-one' vaccine provides protection against both clostridia and leptospirosis diseases.

Navel-ill and joint-ill

This is caused by bacteria infecting the umbilical cord soon after birth, particularly where the calving area is heavily contaminated. Unless treated promptly in young calves, it can lead to severe inflammation or arthritis of the joints. Animals with joint-ill are reluctant to walk and stand for only brief periods. As the infection is carried in the blood stream to all parts of the body, reduced appetite, diarrhea and pneumonia may also occur. If navel infection is a herd problem, swabbing, spraying or dipping navels of newborn calves with 1%, 2% or 7% iodine or 0.5% chlorhexidine as a precautionary measure, and keeping calving facilities clean should help to prevent navel-ill (Mee, 2008; McGuirk, 2011).

4. Diseases that Affect the Reproductive Performance of Dairy Cattle

(Adapted from Risco and Melendez, 2011)

Learning outcome

- A sound knowledge of peripartum diseases that could affect reproductive performance of cows
- Able to diagnose, manage and prevent/control these conditions

Reproductive performance in lactating dairy cows should be viewed as a process that involves estrus detection, breeding, conception, and pregnancy maintenance. The outcome of this process in a dairy farm is to increase the number of pregnant animals in a timely manner at the end of the voluntary waiting period. Diseases that occur throughout lactation can have a major impact on this outcome by affecting cyclicity, conception, and embryo survival (Risco and Retamal, 2011).

4.1. Hypocalcemia-Related Diseases

The total blood calcium concentration in cows is about 10 mg/dL (~5.0 mg/dL ionized calcium). During calving or shortly thereafter, hypocalcemia is inevitable in the dairy cow and is characterized by a plasma calcium concentration <7.5 mg/dL. Milk fever or parturient paresis is the clinical manifestation of hypocalcemia. Clinical signs associated with milk fever occur as progressive muscular weaknesses from changes in neuromuscular tone and range from tremors during the early stages to flaccid paralysis and eventually coma (Van Saun, 2007). Hypocalcemia has been associated with dystocia, retained fetal membranes, uterine infection, and mastitis, diseases that lower fertility in cattle.

Hypocalcemia affects the normal function of organs that contain smooth muscle, such as the abomasum and rumen, without causing the animal to become paretic. This condition is referred to as subclinical hypocalcemia and has been associated with various periparturient disorders such as uterine prolapse, retained fetal membranes, and displaced abomasum. The practice of feeding anionic diets to prepartum dairy cows, in the developed world, has lowered the incidence of clinical hypocalcemia in dairy farms to a point where milk fever is not considered a major health problem. In contrast, subclinical hypocalcemia continues to be a common problem affecting postpartum cow health. Goff and Horst (1998) reported that 10%-50% of cows remained subclinically hypocalcemic during the first 10 days postpartum.

Treatment and prevention of Hypocalcemia

In cases of hypocalcemia, calcium therapy is directed at replacing and maintaining normal plasma calcium concentration in clinical or subclinical cases of hypocalcemia. In cases of milk fever, immediate slow IV infusion of 8-9g calcium is warranted to prevent death. Additionally, in those cows at risk to develop subclinical hypocalcemia (retained fetal membranes, inappetance), calcium therapy would help restore blood calcium concentration and promote normal function of calcium-dependent organs (Risco and Retamal, 2011).

Approximately 85 per cent of cows with milk fever will respond to one treatment. If response is not evident by 5–6 hours, then the cow should be re-examined, the diagnosis reassessed and if necessary a further IV infusion of 8–12g of calcium administered. Relapse of milk fever occurs in 25 per cent of cases treated.

A number of preparations are available for the treatment of milk fever and most are based on calcium borogluconate (CBG) at 20 %, 30 % or 40 % strength. It has been shown that 400ml of 30 % CBG is adequate to treat milk fever in average size cattle and will provide 9g of calcium. However, a preparation of 400 ml of 40% CBG, which will provide 12g of calcium, is in common use (Eddy, 2004).

Treatment of milk fever should also be accompanied by removal of the calf and advice to the farmer not to milk the cow for 24 hours except to check for the presence of mastitis. Cases of relapse usually occur at 18–

24-hour intervals and should be treated in the same way, i.e. by the IV infusion of 8–12g of calcium. Occasionally cows, particularly Jerseys, have been known to relapse on up to seven occasions (Eddy, 2004).

Measures which may prevent hypocalcemia include (Eddy, 2004):

- Administration of supplemental magnesium to dry cows within three weeks of calving if the magnesium status of the herd was found to be low on blood testing
    - Low calcium level interferes with calcium absorption
- Restricting calcium intake
    - To be certain dry-cow diet should contain less than 30 g/day of calcium.
    - A diet containing more calcium should be administered just before parturition to ensure adequate calcium being available over the risk period.
- Feeding acidic diets
    - Should only be used where there is adequate calcium provision
- Vitamin D_3 and its metabolites given by injection
- Maintaining adequate calcium intake by drenching cows daily with 150 g/day of calcium chloride on the day before calving and for four days thereafter

4.2. Diseases Related to Negative Energy Balance

Prior to parturition, a depression in feed intake occurs in dairy cows, and after calving they mobilize fat as well as protein reserves. Consequently, many dairy cows are in a negative energy balance and may develop subclinical ketosis during early postpartum; uterine health can be compromised, predisposing cows to uterine infections (Risco and Retamal, 2011).

Energy balance near calving is associated with uterine health disorders and fever in Holstein cows. Cows with fever (days 1–10 postpartum) and endometritis (cytology at 4 weeks postpartum) experienced lower dry matter intake from −1 week to +5 weeks of calving and were ketotic from −2 to +4 weeks from calving. Cows that were ketotic during postpartum had suppressed neutrophil function, and uterine infections were preceded by negative energy balance prior to calving which was extended into early lactation.

The severity and duration of negative energy balance postpartum is a major factor that influences ovarian activity and resumption of postpartum cyclicity in dairy cows. Dairy cows in progressive negative energy state are less likely to ovulate during the first 65 days postpartum, and get pregnant with the first insemination postpartum. Cows expressing one or more estruses during the first 30 days postpartum had improved pregnancy rates to first service compared with anovular cows.

Body Condition Scores (BCSs)

Body condition scores (BCSs) postpartum are related to the magnitude and severity of negative energy balance and are used to assess body reserves, particularly fat. A scale of 1-5 with increments of 0.25 unit is recommended for dairy cattle. Cows that experience BCS loss early in lactation are in a greater negative energy balance and are more likely to be less fertile- lower conception to first service and pregnancy rate. Conception to first service and pregnancy rates are lower in cows with lower BCS.

Herd Diagnostics for Ketosis Risk

Errors in feeding transition cows can occur, predisposing to subclinical ketosis after parturition. Therefore, from a production medicine perspective, evaluation of herd risk for ketosis should be routinely conducted on dairy farms to determine whether or not parturient cows are transitioning well into lactation. That is, if the risk for ketosis is present, evaluation of feeding and management practices of the transition period is indicated to implement corrective practices. The premise for determination of herd risk for ketosis is based on selective metabolic profiling and dairy production records evaluation (Table 4).

Table 4. Metabolic profiling and dairy production records to evaluate herd risk for ketosis (Van Saun, 2007)

Profile or record	When	Number of animals	Risk for ketosis
NEFA	Prepartum (–14 to –3 days) Postpartum (<30 days)	More than two out of 12 cows sampled have 0.4 mEq/L prepartum or 0.7 mEq/L postpartum	Yes
BHB	Early postpartum	More than two out of 12 cows sampled of 10 or 12 cows have >14.5 mg/dL (1400 μmol/L)	Yes
Milk fat composition	First test day	20% or greater of cows have elevated milk fat (Holsteins, 5%; Jerseys, 6%)	Yes
Fat-to-protein ratio in milk	First test day	40% of cows with 1.33	Yes

NEFA=nonesterified free fatty acids; BHB=beta hydroxy butyrate.

Treatment of Cows with Ketosis (Clinical or Subclinical)

The major objective in ketosis therapy is to reestablish normal feed intake. This requires resolution of hypoglycemia and hyperketonemia. It is vital to identify and to treat concurrent conditions with ketosis (uterine infection, displaced abomasum, mastitis) and to address predisposing factors at the herd level (Risco and Retamal, 2011).

For successful treatment in most cases the following regimen is to be recommended (Eddy, 2004):

- 500ml of 40 % glucose/dextrose IV, followed by
- One dose of glucocorticoid, followed by
- Oral treatment twice daily with 150g of propylene glycol containing cobalt for three to four days.

4.3. Uterine Infections

Economic costs due to uterine disease are related to infertility, increased culling for failure to conceive, reduced milk production, and the cost of treatment. The cost of a single case of metritis has been calculated to be about €292.

Uterine infections within a week of parturition are present in up to 40% of dairy cows; herd rates for clinical signs between 36% and 50% have been reported, and up to 21% of animals have signs of systemic illness such as fever. Uterine infections decrease milk yield and fertility.

Metritis

Puerperal metritis is defined by an abnormally enlarged uterus and a fetid, watery, red-brown uterine discharge associated with signs of systemic illness (decreased milk yield, dullness, or other signs of toxemia) and temperature >39.5^{o}C within 21 d after parturition (Sheldon et al., 2006). It generally is associated with dystocia, retained fetal membranes, and calving trauma.

Clinical Endometritis

This condition is characterized by the presence of purulent (>50% pus) or mucopurulent (approximately 50% pus, 50% mucus) uterine exudates in the vagina, 21 days or more postpartum, without abnormally enlarged uterine horns (Sheldon et al., 2009).

Subclinical Endometritis

This condition has been described as inflammation of the endometrium in the absence of purulent material in the vagina (Sheldon et al., 2009). Subclinical disease is defined by polymorphonuclear (PMN) cells >5.5% and 10% of cells in uterine cytology samples collected by flushing the uterine lumen or by endometrial cytobrush, in the absence of clinical endometritis 20-30 days postpartum (Kasimanickam et al., 2005).

Treatment and Management of Uterine Infections

Therapy for uterine infection includes intrauterine therapy of antibiotics, systemic antibiotics, supportive therapy, and hormone therapy (Hussain and Daniel, 1991).

A variety of broad-spectrum antibiotics have been recommended for parenteral administration to cows with metritis. Penicillin or one of its synthetic analogs is most commonly recommended (20,000–30,000 U/kg bid). Ceftiofur, a third generation cephalosporin that has broad-spectrum activity against gram-positive and gram-negative bacteria, has been reported to reach all layers of the uterus and lochial fluid without having violative residues in milk (Schmitt and Bergwerff, 2000; Chenault et al., 2004). Oxytetracycline has been recommended for intrauterine therapy for postpartum cows affected with metritis or clinical endometritis caused by *Arcanobacterium pyogenes*. Intrauterine administration of antibiotics, however, can result in contamination of milk, and appropriate withdrawal times have not been determined (Bishop et al., 1984).

Nonsteroidal anti-inflammatory drugs such as flunixin meglumine (1.1-2.2 mg/kg of body weight) can be used to treat fever. Furthermore, cows with metritis may experience depressed appetite affecting calcium and energy status. Consequently, therapy with calcium and energy supplements may be warranted (Risco and Retamal, 2011).

A variety of hormones have been administered to cows in attempts to prevent or treat postpartum metritis. Administration of prostaglandin during the postpartum period may enhance the reproductive performance of dairy cows regardless of their periparturient diseases status (Young et al., 1986; Risco et al., 1994).

Retained fetal membranes

A major risk for uterine infection in dairy cows is retained fetal membranes (Grohn et al., 1990) and a diagnosis is generally made when membranes are retained from 12 to 24 h after parturition. The option of no treatment at the time of retained fetal membranes diagnosis has become a common practice on many dairies. However, cows that retain fetal membranes can develop systemic signs related to uterine infections after initially being left untreated. Consequently, producers should monitor cows with retained fetal membranes for systemic disease and provide prompt treatment. Conversely, initiating antimicrobial treatment on cows diagnosed with the disease even without signs of systemic illness is advocated by some veterinarians to prevent uterine infections and related diseases. Systemic administration of ceftiofur in dairy cows affected with dystocia, retained fetal membranes, or both reduced the incidence of metritis by 70% compared with retained cows not treated with antibiotics or those treated with estradiol cypionate (Risco and Hernandez, 2003).

4.4. Mastitis

Mastitis is an economically important disease of dairy cattle due to losses in milk production and replacement and treatment costs. In addition, clinical mastitis can affect reproductive performance by increasing the number of days to first service, days nonpregnant, and services per conception (Barker et al., 1998), together with a higher risk of abortion. Mastitis is one of the major constraints of dairy production in Ethiopia with prevalence ranging between 23.2 and 81.1% (Abebe et al., 2016).

Gram-positive bacteria causing clinical mastitis may be associated with embryonic losses in dairy cows by stimulating the release of inflammatory mediators and pyrexia (Barker et al., 1998). Effects of these inflammatory mediators include alteration of luteinizing hormone (LH) release and follicle-stimulating (FSH) activity, which can alter oocyte development, estrous cycle, and embryonic function (Hansen et al., 2004). In contrast, endotoxin (lipopolysaccharide [LPS]) from gram-negative bacteria increased serum PGF2α levels (Giri et al., 1990; Jackson et al., 1990) and, through its luteolytic action, altered the estrous cycle or caused abortion in cows (Gilbert et al., 1990).

Subclinical mastitis resulted in lower reproductive performance of lactating cows, comparable to that of cows with clinical mastitis (Schrick et al., 2001). Furthermore, a negative effect of a linear somatic cell count (LNSCC) ≥4.5 on embryo survival was reported (Moore et al., 2005). Time to first breeding, time to conception, conception at first service, and number of services to conception are all negatively affected by high LNSCC. Cows registering a high LNSCC during the first 90 days of gestation were reported to have an increased risk of abortion (Pinedo et al., 2009).`

Because either clinical or subclinical mastitis has a significant effect on reproductive performance in dairy cattle, veterinarians engaged in reproductive management of dairy farms should ensure that mastitis prevention practices are well established if reproductive performance is expected to be optimized (Risco and Retamal, 2011).

4.5. Lameness and Reproductive Performance

Due to its painful condition, lameness is an animal welfare concern and reduces reproductive performance in dairy cattle. Therefore, as part of reproductive management programs, dairy farms should strive to implement lameness prevention strategies including prophylactic hoof trimming (Risco and Retamal, 2011).

Lameness delays resumption of cyclicity after calving and prolongs the calving to conception interval in dairy cows. Lameness also changes normal cow behavior and compromises gait; the unwillingness to bear weight in one or more limbs inhibits estrual behavior, reducing detection of estrus in affected cows.

Prophylactic hoof trimming was reported to be beneficial by reducing the incidence of severe lameness. Healthy cows examined for foot lesions and hoof-trimmed around 200 days postpartum (prophylactic hoof trimming) tended to be less likely to be diagnosed lame thereafter than those that were not examined and not hoof-trimmed. Cows not receiving prophylactic hoof trimming tended to be 1.25 times as likely to become lame compared with cows receiving prophylactic hoof trimming.

5. Infectious Reproductive Diseases

(Adapted from Risco and Melendez, 2011)

Learning outcome

- A good knowledge of infectious reproductive diseases in cows and recognize the associated reproductive signs

During gestation, the bovine reproductive system, with its multilayered placenta (epitheliochorial), leaves the fetus in a naive environment susceptible to infection. Abortions may occur due to infection of the placenta, inflammation of the ovary, death of the fetus, and/ or disruption of the cervical plug. Infectious reproductive

diseases are the hardest to protect against. Vaccination must minimize the amount/duration of the viremia/septicemia or prevent disease from moving through the cervix. Due to the numerous causes of reproductive failures (of which infectious agents are a small percentage), vaccination to prevent infectious reproductive losses may not appear to be effective. Table 5 shows some infectious reproductive diseases with respective reproductive syndromes in cattle.

Table 5. Infectious reproductive diseases of cattle

Disease	Causative agent	Reproductive syndrome	Control/Prevention
Bovine viral diarrhea (BVD)	Bovine viral diarrhea virus (BVDV)	Naïve cow exposure in the first trimester • Early embryonic death • Abortion • Mummification • Persistently infected (PI) calf Exposure in the second trimester • Birth defects (primarily involving nervous tissue) • Occasionally PI calf Exposure in the last trimester • Usually no effect on the fetus • Calf will be born with antibodies against BVDV	• Vaccination of heifers at least 3 weeks before breeding. • Identification and elimination of PI animals. • Biosecurity
Infectious bovine rhinotracheitis (IBR), Infectious pustular vulvovaginitis (IPV)	Bovine herpes virus type 1 (BHV-1)	• Abortion (up to 25%-50% of cows abort) – majority in the last trimester of pregnancy • Lower pregnancy rate • Conception failure • Pustular and nectrotic lesions on the vulva and vaginal tract	• Vaccination • Biosecurity
Leptospirosis	Different serovars of *Leptospira interrogans, L. borgpetersenii, L. kirschneri*	• Abortion storms • Reproductive failure • Stillbirths • Birth of premature and weak calves • Conception failure • Early embryonic death • Decrease in fertility	• Vaccination (every six months)
Brucellosis	*Brucella abortus*	• Abortion (usually after 5 month of gestation) • Retained placenta and subsequent metritis • Decreased conception rate • Increased services per conception • Increased number of dead and weak calves	• Vaccination • Test and culling of all positive animals
Bovine Trichomoniasis	*Tritrichomonas fetus*	• Abortion (early in gestation) • Pyometra • Infertility (the most common sign) • Early embryonic death • Long interservice interval • Sterility (rarely)	• Vaccination (with questionable efficiency)
Bovine genital campylobacteriosis	*Campylobacter fetus* subspecies venerialis	• Early embryonic death • Prolonged estrous cycle • Early abortion (rarely) • Sterility (in some animals) • The signs are much higher in heifers	• Vaccination (improved breeding efficiency in vaccinated herds)
Neosporosis	*Neospora caninum*	• Abortion (usually second trimester) • Birth defects • Early embryonic loss	• Prevent canine from defecating on feed, pasture • Restrict dogs from eating aborted material, or dead calves.
Salmonellosis	Many different serotypes of *Salmonella*	• Abortion • Stillbirth • Early embryonic loss • Retained fetal membranes following late-term abortion • Neonatal septicemia	• Farm hygiene
Histophilosis	*Histophilus somni*	• Can be isolated from the reproductive tract of females following early abortions • Believed to be a normal inhabitant of the vaginal tract of cattle	• Vaccines may help immunize cattle against infection

6. Other Infectious Diseases

(Adapted from Radostits et al., 2007)

Learning outcomes

- Identify, manage and control/prevent major infectious diseases of cattle.

6.1. Anthrax

Etiology

Anthrax is a zoonotic disease caused by the sporeforming bacterium *Bacillus anthracis*. Anthrax is most common in wild and domestic herbivores (eg, cattle, sheep, goats, camels, antelopes) but can also be seen in humans.

B. anthracis spores can remain infective in soil for many years. Grazing animals may become infected when they ingest sufficient quantities of these spores from the soil. Feed contaminated with bone or other meal from infected animals can serve as a source of infection for livestock, as can hay that is heavily contaminated with infected soil.

Epidemiology

Anthrax has been reported from nearly every continent and is most common in agricultural regions with neutral or alkaline, calcareous soils. Epizootics are usually associated with drought, flooding, or soil disturbance, and many years may pass between outbreaks. During interepidemic periods, sporadic cases may help maintain soil contamination.

Clinical Findings

Depending on the route of infection, host factors, and potentially strain-specific factors, anthrax can have several different clinical presentations. In herbivores, anthrax commonly presents as an acute septicemia with a high fatality rate, often accompanied by hemorrhagic lymphadenitis.

Typically, the incubation period is 3–7 days (range 1−14 days). The clinical course ranges from peracute to chronic. The peracute form (common in cattle and sheep) is characterized by sudden onset and a rapidly fatal course. Staggering, dyspnea, trembling, collapse, a few convulsive movements, and death may occur with only a brief evidence of illness.

In acute anthrax there is an abrupt fever and a period of excitement followed by depression, stupor, respiratory or cardiac distress, staggering, convulsions, and death. Often, the course of disease is so rapid that illness is not observed and animals are found dead. The body temperature may reach 41.5°C, rumination ceases, milk production is materially reduced, and pregnant animals may abort. There may be bloody

discharges from the natural body openings (Fig. 2). Some infections are characterized by localized, subcutaneous, edematous swelling that can be quite extensive. Areas most frequently involved are the ventral neck, thorax, and shoulders.

Diagnosis

A diagnosis based on clinical signs alone is difficult. Confirmatory laboratory examination should be attempted if anthrax is suspected. Because the vegetative cell is not robust and will not survive 3 days in transit, the optimal sample is a cotton swab dipped in the blood and allowed to dry. This results in sporulation and the death of other bacteria and contaminants.

Specific diagnostic tests include bacterial culture, PCR tests, and fluorescent antibody stains to demonstrate the agent in blood films or tissues. Western blot and ELISA tests for antibody detection are available in some reference laboratories. Lacking other tests, fixed blood smears stained with Loeffler's or MacFadean stains can be used and the capsule visualized; however, this can result in some 20% false positives.

Treatment

Severely ill animals are unlikely to recover but in the early stages, particularly when fever is detected before other signs are evident, recovery can be anticipated if the correct treatment is provided. Penicillin, streptomycin or oxytetracycline are effective in the treatment of clinical cases. Other antibacterials including amoxicillin, chloramphenicol, ciprofloxacin, doxycycline, erythromycin, gentamicin and sulfonamides also can be used. It is desirable to prolong treatment to at least 5 days to avoid a recrudescence of the disease. Antiserum, if available, should also be administered for at least 5 days daily.

Control

Anthrax is controlled through vaccination programs, rapid detection and reporting, quarantine, treatment of asymptomatic animals (post-exposure prophylaxis), and burning or burial of suspect and confirmed cases. Annual vaccination of all grazing animals should be attempted in endemic areas. Vaccination should be done 2-4 wk before the season when outbreaks may be expected.

When an outbreak occurs, place the farm in quarantine, destruct discharges and cadavers, and vaccinate survivors. Prohibition of movement of milk and meat from the farm during the quarantine period should prevent entry of the infection into the human food chain.

Infected carcasses should not be opened but immediately burned in situ or buried, together with bedding and soil contaminated by discharges. If this cannot be done immediately, a liberal application of 5% formaldehyde on the carcass and its immediate surroundings will discourage scavengers.

Livestock at risk should be immediately treated with a single dose of long-acting antibiotic (oxytetracycline

or penicillin). This is followed by vaccination ~7–10 days after antibiotic treatment. Any animals becoming sick after initial treatment and/or vaccination should be retreated immediately and revaccinated a month later. Simultaneous use of antibiotics and live vaccine is inappropriate. Animals should be moved to another pasture away from where the bodies of dead animals had stayed. Suspected contaminated feed should be immediately removed.

Figure 2. A cow died of anthrax (blood oozing from the rectum)

6.2. Blackleg

Etiology

True blackleg, the clostridial myositis of skeletal muscles, is associated with *Clostridium chauvoei*.

Blackleg is a soil-borne infection but the portal by which the organism enters the body is still in dispute. It is presumed that the portal of entry is through the alimentary mucosa after ingestion of contaminated feed or associated with erupting teeth. The bacteria may be found in the spleen, liver, and alimentary tract of normal animals, and contamination of the soil and pasture may occur from infected feces or decomposition of carcasses of animals dying of the disease. True blackleg develops when spores that are lodged in normal tissues are caused to proliferate by mechanisms such as trauma or anoxia. In cattle the disease usually occurs without a history of trauma.

Epidemiology

When the disease occurs it is usual for a number of animals to be affected within the space of a few days. The disease is enzootic in particular areas, especially when they are subject to flooding. The case fatality rate in blackleg approaches 100%.

Typical blackleg of cattle has a seasonal incidence, with most cases occurring in the warm months of the year. In some areas there is an increased prevalence in years of high rainfall. Outbreaks of blackleg in cattle have occurred following excavation of soil, which suggests that disturbance of the soil may expose and activate latent spores.

In cattle the disease is largely confined to young stock between the ages of 6 months and 2 years, although disease occurs occasionally in younger animals and cattle up to 3 years. In the field, risk factors include rapidly growing cattle and a high plane of nutrition.

Clinical findings

Affected animals commonly show severe lameness, usually with pronounced swelling of the upper part of the affected leg, severe depression, complete anorexia, ruminal stasis, a high temperature (41°C) and pulse rate (100-120/min). Pyrexia is not present in all cases. In the early stages the swelling is hot and painful to the touch but soon becomes cold and painless, and edema and emphysema can be felt. The skin is discolored and soon becomes dry and cracked.

Although the lesions are usually confined to the upper part of one limb, occasional cases are seen where the lesions are present in other locations such as the base of the tongue, the heart muscle, the diaphragm and psoas muscles, the brisket, and the udder. Lesions are sometimes present in more than one of these locations in one animal. The condition develops rapidly and the animal dies quietly 12-36 hours after the appearance of signs. Many animals die without signs having been observed. Sheep and cattle with cardiac myositis associated with *C. chauvoei* are usually found dead.

Diagnosis

In typical cases of blackleg in cattle a definite diagnosis can be made on the clinical signs and the necropsy findings. Definitive identification of *C. chauvoei* is by fluorescent antibody staining. Isolation of the organism from specimens collected from the lesion may also be attempted.

Treatment

Treatment of affected animals with penicillin and surgical debridement of the lesion, including fasciotomy, is indicated if the animal is not moribund. Recovery rates are low because of the extensive nature of the lesions. Large doses (40 000 IU/kg BW) should be administered, commencing with crystalline penicillin IV and followed by longer-acting preparations.

Control

On farms where the disease is enzootic, annual vaccination of all cattle between 3 and 6 months with two vaccinations given 4 weeks apart followed by an annual booster vaccination is recommended. This should be done just prior to the anticipated danger period. Maternal immunity persists for at least 3 months and will interfere with active immunity in calves vaccinated before this age.

In an outbreak all unaffected cattle should be vaccinated immediately and injected with penicillin at a dose of 10,000 IU/kg BW IM or a combination of penicillin and benzathine penicillin. Movement of the cattle from the affected pasture is advisable. It is important that carcasses of animals dying of blackleg are destroyed by burning or deep burial to limit soil contamination.

6.3. Contagious Bovine Pleuropneumonia (CBPP)

Etiology

CBPP is caused by *Mycoplasma mycoides* ssp. *mycoides* small colony type.

Epidemiology

The disease has occurred throughout the world at some time or another with the exception of South America and Madagascar. It is eradicated from Australia, North America and from Europe. It reached Africa during the colonialization period.

Under natural conditions cattle, buffaloes and antelopes are susceptible. Natural transmission occurs between animals in close contact through droplet infection either from cattle with the clinical disease or from subclinical carriers ("lungers") The inhalation of infected aerosols which are discharged from infected animals during coughing or exhalation may occur over a distance of up to 20 m and more. Transmission is facilitated by crowding of the animals.. Since the agent remains virulent for years in the sequestered lung tissue of latently infected animals, the disease can be carried through nomadic pastoralism over wide distances and also spread through cattle markets.

Clinical features

The incubation period is uncertain and can last from a few days up to several months. Depending on the resistance level of the animal, and the intensity of exposure, the disease takes an acute, subacute to chronic course, or the acute course is followed by a chronic stage which may last for years (lunger) as a latent phase of the disease. The majority of cases are subclinical. The clinical signs start with the characteristic short, dry cough which becomes more and more painful. With a fever (40^{o}C), the respiration becomes more frequent, feed intake and milk production are reduced, the animals are listless with a staring, lusterless coat and they become emaciated. As the disease progresses, the stage of hepatization of the lung sets in with lobular extension of the disease also reaching the pleura. The febrile reaction increases to 42^{o}C, tachycardia and

dyspnea become evident, the coughing increases, both in frequency and intensity, the animal is reluctant to move and stands with its head extended, mouth open, tongue protruding, and the carpal joints turned out. Contraction of the muscle of the abdominal wall occurs after each inspiration, while expiration is frequently followed by a characteristic grunt or groan. The animals breathe with flared nostrils. There is a bad smelling mucoid discharge from the nostrils, and frothy saliva accumulates around the mouth which becomes mucopurulent in the later stages of disease when subcutaneous edema of the lower parts of the chest and abdomen, as well as emaciation also become evident. The locality of the affected areas in the lungs is revealed by the presence of dull sounds during percussion. Death usually occurs after 2-4 weeks with profuse diarrhea. In some tropical breeds the disease takes a comparatively mild course, and after a convalescence of several weeks, the animals evidently seem to be healthy. These animals mostly harbor capsulated sequesters which excrete the pathogens intermittently.

In subacute cases, the lung lesions are more localized and an infrequent cough may be the only sign. In chronic cases, the only usual clinical signs are emaciation and a cough which usually occurs when the animal stands up. In calves up to the age of 6 months only arthritis and no pulmonary lesions will develop.

Whether a sterile state of immunity is reached after recovery is unclear. It is a fact, however, that animals which have recovered from the disease obtain a challengeable immunity for at least 2 years.

Diagnosis

Diagnosis based on the epidemiology, the clinical picture and necropsy findings is rather easy. The causal organisms can be isolated during the febrile phase or shortly afterwards from blood, and postmortem from pleural exudate and/or affected lung tissue.

To detect latently or chronically infected animals, almost all serological tests are suitable. Antibodies can be detected by CFT, the passive hemagglutination (PHA) and the slide agglutination (SAT) tests.

Treatment

Under practical field conditions, treatment is not applicable and not indicated because treated animals are highly likely to be chronic carriers. High value breeding animals if quarantined appropriately could perhaps be treated with tylosin (705 mg/kg b.w. during 5 days); tetracyclines and chloramphenicol are also effective.

Control

Eradicating infected herds by slaughter as it has been carried out in the industrialized countries.

CBPP can only be prevented efficiently by regular yearly vaccination of all susceptible animals. It is believed that if 100% of a cattle population older than 6 months are vaccinated continuously for 3-5 years the disease will disappear. Only live vaccines provide an efficient immunogenic effect.

6.4. Pasteurelloses

The classification of pasteurellosis is done according to the signs exhibited by the affected animal species as follows:

- Hemorrhagic septicemia (HS), the primary infection of cattle and buffaloes with *P. multocida*
- Pneumonic pasteurellosis in cattle (pasteurellosis in cattle, shipping fever), which is usually a pneumonia which is caused by *P. multocida* and *M. haemolytica*

Depending on their adaptation to the host, pasteurellae cause primary or secondary (opportunistic) infections.

Etiology

Pasteurella spp. are part of the healthy flora of the mucosae especially of the upper air passages of the respiratory tract in mammals and birds. Causal organisms of pasteurelloses of domestic animals are *Pasteurella multocida*, *Bibersteinia trehalose*, and *Mannheimia haemolytica* (with their different serotypes).

B. trehalosi has been isolated from cases of bovine respiratory disease and *M. haemolytica* from the lungs of cattle with pneumonia. Distinct *P. multocida* serotype associations with specific host species and disease syndromes are noted. Pneumonic pasteurellosis in cattle are caused mainly by serotype group A and D, while hemorrhagic septicemia in cattle and buffalo is caused by serotypes B:2 or E:2. *M. hemolytica* may occur associated with *P. multocida*.

Epidemiology

The disease appears to occur most often in animals that have encountered recent stressors such as transportation, weaning, or mixing with other animals from unrelated farms, adverse climatic conditions, and malnutrition. Collectively they pose suppression of the immune system which in turn increases susceptibility to pathogens and to morbidity and mortality.

Co-infection with potentially common pathogenic respiratory virus increases the susceptibility of cattle to secondary pasteurella infection. These include *parainfluenza 3 virus (PI-3)*, *bovine viral diarrhea virus (BVDV), bovine herpesvirus* 1 (BHV1), and bovine *respiratory syncytial virus (BRSV)*. Virus induced injury to the respiratory epithelium is believed to enhance bacterial attachment and subsequent colonization of target tissues. Most of the *Mycoplasma* species are known to contribute to the development of severe pneumonic lesions either alone or in association with pneumonic pasteurellosis of animals.

Pasteurelloses occur worldwide but are a particular problem in the tropics, especially the hot, humid tropics where environmental stress is an important trigger mechanism of this disease complex. Hemorrhagic septicemia is one of the economically most important diseases of cattle and buffaloes. The incidence of the disease peaks in the rainy season. Pasteurelloses of cattle and small ruminants also occur in other parts of the

tropics, but probably are mostly secondary opportunistic infections.

Environmental and other stress-causing influences are prerequisite for the development of pasteurelloses. Pasteurella infection mainly results from invasion of commensal organism during a period of stress, but exogenous transmission may occur by aerosol or contact exposure. Infection occurs by inhalation or ingestion of bacteria. Acutely infected animals excrete large amounts of pathogens which have in the meantime been enhanced in their virulence with saliva, feces, urine and milk. With shipping fever, it is the stress which is exerted on the animal through the conditions of the transportation which triggers the disease.

Clinical Features

Hemorrhagic septicemia (HS): The incubation period usually varies from 3 to 5 days. The disease may run a peracute, acute or subacute course. The peracute disease is characterized by sudden death in cattle, generally with no premonitory signs being noticed, but some animals may develop dyspnea, grunting and protraction for between a few and 24 hours prior to death.

Acutely and subacutely infected animals are inappetent, pyretic and show anorexia, depression, profuse salivation and nasal discharge, a rapid respiratory and pulse rate, sensory disorders and rumen paresis before death. The subacute disease is characterized by the development of a firm, subcutaneous, painful swelling of the submandibular region which may extend to the neck, brisket and forelegs. While at first defecation becomes reduced subsequently profuse diarrhea with evil-smelling and blood-containing feces appears, the anus and the vagina being swollen. Pregnant animals abort. Some animals may show circling movements and incoordination. Death can already appear 3-24 hours after the onset of the febrile reaction, but clinical signs can be present for periods ranging from 8 to 10 days. The disease is generally fatal if animals are not treated; the mortality rate can reach up to 98%. The peracute and acute HS usually is characterized by severe generalized congestion and widespread petechiae and ecchymoses of the serosal surfaces and subcutis. The edematous lesions along the mandibular space, the pharynx, around the parotis, at the neck, the brisket and shoulder are characteristic.

The course of pneumonic pasteurellosis is similar to HS, with serous nasal discharge, lacrimation and photophobia, partial closure of the eyes due to swelling of the eye lids and adventitious sounds audible on auscultation of the cranio-ventral lung-fields being specific clinical signs. Respiration is painful, the nostrils are wide open, and the animals grunt and cough.

Diagnosis

The clinical and pathological features of pasteurellosis are highly suggestive. If pasteurellas are found in a blood smear of febrile animals or fresh carcasses stained either with Giemsa or with polychrome methylene blue the diagnosis will be confirmed. With methylene blue staining, the pasteurella appear as bipolar bacteria.

Specific identification of the isolated organisms as to species and serotype can be done by using ELISA. With this assay, serotypes responsible for causing HS are readily distinguished from those *P. multocida* strains which are non-pathogenic.

Treatment

The infection with *Pasteurella* spp. can be treated efficiently with chloramphenicol, tetracyclines, penicillin, ampicillin and sulphonamides. It is mostly difficult to treat animals because of the acute or peracute course of the disease.

Control

Chemoprophylactic measures for preventing pasteurellosis are useful for preventing the outbreak of the disease, especially when disease-provoking stress is consciously put up with. Application of oxytetracycline LA before shipping animals over a long distance will protect the animals efficiently against shipping fever. The antibiotic chemoprophylaxis of pasteurellosis is the only way to stop the infection immediately during a sudden outbreak and prevents its spreading to other animals or herds.

Vaccines prepared from *Pasteurella* spp. organisms, mostly from the *P. multocida* serovars B and E adding *M. hemolytica* are used worldwide in the tropics to prevent pasteurellosis. So far it has not been possible to develop a vaccine which fulfills all expectations. Vaccines which contain mineral oil as an adjuvant are supposed to protect the animals for 12 months against infection. Prerequisite for the efficiency of a pasteurella vaccine is that it is prepared from the site-specific homologous pathogen and serovar. The application of a homologous or even herd-specific vaccine may often produce spectacular success, especially with opportunistic pasteurella infections.

The efficiency of vaccination schemes in preventing pasteurellosis depends on choosing the time of vaccination in such a way that the animals are in an optimal condition to produce an active immunity. Furthermore, appropriate keeping and feeding of the animals should minimize the emergence of stress.

6.5. Salmonellosis

Etiology

The genus *Salmonella* which belongs to the family Enterobacteriacae consists of two species (1) *S. enterica* which is divided into six sub species (*S. enterica* subsp. *enterica*, *S. e*. ssp. *salamae*, *S. e.* ssp. *arizonae*, *S. e.* ssp. *diarizonae*, *S. e.* ssp. *houtenae* and *S. e.* ssp. *indica*) and (2) *S. bongori* (formerly called S. e. ssp. bongori). The serovars of salmonella are differentiated from each other by the combination of their somatic (O) and flagellar (H) antigens and, to a lesser extent, by their biochemical reactions. Salmonellae are Gram-negative, motile (with exception of S. Gallinarum/Pullorum) rods.

Epidemiology

Salmonellas occur worldwide. A certain site-specificity of some serovars used to exist, but the pathogens have been distributed worldwide through international animal trade, especially by trading feed internationally, in particular fishmeal.

The *Salmonella* infection occurs mostly through the ingestion of infected feed and water. Calves aged between 3 and 12 weeks are the most frequent victims, but mature cattle may also suffer from the clinical disease. *S. Dublin* is regarded as a serovar specific to cattle and only rarely infects other species of animals and humans. Other serovars are not host-specific to cattle and direct or indirect transmission between cattle as well as between cattle and other domestic or wild animals may occur.

Clinical findings

The disease is most satisfactorily described as three syndromes, classified arbitrarily according to severity as septicemia, acute enteritis, and chronic enteritis.

Salmonella Dublin and *S*. Typhimurium are usually involved in cattle salmonellosis.

Septicemia is the common form of the disease in newborn calves under a few weeks of age. There is depression, toxemia, fever, dyspnea, and weakness; nervous signs, including incoordination and nystagmus, may occur. Diarrhea and dysentery may occur but are not common.

Calves older than a week, and adults, are usually affected by acute enteritis, followed in survivors by abortion in pregnant cows and polyarthritis in calves. In severe cases of enteritis, there is often dysentery, with whole blood passed in large clots, and complete agalactia in lactating cows. Abdominal pain, with kicking at the abdomen, rolling, crouching, groaning, and looking at the flanks, may occur in adult cattle. Rectal examination at this stage usually causes severe distress.

Chronic enteritis with inappetence, reduced weight gain, and unthriftiness may follow an attack of acute enteritis or be the only manifestation of the disease. Abortion is a common sequel in pregnant cows that survive an attack of acute enteritis. A sequel to some cases of apparent enteric salmonellosis is the development of terminal dry gangrene due to endarteritis of the extremities, including eartips, tailtip, and the limbs from the fetlock down.

Abortion due to *S*. Dublin may occur spontaneously without any previous clinical evidence of salmonellosis in the herd. Abortion has occurred from days 124 to 270 of gestation. Cows that abort may be ill with a fever, anorexia and hypogalactia and some will retain fetal membranes. In some cases, calves may be born shortly before term and die in the perinatal period.

Diagnosis

Mostly because of non-specific clinical symptoms and necropsy findings, a presumptive diagnosis has to be confirmed by bacteriological examination of feces or specimens from the affected animal. The final typing has to be done by determining the O- and H- antigens.

Treatment

Salmonellas are rather sensitive to a number of antibiotics, for instance ampicilin, amoxicillin, chloramphenicol, gentamicin, trimethorprim-sulphonamide combination, fluoroquinolones and nitrofuran derivatives. In addition to the causal treatment, supportive therapy and good nursing are important.

Control

Hygienic premises, cleanliness, provision of non-contaminated feed and drinking water as well as appropriate feeding are important prerequisites for the prevention of salmonellosis. In the tropics, fishmeal is often highly contaminated and may especially lead to the infection of poultry and pigs.

Infected and/or latently diseased animals have to be separated and treated. They can only return into the herd if they are found to be negative through bacteriological examination. An insufficient treatment may produce shedders which remain in the herd. Such carriers not only are a zoosanitary hazard for the farm but also may become a menace to human health through contaminated animal products.

6.6. Lumpy Skin Disease

Lumpy skin disease is an infectious, eruptive, occasionally fatal disease of cattle characterized by nodules on the skin and other parts of the body. Secondary bacterial infection often aggravates the condition.

Etiology

Lumpy skin disease (LSD) is associated with the Neethling poxvirus, a capripoxvirus. It has close antigenic relationship to sheeppox and goatpox viruses which are also in the same genus.

Epidemiology

LSD appears epidemically or sporadically. It is most prevalent along water courses and on low ground. Biting insects have been suspected as vectors. Experimentally, three species of hard ticks found in Africa have been shown to biologically transmit the virus. Because the disease can be experimentally transmitted by infected saliva, contact infection is considered as another route of infection. African buffalo are suspected as maintenance hosts in Kenya.

Traditionally, LSD is found in southern and eastern Africa, but it extended northwest through the continent into sub-Saharan West Africa. Since 2000, it has spread to several countries of the Middle East and in 2013

was confirmed in Turkey.

Clinical Findings

The incubation period is 4–14 days. 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 (Fig. 3) contain a firm, creamy-gray or yellow mass of tissue. Regional lymph nodes are swollen, and edema develops in the udder, brisket, and legs. 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.

Morbidity is 5%–50%; mortality is usually low. The greatest loss is due to reduced milk yield, loss of condition, and rejection or reduced value of the hide.

Diagnosis

Diagnosis depends on clinical findings of a generalized nodular skin disease with enlarged superficial lymph nodes and laboratory demonstration of antigens and serology.

Antigen detection- Electron microscopic demonstrations of typical capripox virions, virus isolation, antigen detection ELISA, FAT and PCR.

Serology- Virus neutralization, and the indirect fluorescent antibody tests are commonly used.

Treatment

Administration of antibiotics to control secondary infection and good nursing care are recommended.

Control

Vaccination with attenuated virus offers the most promising method of control. The viruses of goatpox and sheeppox passed in tissue culture have also been used.

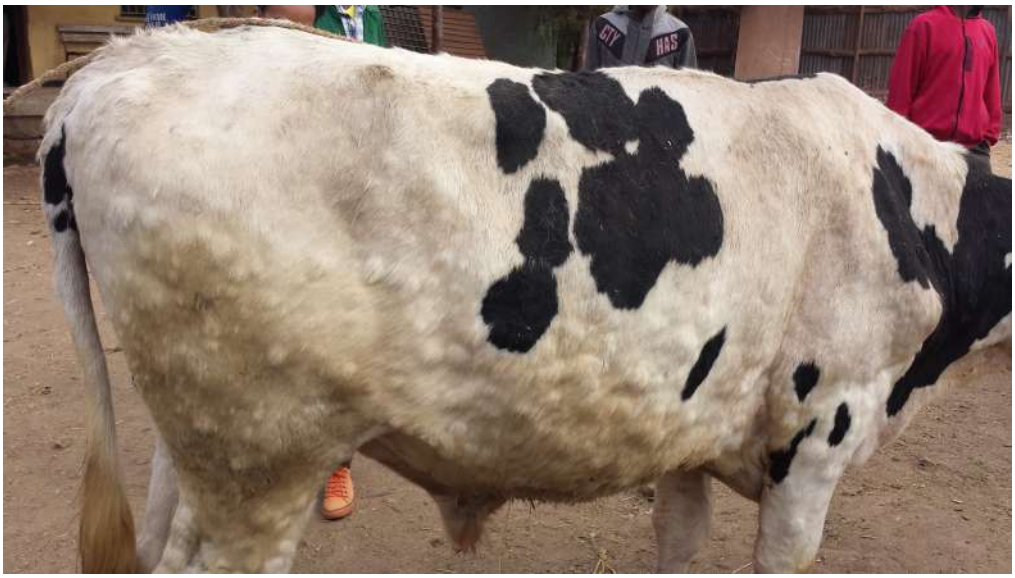


Figure 3. Lumpy skin disease (bull with skin nodules)

6.7. Foot-and-Mouth Disease

Foot-and-mouth disease (FMD) is a highly contageous viral disease of cloven-hoofed species characterized by fever and vesicles in the mouth and on the muzzle, teats, and feet. In a susceptible population, morbidity approaches 100%. The disease is rarely fatal except in young animals.

Cattle are the most susceptible. Domestic pigs are important hosts and are very effective in propagating the disease. Sheep and goats, all species of deer and antelope, Indian elephant, and giraffe are susceptible to FMD.

Most of sub-Saharan Africa has endemic FMD.

Etiology

FMD is caused by an aphthovirus of the family Picornaviridae. There are 7 immunologically distinct serotypes: A, O, C, Asia 1, and SAT (Southern African Territories) 1, 2, and 3. Within each serotype, there are a large number of strains that exhibit a spectrum of antigenic characteristics; therefore, more than one vaccine strain for each serotype, particularly O and A, is required to cover the antigenic diversity.

Epidemiology

Transmission of FMD is generally by contact between susceptible and infected animals. Infected animals have a large amount of aerosolized virus in their exhaled air, which can infect other animals via the respiratory or oral routes. All excretions and secretions from the infected animal contain virus, and virus may be present in milk and semen for up to 4 days before clinical signs appear. Aerosolized FMD virus can spread a considerable distance as a plume, depending on weather conditions, particularly when the relative humidity

is >60%. FMD has been transmitted to calves via infected milk, and milk tankers carrying infected milk have been implicated in the spread of disease between farms. Fodder can become contaminated after contact with infected animals and iatrogenic spread of FMD has been reported.

Ruminants that have recovered from infection and vaccinated ruminants that have contact with live FMD virus can serve as foci of infection and carry the virus in the pharyngeal region for up to 3.5 yr in cattle, 9 months in sheep, and ≥5 yr in African buffalo.

The primary site of infection and replication is usually the mucosa of the pharynx. The viremia persists for 4−5 days. Antibody production can be detected from 3-4 days after the first clinical signs and is usually sufficient to clear the virus.

Clinical Findings

The incubation period for FMD is 2–14 days, depending on the infecting dose, susceptibility of the host, and strain of virus. After the incubation period, anorexia and fever of up to 41°C may develop. Cattle salivate and stamp their feet as vesicles develop on the tongue, dental pad, gums, lips, and on the coronary band and interdigital cleft of the feet. Vesicles may also appear on the teats and udder particularly in lactating cows. Milk yield drops dramatically in milking animals, and all animals show a loss in condition and growth rate that may persist after recovery.

Diagnosis

Samples of vesicular epithelium or vesicular fluid should be sent in phosphate-buffered saline (PBS) (pH 7.4) to the responsible laboratory with due precautions.

Treatment

Treatment with mild disinfectant and protective dressings to inflamed areas to prevent secondary infection is recommended in endemic countries where a slaughter policy is not in force. A good symptomatic response is reported to the administration of flunixin meglumin.

Control

The occurrence of FMD in countries previously free of the disease can have a major effect on local and international trading arrangements. Many countries free of FMD have a policy of slaughter of all affected and in-contact susceptible animals and `strict restrictions on movement of animals and vehicles around infected premises. After slaughter, the carcasses are either burned or buried on or close to the premises, and the buildings are thoroughly washed and disinfected with mild acid or alkali and by fumigation.

In areas or countries free of FMD in which this is not possible, control is by movement restriction, quarantine of affected premises, and vaccination around (and possibly within) the affected premises. This has the

disadvantage that many carrier animals may remain after the outbreak, and quarantine may not be sufficiently long to prevent their subsequent movement.

In countries in which FMD is endemic, protection, particularly of high-yielding dairy cattle, is by a combination of vaccination and prevention of FMD virus entering the dairy premises. This can be difficult if prevalence of FMD in the unvaccinated population is high and climatic conditions are suitable for aerosol transmission.

FMD vaccine is a killed preparation and, at best, affords good protection against challenge for 4–6 months. However, the antigenic diversity of virus strains within each of the serotypes is an additional complication, so it is necessary to ensure that vaccines contain strains antigenically similar to the potential outbreak strains.

6.8. Bovine viral diarrhea (BVD)

Etiology

Bovine viral diarrheal virus (BVDV) is the causal agent of BVD and mucosal disease (MD) complex. Cattle are the primary host for BVDV. Isolates are separated into noncytopathic (NCP) and cytopathic (CP) biotypes. There are two genotypes (species) of BVDV namely, BVDV type 1 and BVDV type 2, and both CP and NCP BVDV are represented in each viral genotype.

Epidemiology

Serologic surveys indicate that BVDV is distributed worldwide. Although cattle of all ages are susceptible, most cases of overt clinical disease are seen in cattle between 6 mo and 2 yr old. Cattle that are persistently infected (PI) with NCP BVDV serve as a natural reservoir for virus.

Clinical Findings

Disease induced by BVDV varies in severity, duration, and organ systems involved. Acute BVD, also termed transient BVD, often is an inapparent to mild disease of high morbidity and low mortality. Biphasic fever (~40°C), depression, decreased milk production, transient inappetence, rapid respiration, excessive nasal secretion, excessive lacrimation, and diarrhea are typical signs. Clinical signs of disease usually are seen 6–12 days after infection and last 1–3 days. Lymphoid tissue is a primary target for replication of BVDV, which may lead to immunosuppression and enhanced severity of intercurrent infections.

Some isolates of BVDV induce clinically severe disease that manifests as high fever (~41–42°C), oral ulcerations, eruptive lesions of the coronary band and interdigital cleft, diarrhea, dehydration, leukopenia, and thrombocytopenia. The duration of overt disease may be 3–7 days. High morbidity with moderate mortality is common.

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 mo of fetal development may lead to embryonic resorption, abortion, growth retardation, or PI. 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.

Persistent infection (PI) is an important sequela of fetal infection with NCP BVDV. PI calves may appear healthy and normal in size, or they may show stunted growth and be prone to respiratory or enteric ailments. They often have a short life span, and death before 2 yr of age is common.

Mucosal disease is a highly fatal form of BVD that may be acute or chronic and is seen infrequently in PI cattle. Mucosal disease is induced when PI cattle become superinfected with CP BVDV.

Diagnosis

Laboratory tests for BVDV include virus isolation and assays that detect antibody in serum or detect viral RNA or viral antigen in clinical specimens and tissues. At necropsy, tissues of choice for viral isolation include spleen, lymph node, and ulcerated segments of the GI tract. Alternatives to viral isolation include antigen-capture ELISA to detect virus in blood, serum, or tissue biopsies; immunohistochemistry to detect viral protein in frozen or fixed tissues; PCR to detect viral RNA in clinical specimens.

Control

Control is based on sound management practices that include use of biosecurity measures, elimination of PI cattle, and vaccination. Replacement cattle should be tested for PI before entry into the herd. Quarantine or physical separation of replacement cattle from the resident herd for 2–4 wk should be considered, and vaccination of replacement cattle for BVD should be done before commingling with the resident herd. Embryo donors and recipients also should be tested for PI. AI should be done only with semen obtained from bulls free of PI.

Inactivated and modified live virus vaccines are available.

6.9. Infectious bovine rhinotracheatis (IBR)

Etiology

The bovine herpesvirus type-1 (BHV-1), or the infectious bovine rhinotracheitis (IBR) virus is the cause of the respiratory disease, abortion, conjunctivitis, and other clinical forms of the disease complex. There are at least three distinct BHV-l subtypes: a respiratory subtype, a genital subtype, and an encephalitic subtype

designated as BHV-1.1, BHV-1.2, and BHV-1.3, respectively. BHV-1.3 has been renamed BHV-5. Antigenic differences between isolates of the virus may account for some of the diverse epidemiological and pathological patterns of behavior of this herpesvirus.

Epidemiology

Disease complexes associated with this virus have been recognized in most cattle-raising parts of the world. Seroprevalence surveys have found that 41 to 67% of cattle are positive to the virus in Ethiopia (Lefevre, 1975; Bekele et al., 1989; Sibhat et al., 2018).

The respiratory form of clinical disease is most common in feedlot cattle, and cattle on diary and beef farms without a routine vaccination program.

The main sources of infection are the nasal exudate and coughed-up droplets, genital secretions, semen, and fetal fluids and tissues. Aerosol infection is the method of spread of the respiratory disease. Venereal transmission is the method of spread of the genital diseases. The virus may survive for up to 1 year in semen frozen at -196°C.

All ages and breeds of cattle are susceptible but the disease occurs most commonly in animals over 6 months of age. Newborn calves are highly susceptible to the systemic form of infection if the level of specific antibody to the virus in the colostrum is inadequate or if there is failure of transfer of passive immunity.

BHV-l virus can become latent. The virus may remain latent indefinitely and recrudescence, reactivation, and shedding of the virus can occur following the effects of stress such as transportation.

Clinical findings

Although infection can occur subclinically (Hage et al., 1996), primary BHV-1 infection is associated with three major clinical syndromes namely infectious bovine rhinotracheitis (IBR), infectious pustular vulvovaginitis (IPV) and infectious pustular balanoposthitis (IPB). The virus also causes a wide variety of other clinical syndromes such as abortion, infertility, conjunctivitis, encephalitis, mastitis, enteritis and dermatitis (Straub, 2000; Nandi et al., 2009).

Rhinitis, tracheitis and conjunctivitis (red nose)

In infected feedlots the disease occurs 10-20 days after the introduction of susceptible cattle.

There is considerable variation in the severity of clinical signs following natural infection. The clinical disease is usually mild in dairy cattle and in range beef cattle. A severe form of the disease can occur in feedlots where crowding and commingling from several sources occur. A severe form of upper respiratory tract disease and encephalitis have been reported in neonatal beef calves.

There is sudden onset of anorexia, loud coughing, fever (up to 42°C), severe hyperemia of the nasal mucosa,

with numerous clusters of grayish foci of necrosis on the mucous membranes of the nasal septum, a serous discharge from the eyes and nose, increased salivation, and sometimes a slight hyperexcitability. The respirations are increased in rate and are shallow. Respiratory distress is evident on exercise. A short, explosive cough is characteristic of some outbreaks.

In dairy cattle the disease is usually mild, characterized by inappetence, coughing, profuse bilateral serous nasal discharge, excessive salivation, nasal lesions, moderate fever, moderate drop in milk production, and recovery in a few days. Several animals may have the corneal form of the disease with obvious corneal edema, conjunctivitis, and profuse ocular discharge. The outbreak of respiratory disease will be followed by abortions in several days up to 90 days after the index case occurred.

Calves less than 6 months of age may develop encephalitis, which is marked by incoordination, excitement alternating with depression, and a high mortality rate. Salivation, bellowing, convulsions and blindness are also recorded.

Systemic disease in newborn calves

In newborn calves under 10 days of age, the systemic form of the disease is severe and highly fatal. Sudden anorexia, fever, excessive salivation, and rhinitis, often accompanied by unilateral or bilateral conjunctivitis, are common. The oral mucous membranes are usually hyperemic, erosions of the soft palate covered by tenacious mucus are common, and an acute pharyngitis covered by tenacious mucopurulent exudate is characteristic. The larynx is usually edematous and respiratory distress is common. Bronchopneumonia is also common.

Abortion

Abortion is a common sequel and occurs some weeks after the clinical illness. It is most common in cows that are 6-8 months pregnant. Retention of the placenta often follows.

IPV is characterized by frequent urination, elevation of the tail, and a mild vaginal discharge. The vulva is swollen, and small papules, then erosions and ulcers, are present on the mucosal surface. Mucosal ulcers may coalesce and sloughing of brown necrotic tissue may occur. Balanoposthitis is characterized by similar lesions of the glans penis and preputial mucosa.

Diagnosis

Virus isolation or detection of the virus using techniques such as ELISA and immunofluorescence, antibody detection using various serological tests, and PCR could be used for diagnosis.

Control

The current strategies for control are natural exposure, biosecurity, vaccination, or eradication of the virus from a herd or even the cattle population of a country.

Natural exposure

Cattle that have recovered from a natural infection with the virus are immune to further clinical disease. Abortion storms occur in herds that are not vaccinated and depend on natural exposure. Vaccination is therefore recommended in areas where the prevalence of infection is high and eradication is not feasible because of the extensive nature of the cattle population and movement of animals from one area to another.

Biosecurity

The introduction of new infections into herds can be prevented or minimized by purchasing animals directly from herds known to be free of a particular disease, testing animals for the infection before entry into the herd and keeping incoming animal in quarantine for several weeks before it is mixed with the other animals.

Vaccination

Both modified live-virus vaccines (MLV) and inactivated virus vaccines are available.

There are two types of MLV vaccines. One is a parenteral vaccine, and the other an intranasal vaccine. MLV vaccines offer 3 advantages over inactivated vaccines: a rapid immune response, relatively long duration of immunity and the induction of local immunity. The traditional MLV vaccines are safe and effective in preventing clinical disease and are more effective than inactivated vaccines.

Inactivated virus vaccines do not cause abortion, immunosuppression or latency. Inactivated vaccines, however, may not be as efficacious as MLV vaccines. They require two doses and protection is not, observed until 7-10 days following the second dose of the vaccine which is usually given 10-14 days after the primary vaccination.

The vaccines available for the control of diseases associated with BHV- l infection, in the developed world, are mostly multivalent antigen vaccines containing other respiratory pathogens such as PI-3, BRSV, and BVDV. Vaccines for IBR are not available in Ethiopia.

7. Trypanosomosis

Trypanosomosis is one of the most important cattle diseases in Ethiopia. It mainly occurs in the southwestern and western parts of the country infested with tsetse flies. The areas infested with the vector flies have conducive agro-climatic conditions for cultivation and livestock production. Trypanosomosis directly affects milk and meat production of animals, reduces calving rates, increases abortion and cause significant mortality. The indirect impact of the disease mostly lies on reduced availability and high cost of animals providing traction power for crop cultivation in affected areas (Swallow, 2000). It is estimated that some 10 to 14 million heads of cattle in Ethiopia are exposed to the risk of trypanosomosis. *Trypanosoma congolense* and *T. vivax* are the predominant species (Leta et al., 2016).

A meta-analysis of 24 published works in Ethiopia reported a pooled prevalence estimate of 8.12% for cattle

trypanosomosis, with significant difference between regions (Leta et al., 2016). The economic benefit expected from control interventions in bovine trypanosomosis in Ethiopia is enormous because of very high livestock densities and the importance of animal traction in crop cultivation (Shaw et al., 2013).

Control of trypanosomosis

Bouyer et al. (2013) discussed control strategies for African animal trypanosomosis that integrates vector control with use of trypanocides. According to them trypanosomosis control relies on implementation of local, integrated control strategies by communities or farmers that must take into account the eco-epidemiological context and the cattle rearing system to be sustainable (Bouyer et al., 2013).

In most situations, farmers and communities will need to conduct tsetse control in order to reduce their density enough to prevent or reduce transmission of trypanosomosis (Bouyer et al., 2013). Tsetse control may be complemented by insecticide treated cattle (ITC), a method which is optimal owing to its individualistic protection (i.e., at the level of the farmer rather than the community) and cost effectiveness (Shaw et al., 2013). For sedentary herds with free grazing activities, restricted application of insecticides using partial spraying every 2 weeks should be recommended. For transhumant herds, the treatment frequency should be reduced to once a month using pour-ons, or total spraying if water is available (Bouyer et al., 2009). For zero-grazing herds, instead of applying the insecticides on the animals, insecticide fences of 1-1.5 m can be used around cattle pens (Bauer et al., 2006).

However, ITC alone cannot protect cattle in sites where tsetse are dispersing from surrounding areas (Vale et al., 1999). Therefore, ITC should be complemented with insecticide treated targets (ITTs), which are either traps or screens impregnated with insecticides, set at strategic locations for maximum effect (Bouyer et al., 2013).

Even if ITC and ITT are applied in these areas, the transmission of highly pathogenic strains still requires the use of prophylactic and curative trypanocides (sanative pair) after diagnosis indicates infection. Clinical cases should be diagnosed and treated quickly using trypanocides, but prophylactic treatments should be avoided in order to reduce the development of drug-resistant strains of trypanosomes (Bouyer et al., 2013).

Therefore, integration of vector control and the use of trypanocides are necessary for reduction of trypanosomosis, the morbidity and mortality of cattle, and decreasing the incidence of drug-resistant strains of trypanosomes (Bouyer et al., 2010).

8. Internal Parasites

Helminth infections of ruminants are a major constraint on efficient livestock production globally. Most grazing ruminants are infected by a variety of helminth parasites which negatively affect growth rate, milk yield and reproductive potential (Knox et al., 2012; van der Voort et al., 2013).

The underlying mechanisms for the impact of helminths on production can be divided into three main categories: (i) direct tissue damage and decreased functioning of the affected organs (Murray et al., 1970), (ii) diversion of energy and protein resources of the host from production towards defense and immune mechanisms (Greer et al., 2008), and (iii) reduced feed intake. Reduced feed intake is a common feature of all helminth infections, is linked with hormonal changes in the host, and is thought to be the major mechanism of subclinical production impact (Fox et al., 1989; Forbes et al., 2009).

8.1. GI nematodes

Gastrointestinal (GI) nematodes are very important in cattle in Ethiopia. They are highly prevalent and widely distributed all over the country. They cause economic losses due to poor weight gain, weight loss, drop in milk production, infertility (Knox et al., 2012; van der Voort et al., 2013) and even mortality particularly in calves. GI nematodes of pathogenic importance in cattle prevalent in Ethiopia include *Haemonchus* spp., *Trichostrongylus* spp., *Strongyloides* spp., *Oesophagostomum* spp., *Bunostomum* spp., *Toxocara vitulorum* and *Trichuris* spp. (Degefu et al., 2011; Bacha and Haftu, 2014; Terfa et al., 2023)

Most GI nematodes cause gastroenteritis and thereby reduce efficiency of nutrient absorption leading to loss of productivity. Some GI nematodes (e.g. Haemonchus, Bunostomum) cause anemia and blood protein loss through feeding on hosts' blood (blood sucking habit of the parasite) and associated bleeding into the lumen of the GI tract.

The lifecycle of GI nematodes is direct i.e., there is no involvement of intermediate host. Adult parasites live in the GI of the host and eggs are shed with the feces. The eggs develop into L3 (the infective larval stage) in the environment and ingestion of L3 with forage/feed leads to infection of the host. Under a favorable environmental condition (high humidity, and temperature – 18-26^{o}C) the eggs develop into L3 within 2 weeks (Urquhart et al., 1996).

Control of GI nematodes

Theoretically GI nematodes can be controlled by strategic use of anthelmintics (strategic deworming) and grazing/pasture management.

Under Ethiopian traditional cattle production systems, where grazing is usually on communal lands, use of anthelmintics might be the only feasible alternative available for the control of helminth parasites. Anthelmintics can be used either for the treatment of affected animals (showing certain signs) or mass treatment of herds at certain times of the year for strategic control. The prevalence of GI nematodes follows a certain pattern in Ethiopia, prevalence being high during the rainy/wet seasons (particularly during the long rains) (Tembely et al., 1997; Terfa et al., 2023). This allows for the use of deworming during critical periods for strategic control of the parasites. Some authors recommend routine deworming twice a year for sheep, though they differ in the exact time of the year the treatment should be administered (Bekele et al., 1987; Njau et al., 1990). The general principle in common use in strategic deworming involves treating animals to reduce pasture contamination with parasite eggs when the external condition is favorable for development of the eggs into infective larvae, and to relieve the animals of infection before they are exposed to conditions that make them more susceptible to effects of the infection. Under Ethiopian condition, this translates into deworming animals at the beginning of the rainy season/at the end of the dry season for prevention of contamination of pasture with parasite eggs during the rainy season when the environment is favorable for their development into infective stage, and the other during the start of the dry season to remove parasites picked during the rainy season. Deworming animals during the dry season would help them better tolerate the nutritional stress common during this season in Ethiopia. Individual animal treatment involves assessment of the animal for potential parasitic infection before deworming. Mature cattle are relatively resistant to nematode parasites while young stock is the most susceptible. Therefore, it might be necessary to compare the cost of routine deworming in adult cattle against the benefits depending on the epidemiology of the parasites in a particular geographic area. Young grazing calves may require more frequent treatment during the rainy seasons.

8.2. Liver flukes (Fasciola)

Fasciolosis is an economically important disease of cattle and sheep. *Fasciola hepatica* and *F. gigantica* are the two species most commonly implicated in fasciolosis (Andrews, 1999). Infection with both *F. hepatica* and *F. gigantica* is very common in Ethiopian cattle (Abebe et al., 2010; Abunna et al., 2010; Aragaw et al., 2012). Infection can significantly affect milk yield, weight gain and reproductive performance of cattle (Hope-Cawdery et al., 1977; Urquhart et al., 1996; Elliott et al., 2015). In addition to its effect on productivity, fasciolosis is a cause of significant economic losses through liver condemnation at slaughter (Abebe et al., 2010; Aragaw et al., 2012).

Clinically liver fluke infection can be either acute/subacute or chronic. Acute infection occasionally occurs in young calves when very large number of metacercaria are ingested in a relatively short duration of time leading to acute damage to the liver due to massive migration of immature flukes through liver parynchyma, and can kill calves. Chronic liver fluke infection, the most common type of the infection, occurs when infection buildup slowly overtime, and can lead to weight loss, anemia, edema and scours.

Fasciola hepatica is prevalent in the highlands of Ethiopia, while *F. gigantica* is the most prevalent species in the lowlands (Yilma and Malone, 1998).

The lifecycle of liver flukes is indirect and involves a snail intermediate host: an amphibious snail *Lymnaea truncatula* for *F. hepatica* and an aquatic snail *L. natalensis* for *F. gigantica* (Urquhart et al., 1996).

Mature liver flukes reside in the bile ducts and eggs are shed with feces. In the availability of sufficient moisture eggs develop and hatch releasing motile ciliated miracidia, which are infective to the snail intermediate host. After infecting the snail the miracidia develop into sporocyst and then redia. The redia develop into cercaria. The cercaria are shed from the snail as motile forms which attach themselves to firm surfaces, such as grass blades, and encyst there to form the infective metacercariae. The metacercariae are the infective stage the ingestion of which leads to infection. The prepatent period of liver fluke infection in cattle is 10 to 12 weeks (Urquhart et al., 1996).

Control of liver fluke infection

Liver flukes may be controlled through control of the population of intermediate snail hosts using various mechanisms or through preventing/limiting access of cattle to the risky grazing areas or through use of anthelmintics. Under the Ethiopian traditional cattle rearing condition use of anthelmintics at certain critical times of the year might be the most feasible control alternative. The amphibious snail intermediate host of *F. hepatica* proliferates only in the long rainy season in the highlands of Ethiopia. Therefore, infection with *F. hepatica* usually occurs at the end of the rainy season (Jacinta, 1983; Njau et al., 1988) and clinical signs are usually observed in the dry season. Therefore, routine deworming at the beginning of the dry season would help to remove infection picked late in the rainy season. Treatment at this time of the year should be made with anthelmintic effective against both immature and mature flukes (e.g., triclabendazole) for better effect. Removing the fluke burden at the beginning of the dry season helps the animals to better tolerate the nutritional stress, prevailing during the dry season. Second treatment at the beginning of the main rainy season would help reduce the number of eggs shed and contaminate the pasture during the rainy season when there will be critical population of snail intermediate hosts in the environment (Tembely et al., 1996). The aquatic snail intermediate host of *F. gigantica* is usually associated with permanent water bodies in the lowlands of Ethiopia. Therefore, transmission is possible throughout the year. However, infection mostly occurs during the dry season when grazing is scarce and animals are therefore taken to areas of permanent water bodies for pasture and watering. Therefore cattle may be treated before (taking the animals to the grazing area) beginning of grazing of the area to reduce the number of eggs shed and infect the intermediate host as miracidia, and at the end of the grazing season (at the water body) to remove the fluke burden. However, it should be remembered that routine anthelmintic treatment is practiced only in areas with high risk of liver fluke infection as evidenced by clinical and laboratory examinations or literature information.

As the types and the epidemiology of internal parasites likely to infect cattle in different areas of Ethiopia may vary, the local epidemiology of helminth parasites should be sought before suggesting/recommending drenching programs.

9. Tick Control

Ticks are very important parasites of livestock widely distributed throughout the world (Estrada-Peña et al., 2014). They have a direct and indirect impact on hosts. The direct economic effect on production results from damage caused by tick bites in heavily infested animals (Rodriguez-Vivas, 2017). Heavy tick infestation produces tick worry, blood loss, reduced weight gain, local skin infection, lesions predisposing the animal to myiasis and damaged hide (Wall and Shearer, 2001). Indirect losses stem from the effects of tick-transmitted pathogens (de Castro, 1997), as ticks are very effective vectors for important animal and human diseases.

Ticks are prevalent in all agro-ecologies in Ethiopia (Shichibi et al., 2017; Bayew and Ewnetu, 2019), although their abundance and species composition differ significantly. The major genera of ticks reported in Ethiopia are *Amblyomma, Boophilus, Haemaphysalis, Hyalomma*, and *Rhipicephalus* (Moges et al., 2012; Mekonnen et al., 2001; De Castro, 1994), and the main cattle tick-borne diseases in Ethiopia include anaplasmosis, babesiosis, heartwater/erlichiosis, and theileriosis (Moges et al., 2012). Ticks not only cause economic losses through direct mortality of high-grade animals, but also by hindering genetic improvement (productive potential) of entire herds or regions (Kariuki et al., 1995; Kivaria, 2006).

In terms of their role in disease transmission and distribution, the most important tick species in Ethiopia are *A. variegatum* (vector of *Erlichia ruminantum* and *Theileria mutans*) and *Boophilus decoloratus* (vector of *Anaplasma marginale* and *Babesia bigemina*). *A. variegatum* has also been associated with susceptibility to dermatophilosis infection in sheep (Lloyd and Walker, 1993).

Several research reports on the epidemiology of ticks in Ethiopia call for the development of strategies for the control of the parasite (Tessema and Gashaw, 2010; Ayana et al., 2021; Adugna and Tamrat, 2022). Although the use of methods such as resistant animals/breeds and anti-tick vaccines are mentioned as complementary or prospective tools in tick control, use of acaricides remains to be the main tick control option throughout the world. Acaricides are conventionally applied to animals by dipping or spraying, while 'pour-on' formulations, and acaricide-impregnated ear-tags and bands have also been developed (Jongejan and Uilenberg, 1994). Hand spraying is the most likely method of choice for herds managed under the traditional mixed crop-livestock and pastoral/agro-pastoral systems in Ethiopia as the cost of establishing and maintaining motorized spray races and dip tanks is high and unaffordable.

However, acaricide use should be judicious, because these chemicals are toxic, leave residues in meat and milk, and cause environmental pollution (Drummond, 1976). Moreover, development of the resistance of

ticks to acaricides due to excess use poses an increasing threat to livestock production (Wharton, 1976). Acaricides need also be used in such a way that it doesn't disturb enzootic stability of tick-borne diseases. If endemic stability is to be established and maintained, complete elimination of ticks must not be the goal of tick control on the farm. This is because ticks are required for sustaining challenge to tick-borne diseases. In order to reduce use of acaricides and limit their side effect on the environment, enzootic stability and food safety, the frequency of their use should take into account severity of infestation, season and breed of the animals. It would however be necessary to determine the threshold level of tick infestation that warrants application of acaricides, and the seasonal variations of tick infestations for strategic control. In order to establish and maintain an endemically stable state, controlled exposure of calves to ticks is encouraged (Kasaija et al., 2021). It is important to spray calves with acaricides during the tick season depending on the severity of the infestation. The indigenous cattle breeds, which constitute almost the entirety of the traditional cattle herds in Ethiopia, are considered to be relatively resistant to ticks and tick-borne diseases.

10. Health Management in Feedlots

(Adapted from Radostits et al., 2007)

10.1. Simple Indigestion

Simple indigestion in ruminants is a minor disturbance in GI function that occurs most commonly in cattle and rarely in sheep and goats. It is typically related to an abrupt change in the quality or quantity of the diet. The disease is common in stall-fed beef cattle because of the variability in quality and the large amounts of feed consumed. It is not commonly observed in pastured beef cattle because they are less heavily fed.

Clinical findings

A reduction in appetite is the first clinical finding. The anorexia may be partial or complete. There is mild depression and dullness. Rumination ceases and the ruminal movements are depressed in frequency and amplitude and sometimes are almost absent. The rumen may be larger than normal if the cause is sudden access to an unlimited supply of palatable feed. There may be moderate tympany, especially with frozen or damaged feeds or in allergy, but the usual finding is a firm, doughy rumen without obvious distension. The feces are usually reduced in quantity and are drier than normal on the first day. However, 24-48 hours later the animal is commonly diarrheic; the feces are softer than normal, voluminous and commonly malodorous. There is no systemic reaction and the heart rate, temperature and respirations are usually within normal ranges. Mild abdominal discomfort may be present for several hours. Most cases recover spontaneously or with simple treatments in about 48 hours.

Cause

The common causes are dietary abnormalities of minor degree including indigestible roughage, particularly when the protein intake is low, moldy, overheated and frosted feeds, and moderate excesses of grain and

concentrate intake. Gross overfeeding usually occurs when cattle gain accidental access to large quantities of grain or are suddenly introduced to high-grain diets in feedlots. A sudden change to a new source of grain may have the same effect. It is probable that limitation of the available drinking water may contribute to the occurrence of the disease during dry seasons.

Management

Most cases of simple indigestion recover spontaneously. Small quantities of fresh, good-quality, palatable hay should be provided several times daily to encourage eating and to stimulate reticulorumen motility.

Administration of warm water or saline (0.9% NaCl) solution (~20 L, orally [eg, as a drench or via stomach tube], once), followed by vigorous kneading of the rumen, may help restore rumen function in adult cattle. Epsom salts (0.5-1.0 kg per adult cow) and other magnesium salts are reasonably effective and have the merit of simplicity and cheapness.

If an excessive quantity of grain is the cause of the simple indigestion, the use of alkalinizers, such as magnesium hydroxide, at the rate of 400 g per adult cow (450 kg BW), is recommended when the rumen contents are excessively acid. Magnesium oxide or hydroxide should be used only if ruminal acidosis is present. If the rumen contents are dry, 15-30 L of water should be administered by stomach tube. Acetic acid or vinegar, 5-10 L, is used when the rumen contents are alkaline as a result of the ingestion of high-protein concentrates.

In cases of indigestion that have run a course of more than a few days, and in animals that have been anorexic for prolonged periods, there will be significant loss of ruminal microflora, especially if there have been marked changes in pH. Reconstitution of the flora by the use of cud transfers from normal cows is highly effective.

10.2. Acute carbohydrate engorgement (Grain overload, Ruminal lactic acidosis, Rumen overload)

Grain overload is an acute disease of ruminants that is characterized by forestomach hypomotility to atony, dehydration, acidemia, diarrhea, depression, incoordination, collapse, and in severe cases, death. The sudden ingestion of toxic doses of carbohydrate-rich feed, such as grain, is the most common cause of the acute form of the disease.

Previous diet and change of ration

Because the type and level of ration consumed by a ruminant affects the numbers and species of bacteria and protozoa in the rumen, a change from one ration to another requires a period of microbial adaptation. Animals being fed a low-energy ration are most susceptible to a rapid change to a high energy ration because satisfactory adaptation cannot occur quickly enough. This results in the rapid onset of abnormal fermentation.

Accidental consumption of excess carbohydrates

The disease occurs commonly following accidental consumption of toxic amounts of grain by cattle gaining sudden access to large quantities of grain. The accidental feeding of a high-level grain ration to cattle that are on a highlevel roughage ration is a common cause of the disease.

Depending on the species of grain, the total amount eaten and the previous experience of the animals, the morbidity will vary from 10-50%. The case fatality rate may be up to 90% in untreated cases, while in treated cases it still may be up to 30-40 %.

Wheat, barley and corn grains are the most toxic when ingested in large quantities. Oats and grain sorghum are least toxic. All grains are more toxic when ground finely or even crushed or just cracked - processes that expose the starch component of the grain to the ruminal microflora. The amount of a feed required to cause acute illness depends on the species of grain, previous experience of the animal with the grain, its nutritional status and body condition score, and the nature of the ruminal microflora.

Pathogenesis

Changes in rumen microflora

The ingestion of excessive quantities of highly fermentable feeds by a ruminant is followed within 2-6 hours by a marked change in the microbial population in the rumen. There is an increase in the number of *Streptococcus bovis*, which utilize the carbohydrate to produce large quantities of lactic acid. In the presence of a sufficient amount of carbohydrate (a toxic or a lethal amount) the organism will continue to produce lactic acid, which decreases the rumen pH to 5 or less, which results in the destruction of the cellulolytic bacteria and protozoa. When large amounts of starch are added to the diet, growth of *S. bovis* is no longer restricted by energy source and it multiplies faster than any other species of bacteria.

Volatile fatty acids (VFAs) and lactic acid in the rumen

The concentration of VFAs increases initially, contributing to the fall in ruminal pH. The low pH allows lactobacilli to use the large quantities of carbohydrate in the rumen to produce, excessive quantities of lactic acid, resulting in ruminal lactic acidosis, which markedly increases ruminal osmolality, and water is drawn in from the systemic circulation, causing hemoconcentration and dehydration.

Some of the lactic acid is buffered by ruminal buffers but large amounts are absorbed by the rumen and some moves into and is absorbed further down the intestinal tract. Lactate is a 10 times stronger acid than the VFAs, and accumulation of lactate eventually exceeds the buffering capacity of rumen fluid. As the ruminal pH declines, the amplitude and frequency of the rumen contractions are decreased and at about a pH of 5 there is ruminal atony.

Systemic lactic acidosis

The absorbed lactic acid is buffered by the plasma bicarbonate buffering system. With nontoxic amounts of lactic acid, the acid-base balance is maintained by utilization of bicarbonate and elimination of carbon dioxide by increased respirations. In severe cases of lactic acidosis the reserves of plasma bicarbonate are reduced, the blood pH declines steadily, the blood pressure declines, causing a decrease in perfusion pressure and oxygen supply to peripheral tissues and resulting in a further increase in lactic acid from cellular respiration. Renal blood flow and glomerular filtration rate are also decreased, resulting in anuria. Eventually there is shock and death.

Chemical and mycotic rumenitis

The high concentration of lactic acid in the rumen causes chemical rumenitis, which is the precursor for mycotic rumenitis in those that survive; this occurs about 4-6 days later. The low pH of the rumen favors the growth of Mucor, Rhizopus and Absidia spp. which invade the ruminal vessels, causing thrombosis and infarction. Severe bacterial rumenitis also occurs. Widespread necrosis and gangrene may affect the entire ventral half of the ruminal walls and lead to the development of an acute peritonitis. The damage to the viscus causes complete atony and this, together with the toxemia resulting from the gangrene, is usually sufficient to cause death.

Hepatic abscesses

Hepatic abscesses commonly occur as a complication as a result of a combination of rumenitis caused by lactic acidosis and allowing *Fusobacterium necrophorum* and *Arcanobacter (Corynebacterium) pyogenes* to enter directly into ruminal vessels and spread to the liver.

Clinical findings

Within a few hours after engorgement, the only abnormalities that may be detectable are a distended rumen and abdomen, and occasionally some abdominal discomfort, evidenced by kicking at the belly. In the mild form, affected cattle are anorexic and still fairly bright and alert, and the feces may be softer than normal. Rumen movements are reduced but not entirely absent. Affected cattle do not ruminate for a few days but usually begin to eat on the third or fourth day without any specific treatment.

Depression, dehydration, inactivity, weakness, abdominal distension, diarrhea and anorexia are typical. The temperature is usually below normal, 36.5-38.5^{o}C. The heart rate in cattle is usually increased and continues to increase with the severity of the acidosis and circulatory failure. In general, the prognosis is better in those with heart rates below 100/min than those with rates up to 120-140/min. The respirations are usually shallow and increased up to 60-90/min. A mucopurulent discharge is common because animals fail to lick their nares.

Diarrhea is almost always present and usually profuse, and the feces are light-colored with an obvious sweet-

sour odor. The feces commonly contain an excessive quantity of kernels of grain in grain overload, and pips and skins when grapes or apples have been eaten. An absence of feces is considered by some veterinarians as a grave prognostic sign but diarrhea is much more common. The dehydration is severe and progressive. In mild cases, the dehydration is about 4-6% BW, and with severe involvement up to 10-12% BW. Anuria is a common finding in acute cases and diuresis following fluid therapy is a good prognostic sign.

Careful examination of the rumen is important. The rumen contents palpated through the left paralumbar fossa may feel firm and doughy in cattle that were previously on a roughage diet and have consumed a large amount of grain. In cattle that have become ill on smaller amounts of grain, the rumen will not necessarily feel full but rather resilient because the excessive fluid contents are being palpated. Therefore, the findings on palpation of the rumen may be deceptive and a source of error. The primary contractions of the reticulorumen are usually totally absent, although low-pitched tinkling and gurgling sounds associated with the excessive quantity of fluid in the rumen are commonly audible on auscultation of the rumen. The ruminal fluid is a milky green to olive brown color and has a pungent acid smell. Collection of a sample of ruminal fluid in a glass beaker will reveal an absence of foam. The pH of the rumen fluid is usually below 5.

Severely affected animals have a staggery, drunken gait and their eyesight is impaired. They bump into objects and their palpebral eye preservation reflex is sluggish or absent. The pupillary light reflex is usually present but slower than normal. Acute laminitis may be present and is most common in cases that are not severely affected and appear to be good treatment risks. Affected animals are lame in all four feet, shuffle while they walk slowly and may be reluctant to stand. The lameness commonly resolves if the animals recover from the acute acidosis. Evidence of chronic laminitis may develop several weeks later.

Recumbency usually follows after about 48 hours but may occur earlier. Affected animals lie quietly, often with their heads turned into the flank. A rapid onset of recumbency suggests an unfavorable prognosis. Evidence of improvement during this time includes a fall in heart rate, rise in temperature, return of ruminal movement and passage of large amounts of soft feces.

Diagnosis

The pH of the ruminal fluid obtained by stomach tube or by rumenocentesis through the left paralumbar fossa can be measured in the field using wide-range pH (2-12) Indicator paper. The ruminal fluid must be examined immediately because the pH will increase upon exposure to air. A ruminal pH of 5-6 in roughage fed cattle suggests a moderate degree of abnormality but a pH of less than 5 suggests severe grain overload and the need for energetic treatment.

Microscopic examination of a few drops of ruminal fluid on a glass slide (with a coverslip) at low power will reveal the absence of ruminal protozoa, which is a reliable indicator of an abnormal state of the rumen,

usually acidosis. The predominantly Gram-negative bacterial flora of the rumen is replaced by a Gram-positive one.

The hematocrit rises from a normal of 30-32% to 50-60% in the terminal stages and is accompanied by a fall in blood pressure. Blood lactate and inorganic phosphate levels rise and blood pH and bicarbonate fall markedly.

The urine pH falls to about 5 and becomes progressively more concentrated; terminally there is anuria.

Treatment

The principles of treatment are:

- Correct the ruminal and systemic acidosis and prevent further production of lactic acid
- Restore fluid and electrolyte losses and maintain circulating blood volumes
- Restore forestomach and intestinal motility to normal.

There are at least two common clinical situations encountered. One is when cattle have been found accidentally eating large quantities of grain, are not yet ill and all appear similar clinically except for varying degrees of distension depending on the amount each animal has consumed. In the other situation, the engorgement occurred 24-48 hours previously and the animals have clinical evidence of lactic acidosis.

When cattle are found engorging themselves, the following procedures are recommended:

- Prevent further access to feed
- Do not provide any water for 12-24 hours
- Offer a supply of good-quality palatable hay equal to one-half of the daily allowance per head
- Exercise all animals every hour for 12-24 hours to encourage movement of the ingesta through the digestive tract.

Those cattle that have consumed a toxic amount of grain will show signs of anorexia, inactivity and depression in approximately 6-8 hours and should be identified and removed from the group for individual treatment. Those cattle that did not consume a toxic amount are usually bright and alert and will usually begin eating hay if it is offered.

After 18-24 hours, those cattle that have continued to eat hay may be allowed free access to water. Those with clinical evidence of grain overload must be identified and treated accordingly. They will engorge themselves with water if allowed free access to it. The rumen becomes grossly distended with fluid and affected cattle may die 18-24 hours later from electrolyte disturbances and acid-base imbalance.

In certain situations, if feasible and warranted by economics, such as when finished beef cattle have

accidentally engorged on grain, emergency slaughter may be the most economical course of action.

Rumenotomy

In severe cases, in which there is recumbency, severe depression, hypothermia, prominent ruminal distension with fluid, a heart rate of 110-130/min and a rumen pH of 5 or below; a rumenotomy is the best course of action. The rumen is emptied, washed out with a siphon and examined for evidence of and the extent of chemical rumenitis, and a cud transfer (10-20 L of rumen juice) is placed in the rumen along with a few handfuls of hay.

The rumenotomy will usually correct the ruminal acidosis and an alkalinizing agent in the rumen is not necessary. The major disadvantages of a rumenotomy are time and cost, particularly when many animals are involved.

Intravenous sodium bicarbonate and fluid therapy

The systemic acidosis and the dehydration are treated with IV solutions of 5% sodium bicarbonate at the rate of 5 L for a 450 kg animal given initially over a period of about 30 minutes. This will usually correct the systemic acidosis. This is followed by isotonic sodium bicarbonate (1.3%) at 150 Ml/kg BW IV over the next 6-12 hours. Cattle that respond favorably to the rumenotomy and fluid therapy will show improved muscular strength, begin to urinate within 1 hour and attempt to stand within 6-12 hours.

Rumen lavage

In less severe cases, in which affected cattle are still standing but are depressed, their heart rate is 90-100/min, there is moderate ruminal distension and the -rumen pH is between 5 and 6, an alternative to a rumenotomy is rumen lavage if the necessary facilities are available. A large 25-28 mm inside-diameter rubber tube is passed into the rumen and warm water is pumped in until there is an obvious distension of the left paralumbar fossa; the rumen is then allowed to empty by gravity flow. The rumen can be almost completely emptied by 10-15 irrigations. With successful gastric lavage, alkalinizing agents are not placed in the rumen but the systemic acidosis is treated as described above.

Intraruminal alkalinizing agents

In moderately affected cases, the use of 500 g of magnesium hydroxide per 450 kg BW, or magnesium oxide in 10 L of warm water pumped into the rumen and followed by kneading of the rumen to promote mixing will usually suffice.

Control and prevention

Cattle can be started, grown and finished on high-level grain rations successfully, providing they are allowed a gradual period of adaptation during the critical period of introduction. The important principle of prevention is that the ruminant can adapt to an all-concentrate ration.

10.3. Ruminal tympany (Bloat)

Ruminal tympany is abnormal distension of the rumen and reticulum (Fig. 4) caused by excessive retention of the gases of fermentation, either in the form of a persistent foam mixed with the rumen contents or as free gas separated from the ingesta. The volumes of gas in a bloated cow are large, 50-70 L, and there is an exponential increase in intraruminal pressure with increasing rumen volume, especially as the potential for further increases in the abdomen diminishes.

What causes it? Predisposing factors

Normally, gas bubbles produced in the rumen coalesce, separate from the rumen contents to form pockets of free gas above the level of the contents and finally are eliminated by eructation.

There are two types of bloat: primary (frothy) bloat and secondary (free-gas) bloat.

Primary ruminal tympany (frothy bloat)

Primary ruminal tympany or frothy bloat is caused by the production of a stable foam that traps the normal gases of fermentation in the rumen. The essential feature is that coalescence of the small gas bubbles is inhibited and intraruminal pressure increases because eructation cannot occur.

A grassfed cow can produce 100 L during the first hour of feeding. A cow maintained on a legume diet may produce 200 L per hour.

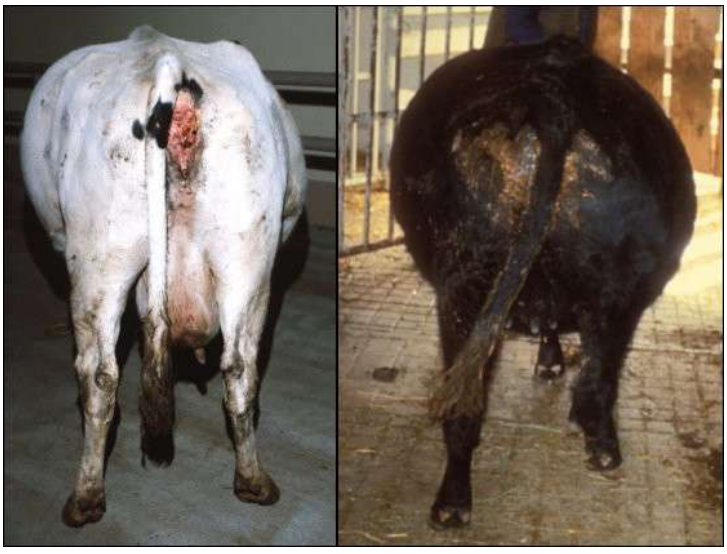


Figure 4. Bloated cattle

Pasture bloat

Pasture bloat occurs in both dairy and beef cattle that graze pastures consisting of bloating forages. The

incidence is highest when the pasture is lushest and contains a high concentration of soluble proteins. Leguminous or pasture bloat is due to the foaming qualities of the soluble leaf proteins in bloating legumes and other bloating forages. In general, bloat-causing legumes are susceptible to rapid digestion by rumen microflora, while bloat-safelegumes are digested more slowly.

Distension of the rumen occurs quickly, sometimes as soon as 15 minutes after going on to bloat-producing pasture, and the animal stops grazing. The distension is usually more obvious in the upper left paralumbar fossa but the entire abdomen is enlarged. There is discomfort and the animal may stand and lie down frequently, kick at its abdomen and even roll. Frequent defecation and urination are common. Dyspnea is marked and is accompanied by mouth breathing, protrusion of the tongue, salivation and extension of the head. The respiratory rate is increased up to 60/min. Occasionally, projectile vomiting occurs and soft feces may be expelled in a stream.

The course in ruminal tympany is short but death does not usually occur in less than 3-4 hours of the onset of clinical signs. Collapse and death almost without struggle occur quickly. If animals are treated by trocarization or the passage of a stomach tube, only small amounts of gas are released before froth blocks the cannula or tube.

To prevent pasture bloat it is important to feed hay before turning cattle on pasture. Continual administration of an antifoaming agent during the risk period is also a satisfactory method to prevent pasture boating.

Feedlot bloat

Feedlot bloat occurs in feedlot cattle during the 50-100 days when cattle are fed large quantities of grain and small quantities of roughage. In some cases the use of pelleted, finely ground feed has been associated with outbreaks of feedlot bloat. Finely ground grain promotes frothiness of rumen contents although the cause is not clear.

The feeding of large quantities of grain to cattle results in marked changes in the total numbers and proportions of certain ruminal protozoa and bacteria. Some species of encapsulated bacteria increase in numbers and produce a slime which may result in a stable foam. Feedlot bloat may also be of the freegas type based on the observations that gas may be easily released with a stomach tube. Feedlot cattle that die of bloat are commonly found dead in the morning.

To prevent feedlot bloat, rations should contain ≥10-15% cut or chopped roughage mixed into the complete feed. Preferably, the roughage should be a cereal, grain straw, grass hay, or equivalent. Grains should be rolled or cracked, not finely ground.

Treatment

The approach to treatment depends whether the bloat is frothy or due to free gas, and whether or not the bloat is life threatening.

Emergency rumenotomy

Using a sharp knife, a quick incision 10-20cm in length is made over the midpoint of the left paralumbar fossa through the skin and abdominal musculature and directly into the rumen. There is remarkably little contamination of the peritoneal cavity, and irrigation and cleaning of the incision site followed by standard surgical closure usually results in uneventful recovery with only occasional minor complications.

Promote salivation

For less severe cases, owners may be advised to tie a stick in the mouth like a bit on a horse bridle to promote the production of excessive saliva, which is alkaline and may assist in denaturation of the stable foam. Walking animals for a few minutes usually help them to eructate. Walking may result in loss of stability of foam and thereby help to coalescence into large bubbles.

Antifoaming agents

Any nontoxic oil, especially a mineral one that persists in the rumen, not being biodegradable, is effective. Their effect is to reduce surface tension and foam. A dose of 250 mL is suggested for cattle but doses of up to 500 mL are commonly used. An emulsified oil or one containing a detergent such as dioctyl sodium sulfosuccinate is preferred because it mixes effectively with ruminal contents. Of the synthetic surfactants, poloxalene is the one in most general use for leguminous bloat at a dose of 25-50 g. It is not as effective for feedlot or grain bloat.

Secondary ruminal tympany (free-gas bloat)

In free-gas bloat the gas bubbles coalesce and separate from the rumen fluid but the animals cannot eructate the pockets of free gas because of abnormalities of the reticulorumen or esophagus. Physical obstruction to eructation occurs in esophageal obstruction caused by a foreign body, by stenosis of the esophagus, or by pressure from enlargements outside the esophagus. Failure of eructation may also result from ruminal atony. The excess gas is present as a free gas cap on top of the ruminal contents. There is usually an increase in the frequency and strength of ruminal movements in the early stages followed by atony. Passage of a stomach tube or trocarization results in the release of large quantities of gas. If an esophageal obstruction is present it will be detected when the stomach tube is passed. If other methods fail to correct the condition rumenotomy should be conducted.

11. Lameness in Dairy Cattle

(Adapted from Risco and Melendez, 2011)

Learning outcome

- Identify risk factors, diagnose, manage and control lameness in dairy cattle.

Lameness constitutes any foot or leg condition of infectious or non-infectious origin that negatively impacts cow mobility, posture, and gait (Archer et al., 2010). The greatest incidence (90%) of lameness involves the foot, and of these, 90% involve the rear feet. In dairy cows, the main cause of lameness is claw lesions, which are either non-infectious (white line disease, sole ulcer, sole abscess, toe ulcer) or infectious (including digital dermatitis, interdigital dermatitis, and foot rot) (Van Nuffel et al., 2015).

Lameness is one of the most important diseases of dairy cattle because of its negative impact on animal welfare, milk production, reproductive efficiency, weight gain, increased risk of culling and the additional labor costs to manage these cows (Pavlenko et al., 2011). Lameness is one of the most economically important diseases of dairy cattle, at a cost of about $90 per cow (Sen, 2021).

Lameness is common among dairy cows and estimates of its incidence ranged from 13 to 53%. The affected cows show symptoms of pain and frustration, especially when performing normal locomotive behaviors.

11.1. Mobility scoring (locomotion or lameness scoring)

Active monitoring and prevention of lameness at a herd level through the use of herd health plans requires careful record-keeping of lameness levels on the farm. The process of mobility scoring involves an assessor observing cows from the side and rear while they walk past. The assessor uses a standard assessment framework to categorize the cows according to the severity of the signs observed (Bell, 2015). Numerous scoring methods are in use. In general, scoring methods use the signs of pain, which include walking at a slower speed, a shortened stride length, an arched back and diminished weight-bearing of affected limbs. A four-category mobility scoring system is presented in Fig. 5 (Dairy Australia Limited, 2019).

Mobility scoring is a useful starting point for any foot health audit or investigation. For ongoing evaluation, mobility scoring should occur at least four times a year to capture seasonal variation. For enhanced screening of lame cows, scoring at least fortnightly is recommended, although some herds with observant stockmen spotting cows between scoring sessions can manage with monthly intervals or without formal screening at all (Bell, 2015).

Mobility scoring generates valuable information about which cows to prioritize for treatment by the farm staff (mild lameness and first aid treatment), foot trimmer (routine trims generally) or the veterinary surgeon (lesions requiring extensive debridement or surgery under local anesthesia) (Bell, 2015).

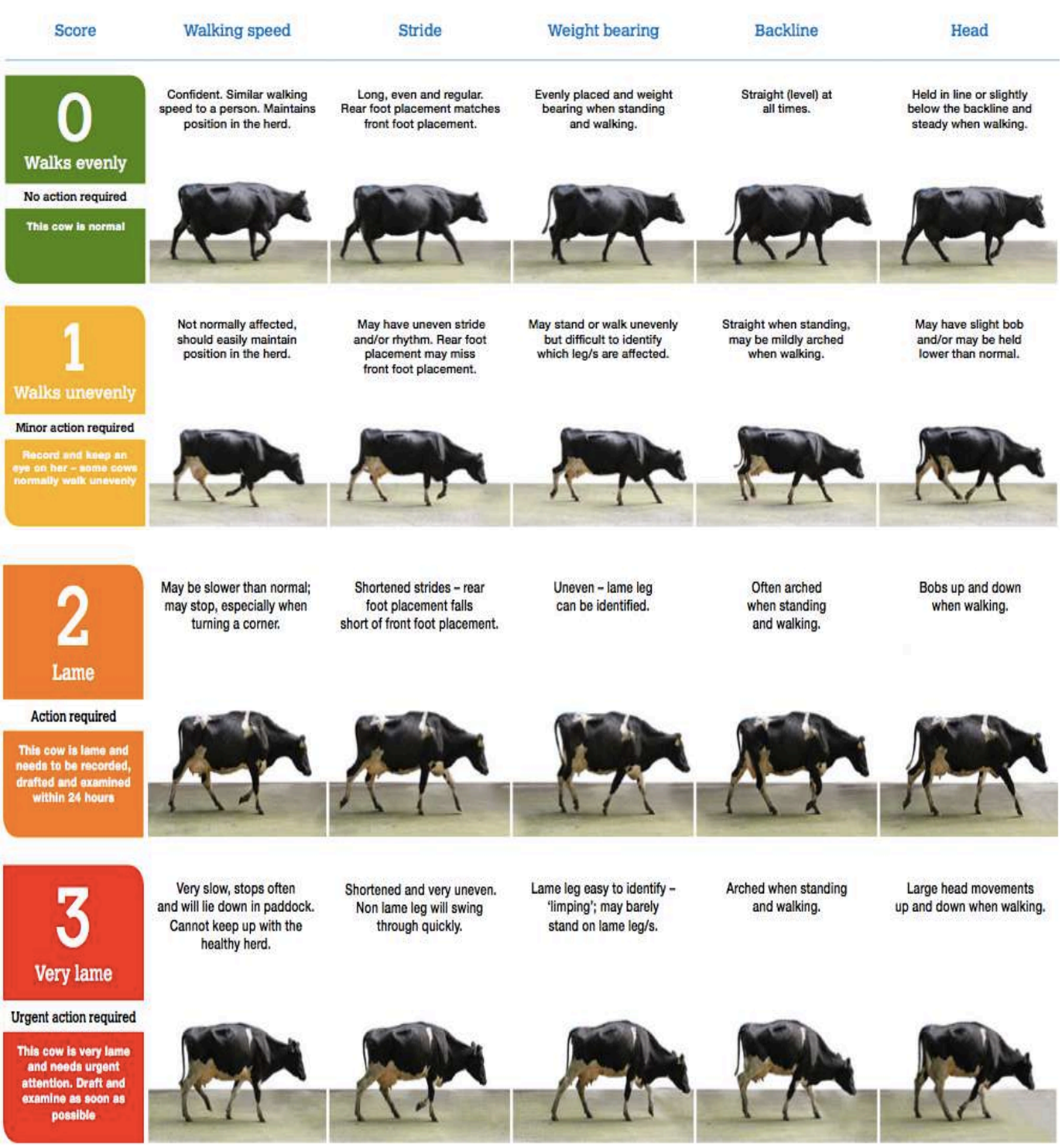


Figure 5. A four-category lameness scoring (Dairy Australia Limited, 2019)

11.2. Noninfectious Disorders of the Bovine Foot

The most common noninfectious causes of lameness affecting the bovine digit (Fig. 6 and 7) are ulcers, white line disease, and traumatic lesions of the sole. Some of these conditions are predisposed by metabolic disorders including rumen acidosis and laminitis along with other physiological factors (enzymes and hormones) that affect the integrity of the suspensory apparatus of the third phalanx, particularly during the transition period. Thickness of the digital cushion (DC), which is directly proportional to body condition of the cow is also an important factor affecting occurrence of lameness in cows. Laminitis is an important underlying cause of disorders affecting the digit in cattle.

Long periods of standing on hard surfaces can cause excessive pressures that crush and destroy horn-producing tissues under the sole. Inadequate time and facilities for rest increase the risk. Trauma from abrasive concrete, uneven or slippery walking surfaces, may predispose animals to lameness. Extremely wet conditions are associated with higher rates of lameness in dairy cows. Prolonged exposure to moisture causes the hooves to soften, making bruising, sole penetration and white line disease more common.

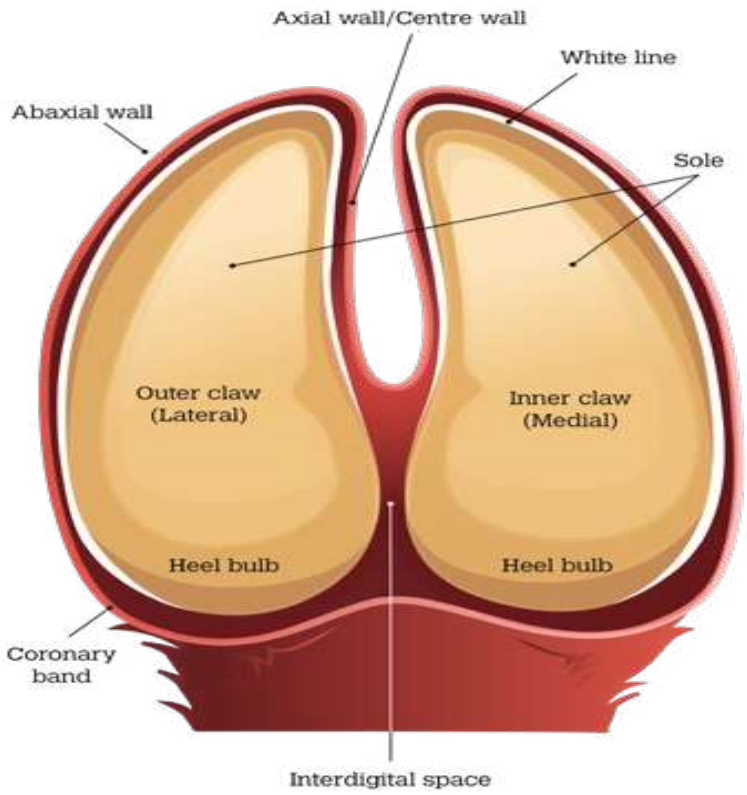


Figure 6. Parts of the hoof from the under view (Dairy Australia Limited, 2019)

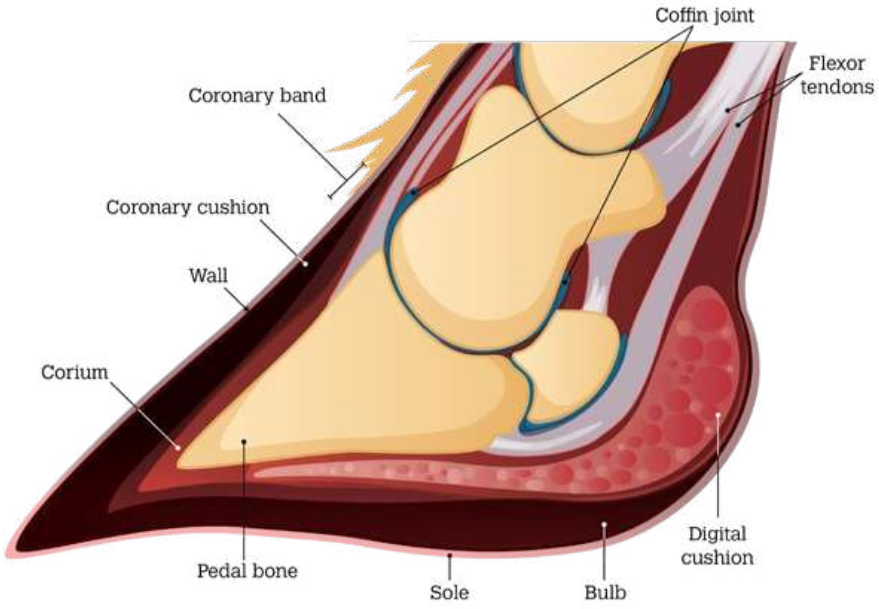


Figure 7. Sagittal section of claw (Dairy Australia Limited, 2019)

Ulcers of the Sole, and Heel

An ulcer is defined as a full-thickness defect or break in the epidermis that exposes the corium. Displacement of P3 results in compression of the solar and perioplic corium between P3 and the sole (Raven, 1989). Contusion and bruising of the corium at the toe, sole, and heel causes injury and dysfunction of the corium. In cases where displacement of P3 involves severe rotation of its apex, a toe ulcer may develop. If, on the other hand, sinking of P3 is such that the rear portion sinks furthest, compression of the solar and perioplic corium of the heel will lead to development of a sole ulcer ("Rusterholtz ulcer") at the heelsole junction.

One of the earliest indications of a developing sole ulcer is hemorrhage in the sole (Fig. 8), particularly when it occurs at the heel-sole junction. If the animal exhibits pain when pressure is applied to this area, it offers good evidence that the ulcer is in the clinical stage. With additional time and trauma associated with weight bearing, this lesion will likely progress to a full-thickness horn defect or ulcer. In the preclinical or early stages of development, despite the size of some hemorrhages, pressure as might be applied with a hoof tester causes little or no discomfort.

Mature ulcers are usually accompanied by lameness and even gentle pressure with a hoof tester over the ulcer site will elicit a positive pain response. Removal of superficial layers of horn may uncover an area of exposed corium that is extremely sensitive.

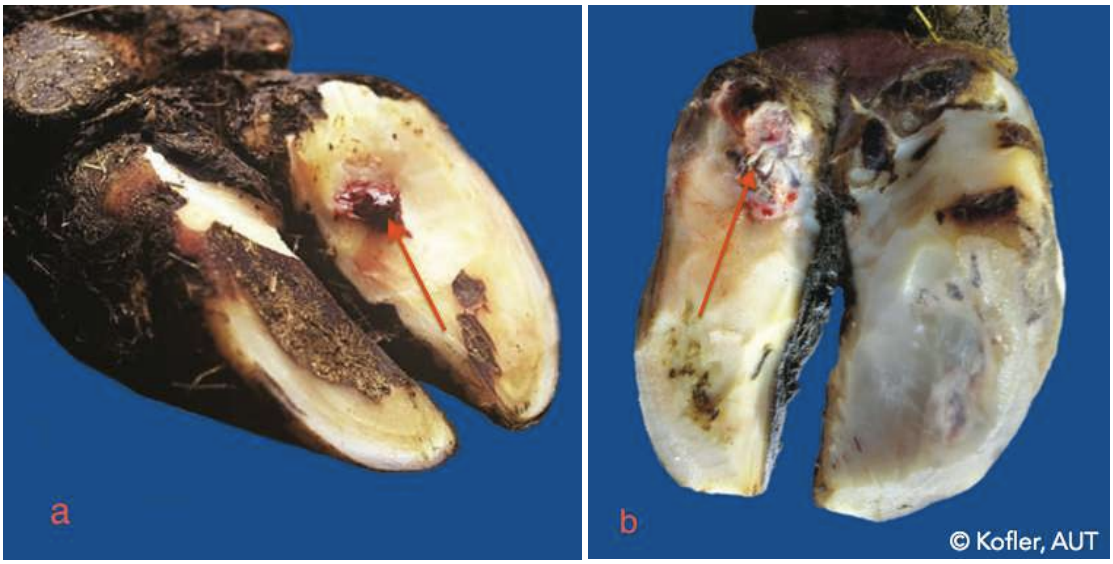


Figure 8. Sole ulcer (a) and Heel/Bulb ulcer (b) (Egger-Danner et al., 2020)

Treatment of ulcers

Treatment of ulcers in preclinical or early stage may be accomplished by lowering the heel on the affected claw so that it may have time with reduced weight bearing for rest and repair. Apart from lowering the heel of the affected claw also consider application of a foot block to the healthy claw to insure complete relief from weight bearing in severe cases (Raven, 1989). When identified early, these cases will usually recover quite rapidly (within 3–4 weeks).

Mature ulcers may be treated by thinning horn around the base of the ulcer and lowering this area relative to the weight-bearing surface of the healthy claw. It is also advisable to avoid leaving a crater or hole in the sole that will fill with organic matter. Recovery time for ulcers requires a minimum of 20–30 days, and as much as 50-60 days in severe cases. The objective should be to provide relief from weight bearing on the affected claw for at least 1 month by means of a foot block, and an additional 20–30 days by corrective trimming to adjust load bearing between the two claws.

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

White Line Disease

White line disease (WLD) is separation of the wall of the hoof from the sole, which can progress to impaction of dirt, grit and small stones into the white line and abscess formation (Fig. 6). A consequence of dirt and stones infiltrating into the space is that the infection may extend up the lamellae under hoof wall. This infection may result in an abscess which discharges at the coronary band (Dairy Australia Limited, 2019).

Major risk factors

- Excess twisting and turning of cows in the milking yard. Allowing impaction with small stones and dirt
- Wet and dirty underfoot conditions that soften claw horn
- Trauma from abrasive concrete yards
- Claw deformities
- Laminitis

The area of the white line most commonly affected is the abaxial heel-sole-wall junction of the lateral claw. The white line in this region is naturally predisposed to greater mechanical impact and wear during locomotion. Overgrowth and overloading of the outer claw tends to exacerbate load bearing and may increase white line disease problems.

Lesions within the white line normally begin as small cracks or spaces that become infiltrated with stones, dirt, or other types of organic matter. Abscess formation associated with white line disease creates severe lameness. These abscesses and the associated purulent material may accumulate in the sub-solar region of the sole and heel, or in many cases they will migrate caudally toward the heel or upward beneath the wall. In the worst-case scenario, these may rupture, forming a sinus tract at the skin horn junction (Fig. 9 b).

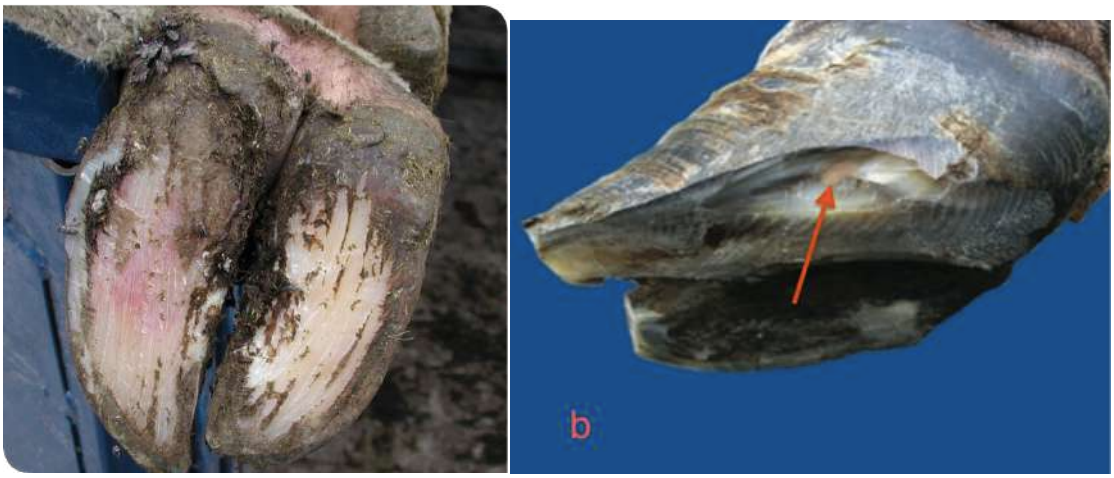


Figure 9. White line disease

Treatment of WLD lesions

Treatment of WLD lesions requires paring away horn in an abaxial direction over the lesion at roughly a 45-degree angle (Raven, 1989). Paring of the tract leading to the abscess is required until all necrotic, loose, and undermined horn is removed and drainage (in the case of abscess formation) is accomplished. Always attempt to minimize collateral damage of normal healthy tissues. There is no need for systemic antibiotic therapy unless the infection extends to deeper tissues of the foot as evidenced by swelling and severe lameness.

Bandaging of claw lesions is unnecessary. Bandaging should be reserved for conditions where it is desirable to provide temporary protection of corium, such as when large areas of the corium have been exposed during corrective trimming procedures.

Abscesses occurring secondary to white line disease or ulcers are extremely painful. Pain can be alleviated through the application of a claw block to the healthy claw of the affected foot. Elevation of the damaged claw suspends weight bearing, reduces discomfort, and promotes recovery.

Sole Abscess Associated with Traumatic Lesions (Subsolar Abscess)

Beyond ulcers and white line disease, one of the more important causes of lameness is sole abscess. Sole abscesses are normally a secondary condition that may occur subsequent to ulcers, white line disease, or traumatic lesions of the sole such as a foreign body (for example, nails, stones, teeth, and wire).

Treatment of this condition is as described above for white line disease lesions. Remove all loose and damaged horn around the solar lesion and apply a foot block to the healthy claw.

Toe Lesions in Cattle

Toe lesions in cattle are common and often difficult to manage. One reason for this is the tendency for toe lesions to extend into the third phalanx. Once an osteitis is established, the lesion often becomes chronic and very difficult to treat, short of the complete removal of all involved tissues. The common toe lesions in cattle include toe ulcer and toe necrosis (Fig. 10).

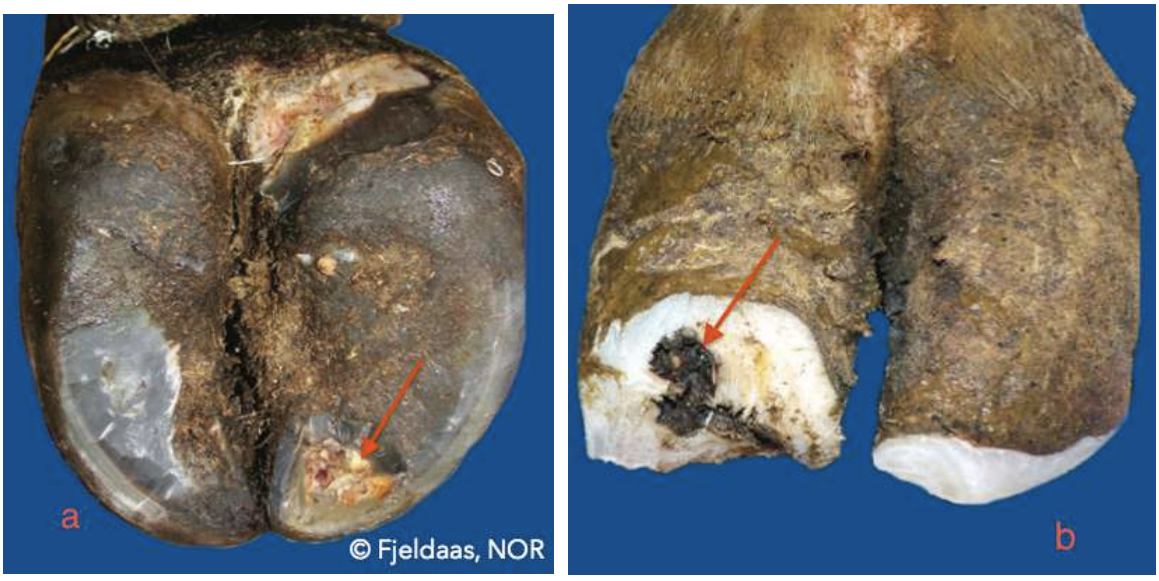


Figure 10. Toe ulcer (a) and Toe necrosis (b) (Egger-Danner et al., 2020)

Thin Soles

Normally the outer claw of the rear feet is most severely affected; however, in thin sole herds, the soles of all four feet usually show evidence of thinning: heels are shallow, and sole horn yields when finder pressure is applied (Egger-Danner et al., 2020).

Underlying Causes of Thin Soles

Sole horn growth rates are affected by age, diet, and length of the daily photoperiod. Wear rates are influenced by the abrasiveness of flooring surfaces, cow comfort, horn quality, and claw horn moisture. Therefore, the shape of the claw capsule is a product of growth and wear.

In housing systems where the rate of sole horn wear exceeds the rate of growth, excessive thinning of the sole is likely to occur. Claw horn hardness is influenced by nutrition, contact with manure slurry, and moisture content of claw horn. In some facilities, wear rates are exacerbated by abrasive flooring conditions that include sharp turns, sloped walkways, and new concrete.

The occurrence of thin soles is also influenced by conditions contributing to poor cow comfort such as overcrowding and insufficient bedding. When stall numbers are equivalent or less than the total number of cows in the barn, timid animals such as heifers may have less opportunity to rest. A common recommendation is that dairymen provide at least 10% more stalls than cows to permit greater choice and encourage lying time, especially around the time of transition.

Treatment and Management of Thin-Soled Cows

Treatment of thin-soled cows requires careful evaluation of the sole of all claws. The first objective in treatment is to determine if the more sound of the two claws being evaluated on each foot can support the weight on that limb if fitted with a foot block to relieve weight bearing on the thin-soled claw. If the answer is yes, then a block is fitted to the most sound of the two claws. On the other hand, if it is determined that neither claw can support the weight of the respective limb, then neither claw should be fitted with a block, and the animal should be housed in an area free of concrete or other hard and potentially abrasive surface.

In cases where the condition has progressed to the point of ulceration, subsolar abscess formation, or osteitis of the third phalanx, additional corrective trimming and debridement procedures are necessary. Cows that are thin-soled on all claws with one or more toe ulcers and abscesses in the sole horn and severely lame irrespective of age, pregnancy, or lactation status are considered to have a poor prognosis, and culling or euthanasia is recommended based on economic and welfare considerations.

One approach to alleviating problems presented by abrasive flooring surfaces is the strategic application of rubber belting or mats to holding areas, walkways, or along feed mangers.

Corkscrew Claw (Screw Claw)

Corkscrew claw is most commonly observed in the lateral claw of the rear leg in cattle. It is reported to be a heritable condition; however, other factors such as age, previous claw disease, and housing conditions are likely to influence its occurrence. It is characterized by rotation of the toe that displaces the sole, axial wall, and white line in an axial direction (Fig. 11). This curving of the claw and its internal structures results in weight bearing on the mid to caudal portion of the abaxial wall. The corkscrew claw is also normally larger and bears the majority of weight in the foot. As a consequence, the inner claw frequently atrophies from the lack of weight bearing. These abnormalities contribute to a greater potential for white line lesions in the abaxial region of the toe. Trimming generally requires great care as it is quite easy to expose the corium in this region during the trimming process. Overgrowth of the corkscrew claw is common and often predisposes to the development of sole ulcers as a consequence of elevated weight bearing. Trimming of the corkscrew claw at 3- to 4-month intervals helps prevent extreme overgrowth, overloading, and thus sole ulcers and conditions that may lead to toe lesions.

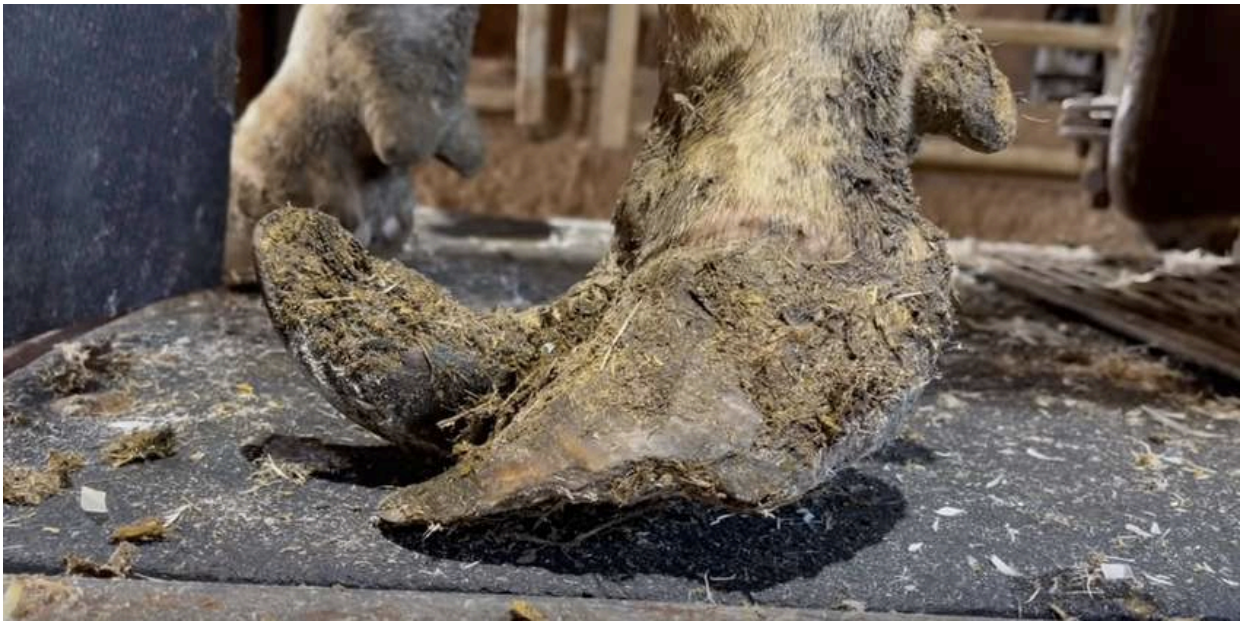


Figure 11. Corkscrew claw

Wall Cracks in Cattle

Cracks or fissures in the hoof wall are common in cattle. Those which run in a vertical direction (from the coronet to the weight-bearing surface) are referred to as vertical wall cracks or sand cracks (Fig. 12). Incidence rates are usually less than 1% in dairy cattle, but when lameness does occur, it may be difficult to treat or manage.

Cracks or fissures that run in a horizontal direction (i.e., parallel to the coronet) are referred to as horizontal wall cracks. These are common in both beef and dairy cattle, and when severe, may result in profound

lameness. Horizontal wall cracks in some cases, simply signal a “physiological change” that has resulted in a mild to moderate interruption of horn growth and formation in the basal cell layer of the coronary corium. In others, they are representative of conditions (often related to disease disorders) that have led to significant “physiological stress” and severe interruption of horn formation by the coronary corium. These severe disruptions in horn formation are exhibited as very distinct ridges and grooves that run in a horizontal direction on the hoof wall. They are often referred to as “hardship grooves” or “stress lines.” In the most extreme cases where the fissure is sufficiently deep to result in a full thickness defect of the wall, the lesion is often referred to as a “thimble.”

Treatment of horizontal wall cracks is generally unnecessary; however, when pinching of the corium occurs corrective procedures may be required. The objective is to stabilize the lose sections of the wall by removal of the loose portions. Application of a foot block on the opposite claw often provides relief (Shearer and van Amstel, 2011).

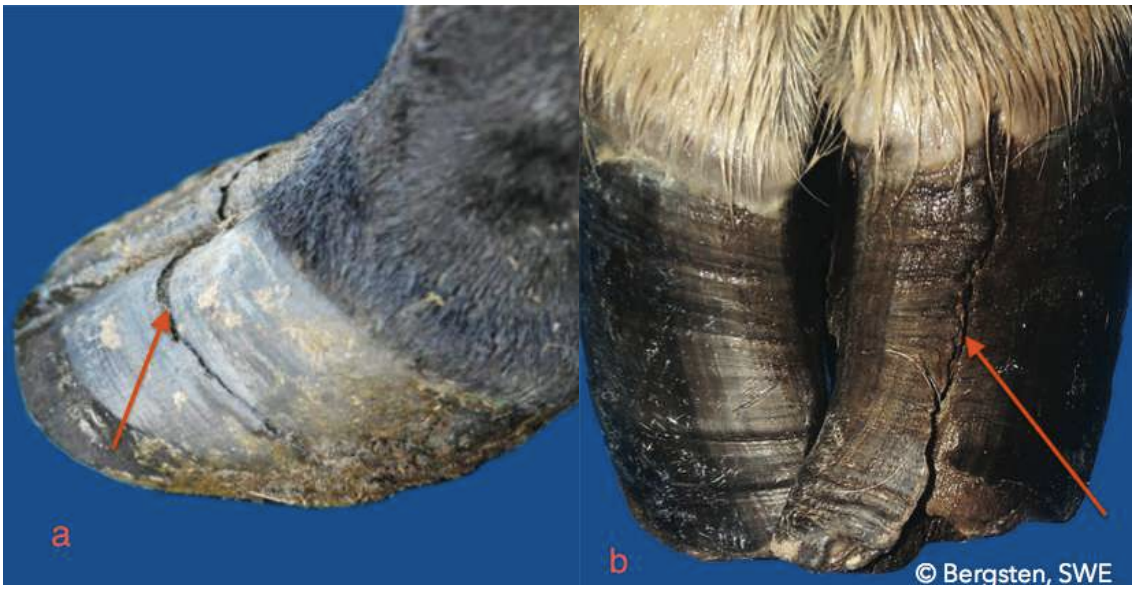


Figure 12. Horizontal (a) and Vertical (b) wall crack (Egger-Danner et al., 2020)

11.3. Infectious Disorders of the foot skin

These represent some of the most common and important causes of lameness in cattle; however, these diseases affect the “skin” of the interdigital space, heel bulbs, and interdigital cleft (on the back of the foot above the interdigital space).

Digital Dermatitis (DD) (Hairy Heel Warts)

The precise cause of DD remains to be determined, but most studies to date indicate that the organisms observed in most lesions are bacterial spirochetes belonging to the genus *Treponema* (Shearer and van Amstel, 2011).

The lesions of DD typically occur on the plantar aspect of the rear foot on the skin adjacent to the interdigital cleft, or at the skin-horn junction of the heel bulbs (Fig. 13). On occasion, lesions may be found adjacent to the dewclaws or bordering the dorsal interdigital cleft (particularly on front feet). Most lesions are circular or oval with clearly demarcated borders. Filiform papillae often extend from the surface of chronic lesions.

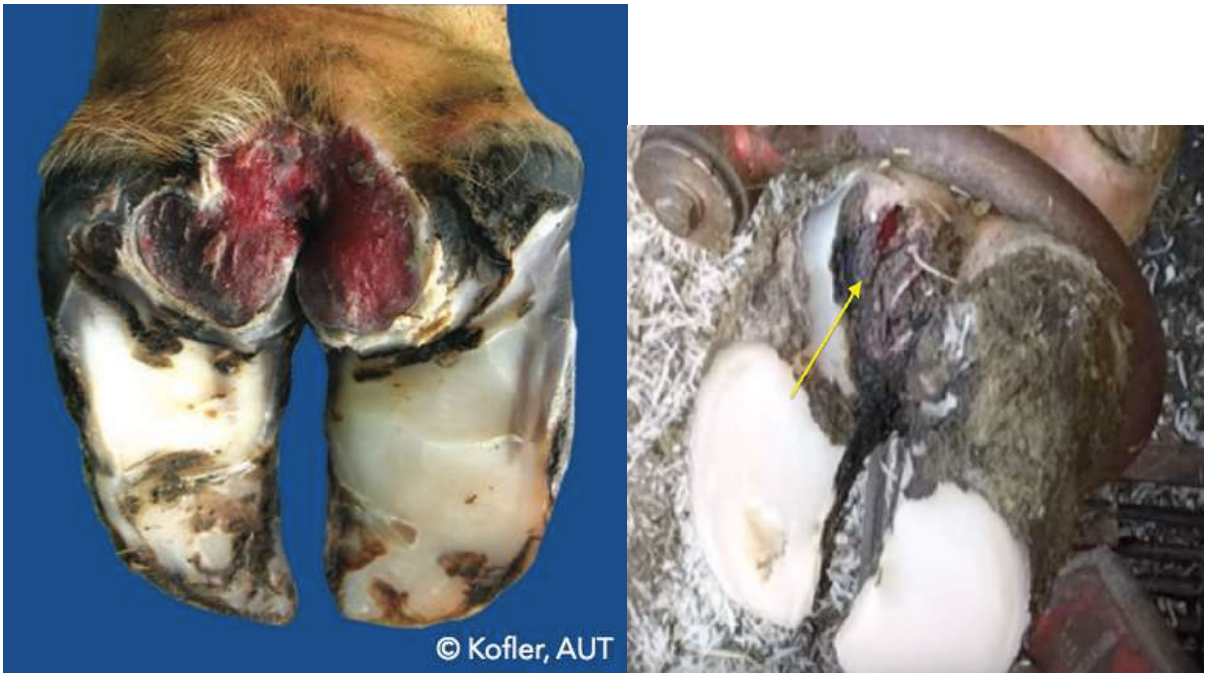


Figure 13. Digital dermatitis (Egger-Danner et al., 2020)

Even a mild disturbance of the inflamed tissue tends to result in extreme discomfort and mild to moderate bleeding. Therefore, cows will alter their posture and/or gait to avoid direct contact between lesions and the floor or other objects. These pain avoidance adaptations also lead to abnormal wear of the weight bearing surface of affected claws. Lesions associated with the plantar interdigital cleft usually cause the cow to shift weight bearing toward the toe. This results in increased wear at the toe, decreased wear in the heel, and an overall reduction in the weight-bearing surface of the affected claw (Shearer and van Amstel, 2011).

The prevalence of DD in herds is quite variable. Rates of 20% to more than 50% have been reported. The highest incidence of DD in cows is usually observed in early lactation.

The housing, environment, and management conditions most consistently identified as predisposing causes of DD include large herd size, wet and muddy corrals, and the purchase of replacement animals. Other risk

factors include housing on grooved concrete and use of a trimmer who trimmed feet at other farms. Housing and environmental hygiene are important factors in control of this disease.

Topical spray-on treatment with antibiotic and some nonantibiotic preparations have been shown to be effective when used in a scheme of daily treatment for a period of 8-10 days. Topical antibiotic treatment under a bandage is particularly effective. Footbaths containing various compounds including 3%–5% formalin, 5%-10% copper sulfate, 20% zinc sulfate, oxytetracycline 1-4 g/L, lincomycin 1-4 g/L, or lincomycin/spectinomycin 1-4 g/L have been recommended, though results vary widely.

Interdigital dermatitis (ID) (Slurry Heel/ Stinky foot)

ID is an acute or sometimes chronic inflammation of the interdigital skin, extending to the dermis. It is extremely common in dairy cows that are continuously exposed to wet manure slurry or muddy corral conditions (Raven, 1989). The disease is believed to be caused by a mixture of bacteria: *Fusobacterium necrophorum*, bacterial spirochetes, and possibly *Dichelobacter nodosus* (Shearer and van Amstel, 2011).

In the early stages, ID is characterized by superficial erosion of the interdigital skin that some are able to recognize by its distinctive foul odor. The interdigital lesion is usually painful to the touch and is followed by extension of the infection to the heel horn resulting in heel erosion, the most readily visible feature of this disease. Excessive hoof formation leads to overgrowth and overloading of the affected claws, which may in turn predispose cows to claw disease problems, particularly sole ulcers in confined conditions. The clinical diagnosis of ID is based on the presence of a thickened interdigital skin, pungent characteristic odor, pain to the touch, and the concurrent presence of heel horn erosion (Shearer and van Amstel, 2011). ID shares a number of clinical and histopathological similarities with DD. As a result, there are yet unresolved questions about whether these disorders are in fact one and the same.

Foot Rot (Interdigital Phlegmon)

Foot rot is an infectious disease of the interdigital skin characterized by the presence of an interdigital lesion, swelling, and moderate to severe lameness. Fever ranging from 39.4-40.6^{o}C is a consistent finding during the acute stages. It is believed that foot rot develops following injury or abrasion of the interdigital skin. This interdigital injury is secondarily infected by *Fusobacterium necrophorum* alone, or in combination with *Bacteriodes melaninogenicus*, organisms that encourage progression to a more severe and necrotic-type of lesion. Failure to institute treatment early in the course of the disease may lead to complications involving surrounding soft tissues (tendons, tendon sheaths, joint capsules, and bone), ultimately resulting in deep digital sepsis. At this stage, response to medical therapy is quite often unrewarding, thus limiting one's options to either surgery, or possibly euthanasia, in particularly severe cases (Shearer and van Amstel, 2011).

Foot rot is responsive to most antibiotics in common use for cattle. The key to achievement of a successful therapeutic outcome is dependent upon prompt recognition and early implementation of treatment procedures.

Systemic therapy plus topical treatment of the interdigital lesion have long been the preferred methods of treatment. Treatments of choice are Ceftiofur Sodium, Penicillin, and tetracyclines (extra-label in dairy cattle). Some prefer to simultaneously treat the interdigital lesion as well. Various antiseptic products may be used as topical treatments. Bandaging of the foot is unnecessary (Shearer and van Amstel, 2011).

12. Immunology and Vaccination of Cattle

(Adapted from Cortese, 2011)

Learning outcome

- Sound knowledge of the role of colostrum on calf immunity
- Understand factors considered in the choice and timing of vaccination
- Able to develop and recommend relevant vaccination programme for a herd.

Improper handling of the immune system precalving can lead to increased postcalving problems and decreased milk production and increased reproductive failures. Improper knowledge of the immune system can lead to increased calf problems that will lead to life-long decreases in milk production and increased health problems and culling rates.

In order to scientifically choose a vaccine or design a particular vaccination program, it is necessary to consider many variables. When designing a vaccination program, a good history is needed before the program can be built. This should include:

1. Presence and degree of challenge of the particular diseases on the herd.
2. Management practices on the facility that lend themselves to or hinder the implementation of vaccination programs.
3. At what times or ages are the disease problems occurring and are they associated with any stresses?
4. What is the status of the herd? Is it open or closed? Are the owners purchasing animals and at what age? Are the calves home raised or grown by others?
5. What is the breeding program? Are cleanup bulls used? Source of the bulls and age of the bulls at purchase.

Challenge

The level of disease challenge and degree of protection are in a continual state of fluctuation on a herd and in a particular animal. The level of protection is different in every vaccinated animal due to biological variability and day-to-day stresses the animal may be undergoing. The same is true with the amount of exposure to a pathogen. Overwhelming challenge can override the immunity and lead to disease even in well-vaccinated animals.

Timing of Disease

Many farms will have consistent times when certain diseases occur. The timing may give some insight into stresses that are occurring in management of the cattle. Correcting these stresses can have a positive impact on vaccination and lessen disease susceptibility. Furthermore, this type of history is helpful to determine the timing of vaccinations. Knowing when a problem has historically occurred will allow vaccinations to be scheduled as to when they will give maximum immune responses in preparation for anticipated challenges. As a general rule, vaccines should precede the anticipated problem by at least 2 weeks.

12.1. Colostrum and Calf Immunity

Since the placenta is of the epitheliochorial type in food animal species (cattle, pigs, sheep), there is no transplacental transfer of antibodies or white blood cells. Therefore, no discussion on bovine neonatal immunology is complete without a discussion on an important component of the newborn calf's defense mechanism, colostrum.

Colostrum is the most important example of passive immunity. Defined as the "first" secretions from the mammary gland present after birth, colostrum has many known and unknown properties and components. The information on both the short- and long-term impacts of colostrum in calves continues to grow. Good passive transfer impact morbidity and mortality in the young calf, and also has a positive impact on long-term health and production. Constituents of colostrum include concentrated levels of antibodies and many of the immune cells (B cells, CD cells, macrophages, and neutrophils), which are fully functional after absorption by the calf. Additional components of the immune system such as interferon are transferred via colostrum. The primary colostral antibody in most domestic species is immunoglobulin (Ig) class G; in ruminants this is further defined as IgG1. The function of the various cells found in colostrum is still undergoing much research. The cells are known to enhance defense mechanisms in the newborn animal in the following ways: transfer of cell-mediated immunity, local bactericidal and phagocytic activity in the digestive tract, and increased lymphocyte activity. Finally, calves deprived of maternal colostral leukocytes upregulate receptors associated with physiological stress.

These cells contained in colostrum are destroyed by freezing and naturally disappear from the calf between 3 and 5 weeks of age.

Colostrum Absorption

When calves are born, the epithelial cells that line the digestive tract allow absorption of colostral proteins via pinocytosis. As soon as the digestive tract is stimulated by ingestion of any material, this population of cells begins to change to those that no longer permit absorption. By 6 h after birth, only about 50% of the absorptive capacity remains, by 8 h 33%, and by 24 h no absorption is typically seen. So colostrum transfer is a function of quality and quantity of the colostrum as well as the timing of colostral administration. In the

Holstein breed, the first feeding should be a minimum of 3L and preferably 3.8L of high quality, clean colostrum. In spite of all of the information regarding the importance of colostrum administration to the calf, some degree of failure of passive transfer is common. Colostral supplements are available as well as products for oral or systemic administration, which contain specific antibodies or general IgG concentrations. There is tremendous variability in the IgG concentration of colostral supplements. Although mixed results have been observed pertaining to the efficacy of these products, in colostrum-deprived calves, they may have a significant value in decreasing mortality and/or severity of disease.

Vaccination to Improve Colostral Quality

It has long been thought that vaccines administered to a cow before calving will increase colostral antibodies against specific antigens. This has been best demonstrated with vaccines against neonatal diarrhea pathogens that are administered to cows. These vaccines are designed to increase the colostral antibody concentration against specific organisms that cause diarrhea in calves such as *E. coli*, rotavirus, and coronavirus.

Maternal Antibody Interference Revisited

One of the commonly held beliefs in neonatal immunology is that the presence of maternal antibody will block the immune responses associated with vaccination. This has been based on vaccinating animals followed by evaluating subsequent levels of antibody titers. It is clear from many studies that if animals are vaccinated in the presence of high levels of maternal antibody to that antigen, they may not display increased antibody titers following vaccination. However, recent studies have shown both the formation of B cell memory responses as well as cell-mediated immune responses in the face of maternal antibody when attenuated vaccines were used. It is clear from these studies that maternal antibody interference of vaccines is not as absolute as once thought. The immune status of the animal, particularly against that antigen, the specific antigen, and presentation of that antigen should be considered when trying to design vaccination programs when maternal antibody may be present. In summary, vaccination against diseases that have a primary cell-mediated protective mechanism may be more likely to stimulate an immune response in the face of maternal antibody than those of which humoral immunity is the primary protective mechanism.

12.2. Impact of Stress on Immunity

Stress impacts the calf's immune system as it does in older animals. There are several factors that can affect the immune system that are unique to the neonatal animal. The calving process has a dramatic impact on the newborn's immune system due to corticosteroid release. Furthermore, the newborn has an increased number of suppressor T cells. These factors along with others dramatically decrease systemic immune responses for the first week of life. After birth, there is a decrease in immune responses until day 3 when they are at their lowest levels. By day 5, these responses are back to level of immune responses seen on the day of birth. Systemically administered vaccinations during this time should be avoided due to these decreased responses.

Vaccination immediately after birth may even have undesired effects. Furthermore, other stresses should be avoided in the young calf to try and maintain immune system integrity in the immunologically frail newborn. Procedures such as castration, dehorning, weaning, and movement need to be considered as stresses that have the potential to decrease immune system function temporarily. In cows, decreased immune function can be measured beginning 4 weeks prior to calving and does not rebound to normal levels until 5 weeks postcalving. This immune suppression may also delay or impair response to vaccines: Therefore, postparturient or poststress vaccination should be delayed until reasonable immune responses can be expected.

12.3. Choosing Vaccines

Modified Live Versus Inactivated Vaccines

Each company's development and manufacture of cattle vaccines is different; thus, the composition of the vaccine will vary dramatically among different manufacturers. Outlines of production are proprietary for each manufacturer; however, some information can be found in technical and marketing pieces. For example, some viral vaccines are grown on bovine-derived kidney cell lines whereas others are grown on porcine-derived kidney cells. Some vaccines are grown on only calf serum and some are grown on both calf and fetal calf serum. Differences in passages may be found as well. The variability is seen in the following areas:

- Strain(s) chosen for the vaccine
- Number of passages chosen in the growth
- Growth medium
- Number of viral or bacterial particles in the vaccine particles

There are basically three different technologies available today in cattle viral and bacterial vaccines.

1. Modified live (attenuated) vaccines contain living bacterial or viral organisms. They are usually collected from a field disease and then grown in abnormal host cells (viral) or media (bacterial) to change or attenuate the pathogen. Each time the pathogen is grown through a replication it is called a passage, and it is administered back to the animal to see if it is still virulent. After several passages, the pathogen will begin to lose virulence factors since it cannot cause "disease" in these unnatural host cells. Once the pathogen can no longer cause "disease" in the target species, it is then tested to see if it can confer protection. The final vaccine is usually passed a number of times beyond the passage where virulence is no longer seen. This decreases the risk of reversion to a virulent pathogen. These vaccines usually require good quality control to decrease the risk of a contaminant entering the vaccine.

2. Inactivated (killed) vaccines are easier to develop since virulence after growth is not a problem. The same pathogen is isolated from a disease outbreak. The pathogen is grown and then chemically or physically killed.

The inactivation is usually achieved by either adding a chemical to the pathogens or using ultraviolet rays. The major concern with inactivation is the potential loss of important epitopes. An adjuvant is normally added to inactivated vaccines to heighten the immune response. The vaccine is then tested for efficacy.

3. Genetically engineered vaccines have been altered genetically. This may be induced by several different methods, but the ensuing bacterium or virus has different properties that may alter virulence or growth characteristics. Most of these vaccines are modified live mutants (temperature-sensitive viral vaccines; streptomycin- dependent Pasteurellas), but inactivated marker vaccines are also genetically engineered. These vaccines have been engineered to delete a gene and cause an immune response deficient in antibodies to a certain epitope, allowing diagnostics to differentiate between vaccine and natural exposure responses (e.g., gene-deleted infectious bovine rhinotracheitis vaccines [IBRVs]).

12.4. Designing a Vaccination Program

Vaccination programs in a cowherd need to be custom-designed for the particular needs of the herd. Vaccination programs in the replacement stock have two specific goals that need to be met. The first is to protect the calf against any pathogens that are prevalent in the calves. The second is to prepare the calf for entry into the adult herd with a good foundation of protection from which to build herd immunity. Vaccines that utilize the mucosal immune system have been tested and licensed for use in the young calves, including the newborn. These vaccines include modified live, intranasal IBR/parainfluenza type 3 (PI3) vaccines, modified live, oral rotavirus/coronavirus vaccine, and new intranasal vaccines containing either bovine viral diarrhea virus (BVDV) types 1 and 2, bovine herpesvirus (BHV)-1, PI3, and bovine respiratory syncytial virus (BRSV) or BRSV in combination with PI3 and adenovirus. Exact timing of early vaccination will vary somewhat depending on antigen and presentation. There are equivocal results regarding the immunological response to early vaccination in calves. In general, vaccination in the young calf should precede anticipated or historical times of disease by at least 10 days, allowing the immune system to respond before exposure. If a booster dose is required, then the booster should be given at least 10 days before the expected disease occurrence.

Vaccination programs are tailor-made to each herd; however, there are some basic vaccination recommendations for cattle herds. The cornerstone of the herd program is based first on protection against high-prevalence diseases that can have catastrophic impacts on the herd when infections occur. In many parts of the world, the minimum vaccination program should be built around the four major viral diseases: BVDV (types 1 and 2), BHV-1, and BRSV. Many would also include vaccination against the five primary *Leptospira* serovars of cattle due to the potential for high abortion rates, as well as the major *Clostridia* diseases, core endotoxin vaccines, and *Brucella*. The rest of the vaccines mentioned above, except blackleg, are not currently available on the Ethiopian market. The common vaccines for cattle in use in Ethiopia are anthrax,

blackleg, lumpy skin disease, bovine pasteurellosis, foot and mouth disease and contagious bovine pleuropenumonia.

Booster Importance

It is important to follow the label directions for administering vaccines. Most inactivated vaccines require a booster before protection is complete. The first time an inactivated vaccine is administered, the primary response occurs. This is fairly short-lived, not very strong, and is predominantly comprised of IgM. The response seen after a booster vaccination is called the secondary response or anamnestic response. This is much stronger, of longer duration, and is primarily composed of IgG. If the booster is given too early, the anamnestic response does not occur; and if too much time elapses before the booster is given, it acts as an initial dose, not as a booster. With most modified live vaccines (with the exception of most BRSV vaccines), the primary vaccination also stimulates the secondary response without needing a booster since the virus or bacteria is replicating in the animal.

Adverse Reactions

Adverse reactions are a potential risk with any vaccination. However, dairy cattle appear to have a higher risk of postvaccination reactions than other cattle. These reactions fall into three primary types.

- IgE and the release of granules from basophils and mast cells mediate immediate hypersensitivity. This reaction is seen within minutes of vaccination and often begins with shaking or sweating. The majority of these animals will respond to epinephrine.
- Delayed hypersensitivity is mediated by an antibody–antigen complex attaching to complement and the ensuing activation of the complement cascade. The resultant reaction may occur locally or systemically. The reaction may be delayed as the complexes form and the cascade begins and subsequent by-products begin to exert their effects. The signs are similar to immediate hypersensitivity and treatment is epinephrine.
- One of the more common reactions seen in dairy cattle has been associated with the endotoxin and other bacterial components found in most gram-negative vaccines. This is seen primarily in Holsteins due to some genetic predisposition. The signs seen vary depending on the individual's sensitivity to gram-negative bacterial components. As a general rule, no more than two gram-negative vaccines should be administered on the same day to dairy cattle. These adverse reactions include: anorexia and transient decreases in milk production, early embryonic deaths, abortions and gram negative bacterial (endotoxic) shock requiring fluxinin or ketoprofen, steroids, antihistamines, and fluids.

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