CHAPTER 1

Diseases of Neonates and Calves

INTRODUCTION

Success in calf rearing requires good husbandry for steady health maintenance. Many risk factors interfere with the health status of the neonate and the calf in the first weeks of life. Imposing factors include compromised immune status, poor sanitation and ventilation, inadequate nutrition, overcrowding, lack of vaccination and wet and cold weather conditions.The goal of calf rearing should be the creation and maintenance of a protective environment.

Two organ systems of the growing calf are particularly susceptible to disease: the respiratory and the alimentary systems. Most topics in this chapter will deal with infectious agents responsible for the entities of calf pneumonia and calf scours. Additional topics include examples of more common congenital anomalies, nutritional and metabolic enzymatic deficits, as well as two iatrogenic-induced disorders: bovine neonatal pancytopenia (BNP) and floppy ear syndrome.

In this chapter, diseases and disorders of dairy and beef calves are considered arbitrarily up to the age of 3–4 months.

Fig. 1.1. Ox. Conjoined neonate twins. Diprosopus. Craniofacial duplication is a rare form of a conjoined monozygotic twin. Duplication consists of two heads (bicephalic or dicephalic, or polycephalic) and necks, and includes the oral and respiratory anatomic structures associated with these two heads (double tongue, esophagus, trachea). (Courtesy of Department of Pathology, WCVM, University of Saskatchewan, Saskatoon, Canada.)

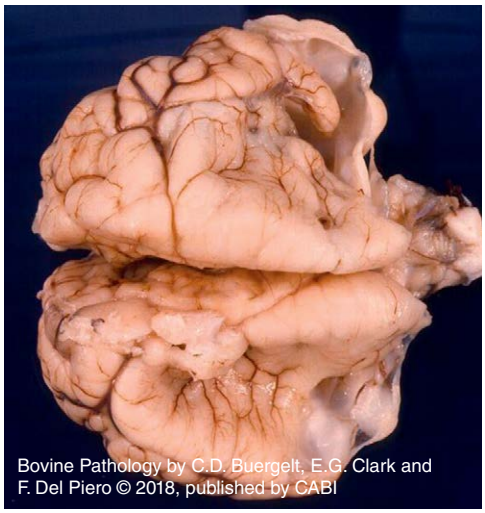

Fig. 1.3. Ox. Head. Hydranencephaly and cerebellar hypoplasia. There is evidence of complete loss of the cerebral parenchyma. The cerebral tissue is replaced by fluid accumulating (drained) within a sac covered by intact leptomeninges. The cerebellum is significantly reduced in size. Transplacental teratogenic viruses may be involved in the pathogenesis.

1.1 CONGENITAL ANOMALIES

Introduction. Many of the congenital anomalies occur spontaneously; some of them are the result of genetic mutants and chromosomal abnormalities, and some are the result of environmental factors such as teratogenic toxic plants or transplacentally transmitted teratogenic viruses. Calves with congenital defects are frequently stillborn. This segment presents examples of the more common abnormalities; a few additional defects are listed with the respective organ system chapters in which they occur (ocular, skeletal, reproductive, and dermal).

Diprosopus

Clinical complication: Dystocia

Hydrocephalus

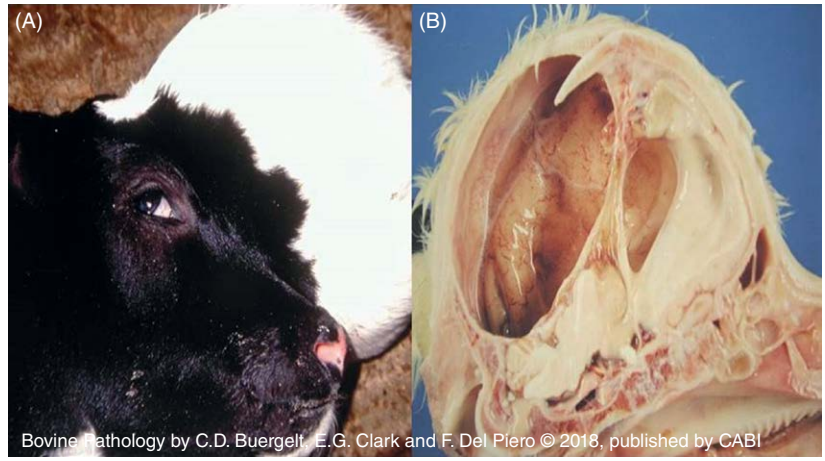

Fig. 1.2. Ox. Head. Hydrocephalus. Inherited forms are found in Hereford, Charolais and Holstein breeds. Grossly, a marked doming (A) of the forehead is visible. (B) A midsagittal section reveals a loss of cerebral tissue and marked distension of the ventricles by excessive CNS fluid (drained in the image) contained by leptomeninges. Transplacental teratogenic viruses such as bluetongue, bovine viral diarrhea virus, Akabane and other bunyaviruses have to be considered as a cause of the condition. Also, vitamin A deficiency may be a cause.

Clinical sign. Dystocia.
Differential diagnoses. Exencephaly, meningocele.

Hydranencephaly

Aplasia/hypoplasia

Clinical signs. Incoordination, ataxia, blindness.
Differential diagnosis. Genetic induction in Angus and Scottish Highland calves.

Spina bifida

Cranium bifidum is defined as a neural tube defect associated with the cranium.
Clinical signs. Hind-leg paresis, sometimes with anal and tail malfunction and/or arthrogryposis.
Differential diagnoses. Trauma, epitheliogenesis imperfecta.

Cleft palate (Palatoschisis)

Clinical signs. Nasal discharge, regurgitation of milk.

Segmental aplasia

The hypothesis that this defect is iatrogenically introduced during early pregnancy (40–42 days) via rectal palpation has been disproved.
Clinical signs. Abdominal distension, mucinous defecation.
Differential diagnoses. Jejunal volvulus, intussusception, atresia ani.

Fig. 1.7. Ox. Colon. Segmental aplasia. Blindly ending segments of the intestinal tract are indicated by asterisks. The proximal segment is markedly distended by digesta, while the distal segment is small and contains only mucus. The jejunum is another site for segmental aplasia in calves.

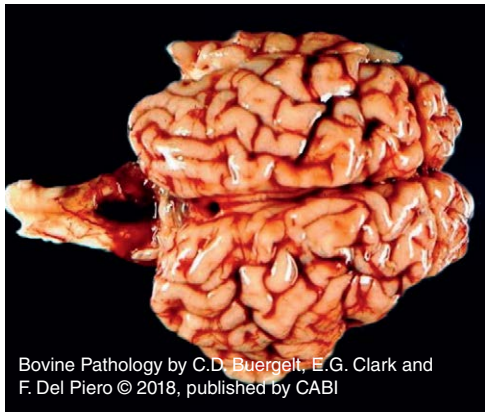

Fig. 1.4. Ox. Cerebellum. Aplasia/hypoplasia. Caused by *in-utero* infection of the dam at days 100–200 of gestation by the bovine viral diarrhea virus, all parts of the cerebellum or portions of the vermis and lateral lobes can be involved. The transmitted virus selectively causes cytolysis of neuroblast precursors interfering with differentiation of the permanent cerebellar cell population.

Fig. 1.5. Ox. Spinal cord. Spina bifida. Myelodysplasia (also known as spinal dysraphism) associated with spina bifida is an example of a neurotubal defect and usually occurs in Holstein neonates. Protrusions (arrows) of the spinal cord and meninges are visible at the site of the lumbosacral skin due to a defect of the dorsal spine. The spinal cord directly connects to the skin. (Courtesy of Dr J.M. King and the Section of Anatomic Pathology, Cornell University, USA.)

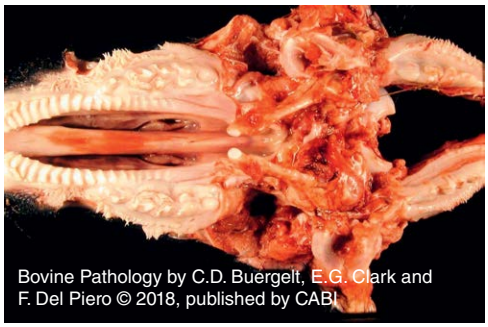

Fig. 1.6. Ox. Palate. Palatoschisis. A cleft affecting soft and hard palates exposes nasal turbinates. Aspiration pneumonia is a fatal complication.

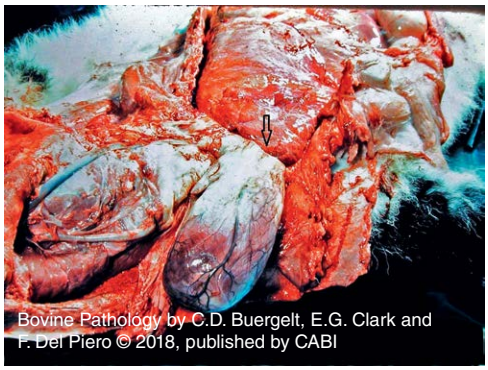

Fig. 1.8. Ox. Ventral neck. Ectopic heart (ectopia cordis). The exposed heart is located within the subcutis of the ventral neck (arrow). It is connected to the major vessels located in the thorax. Other ectopic cardiac locations are the subcutis of the sternum and the inside of the cranial abdomen. The subcutaneous location is often prone to trauma, leading to cardiac tamponade.

Ectopic heart

Clinical sign. Dermal pulsation.
Differential diagnosis. Venous congestion secondary to chronic heart failure.

Ventricular septal defect

Clinical signs. Lack of exercise, depression, respiratory distress.
Differential diagnoses. Tetralogy of Fallot, transposition of greater vessels.

Polycystic kidney

Clinical signs. Anuria, dysuria, or none.
Differential diagnosis. Obstruction of lower urinary system, e.g. hydronephrosis.

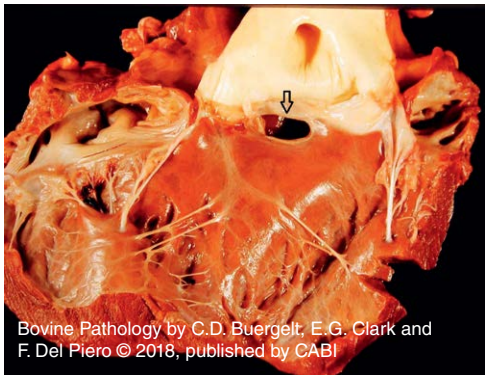

Fig. 1.9. Ox. Heart. Ventricular septal defect (VSD). It is the most common cardiovascular anomaly in ruminants. A cleft (arrow) is present in the membranous part of the ventricle (high VSD). Depending on the size of the defect and on the time of survival, a reversal of the original left-to-right shunt to right-to-left may lead to passive congestion, with cyanosis.

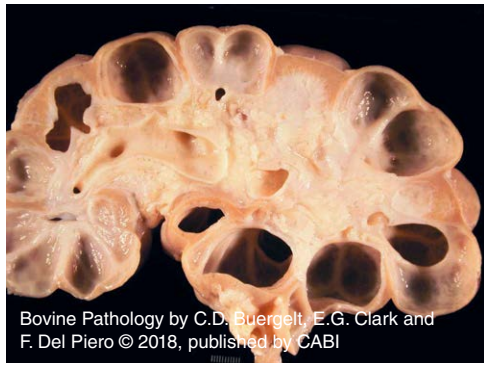

Fig. 1.10. Ox. Kidney. Polycystic kidney. A transverse section shows multiple cysts in the cortex, but also in the medulla, affecting more than 50% of the kidney parenchyma. The condition, when congenital, is the result of a failure of tubulogenesis during renal embryogenesis. Acquired hydronephrosis also has to be considered.

Hypospadia

Clinical signs. Abnormal urination and urine staining.

Fig. 1.11. Ox. Perineum. Hypospadia. Seen in neonatal male calves, it is the result of an incomplete ventral closure of the urethral fold and abnormal perianal or scrotal location of the urethra.

Omphalocele

Clinical signs. None.
Differential diagnosis. Amorphous globosus.

Fig. 1.12. Ox. Stillborn. Omphalocele (umbilical hernia). (A) Connected to the umbilical cord is a blindly ending attachment (arrow). (B).When opened, it shows a tubular organ filled with green, pasty material, indicating meconium. (Courtesy of Dr M. Drost, Drost Project, University of Florida, USA.)

Pulmonary dysplasia

Clinical signs. Respiratory shortness.
Differential diagnosis. Pulmonary emphysema and edema.

There are many additional abnormalities affecting various organ systems and sometimes occurring as multiple defects, including developmental duplications. Genetic, environmental, nutritional, plants, and infectious agents should be taken into consideration in the understanding of the etiology (see list of teratogenic viruses and plants).

List of Teratogenic Viruses
- Bluetongue virus (Orbivirus)
- Bovine viral diarrhea virus (BVDV) (Pestivirus)
- Akabane virus (Bunyavirus)
- Schmallenberg virus (Bunyavirus)

List of Teratogenic Plants
- Lupine (*Lupinus caudatus*)
- Locoweed (*Astragalus* spp./*Oxytropis* spp.)
- Poison hemlock (*Conium maculatum*)

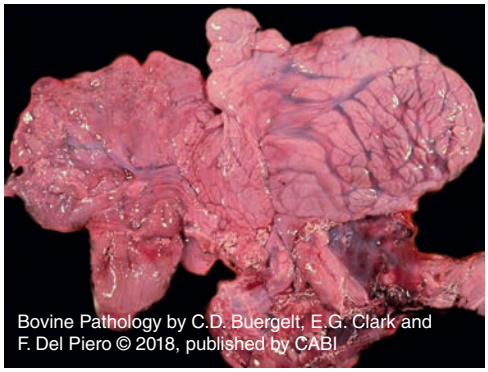

Fig. 1.13. Ox. Lung. Pulmonary dysplasia (hamartoma). A pseudolobulated. bulbous, enlarged gray lung is present on the right. The part on the left represents fetal lung. The dysplasia is histologically characterized by disorganized airway structures missing an alveolar component. This lesion has been hypothesized to be associated with an *in-utero* infection with the bovine viral diarrhea virus.

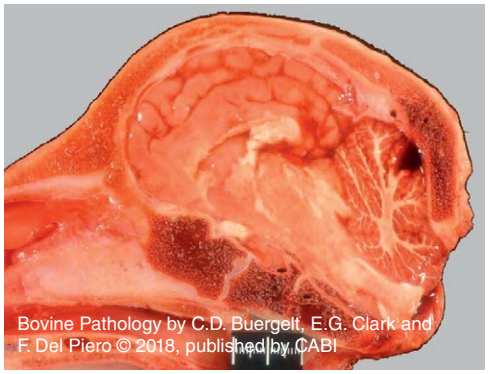

Fig. 1.14. Ox. Cerebrum, cerebellum. Edema. Coning. The cerebrum is markedly edematous. The edematous cerebellum is extremely compressed within the calvaria. It is partially located within the foramen magnum.

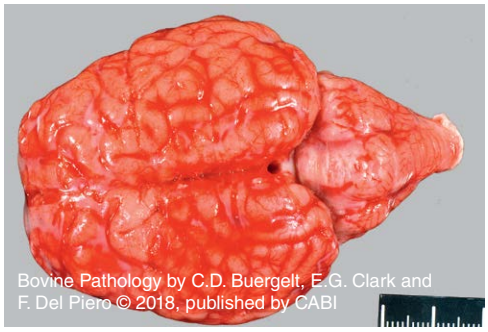

Fig. 1.15. Ox. Cerebrum, cerebellum. Acute meningitis. Meninges are markedly reddened. There is evidence of cerebellar coning.

Fig. 1.16. Ox. Cerebellum and brainstem. Purulent meningitis. The brainstem, and to a lesser degree the cerebellar surfaces, are covered by pus. Gram-negative bacteria are frequently involved, including *Escherichia coli* and *Salmonella* spp.

1.2 NERVOUS SYSTEM

1.2.1 Edema and inflammation

1.2.1.1 Cerebellar herniation (coning)

Introduction. If intracranial pressure increases, such as in edema, acute inflammation, hematoma or expending neoplasia, the cerebellar vermis herniates through the foramen magnum, resulting in hemorrhage and sometimes pressure necrosis.
Clinical signs. Ataxia, pain.
Differential diagnosis. Hypovitaminosis A.

1.2.1.2 Meningitis

Introduction. Commonly associated with septicemia, and usually bacterial in origin, it causes behavioral and postural changes in the calf.
Clinical signs. Incoordination, dullness, head pressing, recumbency.
Differential diagnoses. Viral encephalitis, otitis media.

1.2.2 Neoplasia

Introduction. A rare embryonal tumor involving the cerebellum of calves is defined as medulloblastoma.
Clinical signs. Ataxia, opisthotonos.
Differential diagnoses. Cerebellar, abscess.

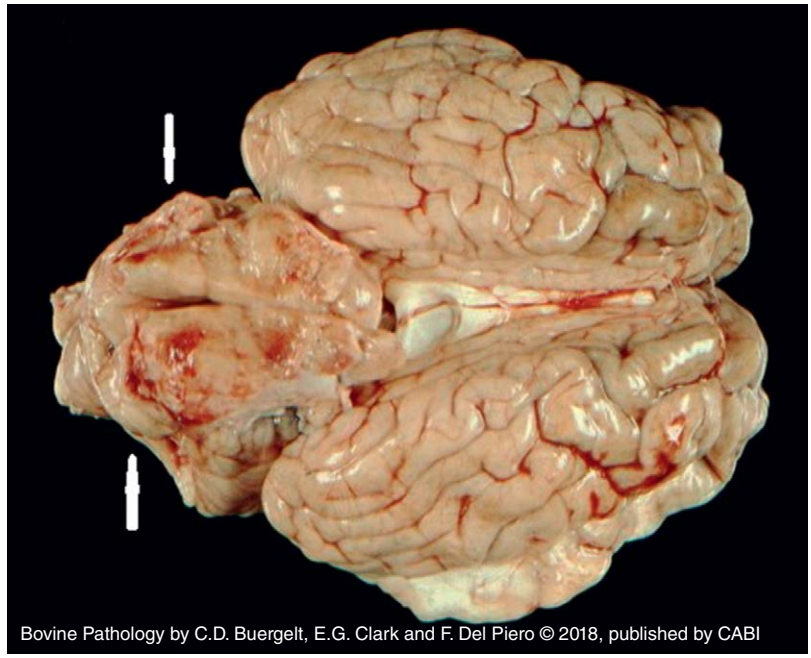

Fig. 1.17. Ox. Cerebellum. Medulloblastoma. A circumscribed gray growth with hemorrhage covers the midline of the vermis (arrows).

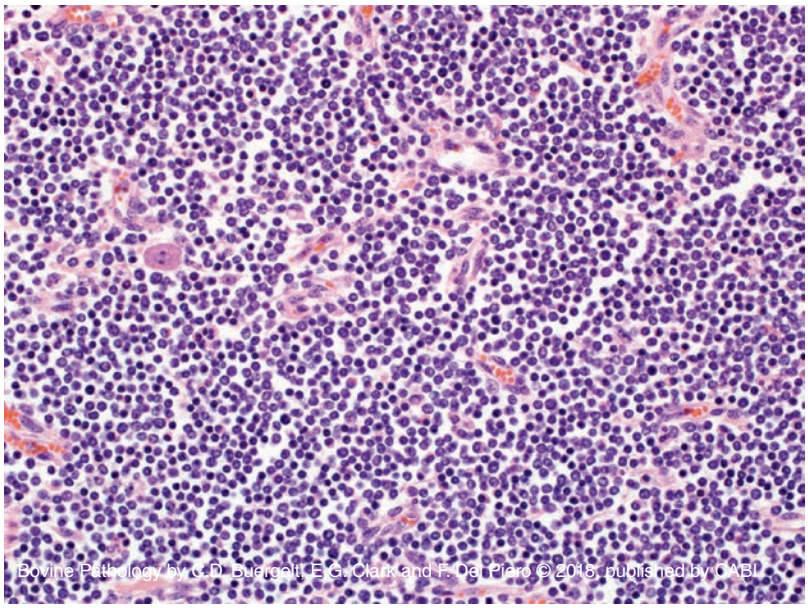

Fig. 1.18. Ox. Cerebellum. Medulloblastoma. The microscopic appearance is that of a densely cellular, basophilic growth with bundles of ovoid and elongated small tumor cells (Hematoxylin and eosin (H&E)).

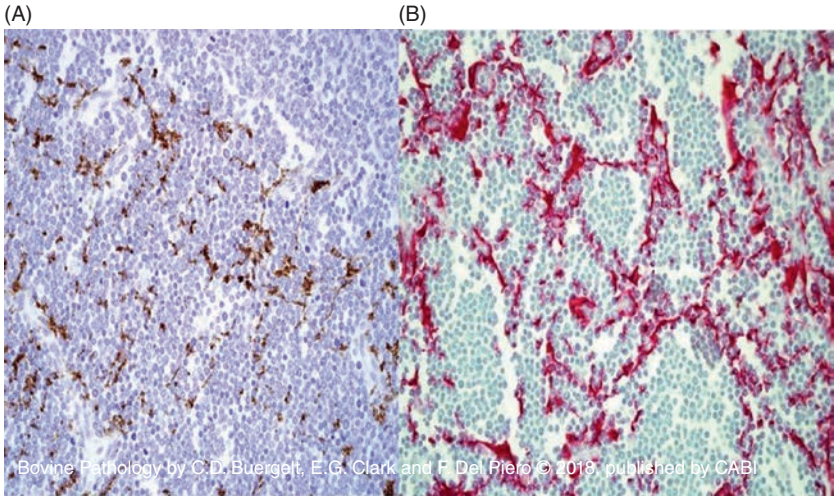

Fig. 1.19. Ox. Cerebellum. Medulloblastoma. Immunoreactivity is positive for fibrillar-oriented synaptophysin (A) and glial fibrillary acid protein (GFAP (B). Other tumor markers are neuron-specific enolase (NSE) and S-100. (Indirect immunohistochemistry (IHC)).

1.2.3 Inherited metabolic disorders

Introduction. Being the result of inherited enzyme deficiencies, the conditions are rare in cattle.

1.2.3.1 Bovine maple syrup urine disease

The disease occurs as an autosomal recessive trait in neonates and calves. It is the result of a branched-chain ketoacid dehydrogenase (BCKAD) complex deficiency. The histologic changes are that of spongiform encephalopathy with spheroids, gitter cells and mild astrogliosis.
Clinical signs. Dullness, recumbency, opisthotonos.
Differential diagnosis. Polioencephalomalacia.

1.2.3.2 Bovine citrullinemia

The progressive central nervous disease is caused by an autosomally inherited dysfunction of the urea cycle enzyme, arginine-succinate synthetase. Edema of the cerebral cortex is a consistent histopathological finding. The defect has been described in Australian and New Zealand Holstein-Friesian cattle. Affected animals appear healthy at birth, but die with acute onset neurologic disease within 1–4 days.
Clinical signs. Disorientation, depression, head pressing, convulsion.
Differential diagnosis. BVDV-induced cerebellar hypoplasia.

1.3 RESPIRATORY DISORDERS

1.3.1 Larynx

1.3.1.1 Necrotic laryngitis

Introduction. The larynx can be the portal of entry and reservoir for some pulmonary pathogens such as *Histophilus somni* and *Mycoplasma bovis*. Another pathogen consistently isolated is *Fusobacterium necrophorum*, causing laryngeal necrosis (calf diphtheria).
Clinical signs. Salivation, dyspnea, stretched neck, odor in breath.
Differential diagnoses. *Histophilus somni* necrohemorrhagic laryngitis, infectious bovine rhinotracheitis (IBR), fungi, trauma (drenching tube) or balling gun.

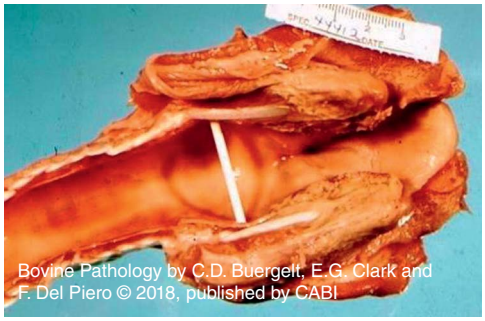

Fig. 1.20. Ox. Larynx. Necrocaseous laryngitis (calf diphtheria). Diphtheritic membranes are present on both sides of the larynx and vocal cords. The inflammation resulting from *Fusobacterium necrophorum* is secondary to the trauma to the region.

1.3.2 Lung

1.3.2.1 Bronchopneumonia

Introduction. Bronchopneumonia in calves is the result of multiple viruses and bacteria leading to the disease entity of enzootic pneumonia affecting dairy calves. Viruses predispose to bacterial infection in many instances. Morbidity is high. Risk factors such as lack of colostrum, poor hygienic management, overcrowded housing, faulty ventilation conditions, and inadequate nutrition are contributors to the entity of enzootic calf pneumonia.
Clinical signs. Nasal discharge, coughing, dyspnea, poor growth.
Differential diagnosis. Aspiration pneumonia.

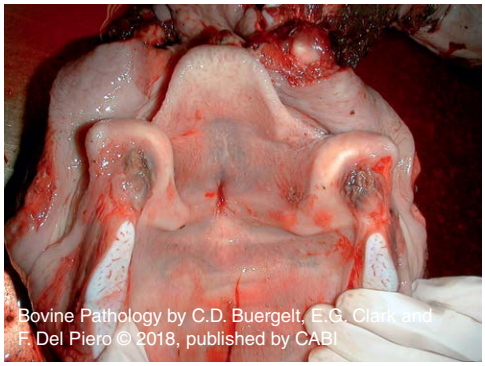

Fig. 1.21. Ox. Larynx. Fibrinonecrotic laryngitis. *Histophilus somni*. Focal mucosal necrosis is present next to the arytenoid cartilage. Early foci of infection may show vesiculopapular changes on the surface of cartilage.

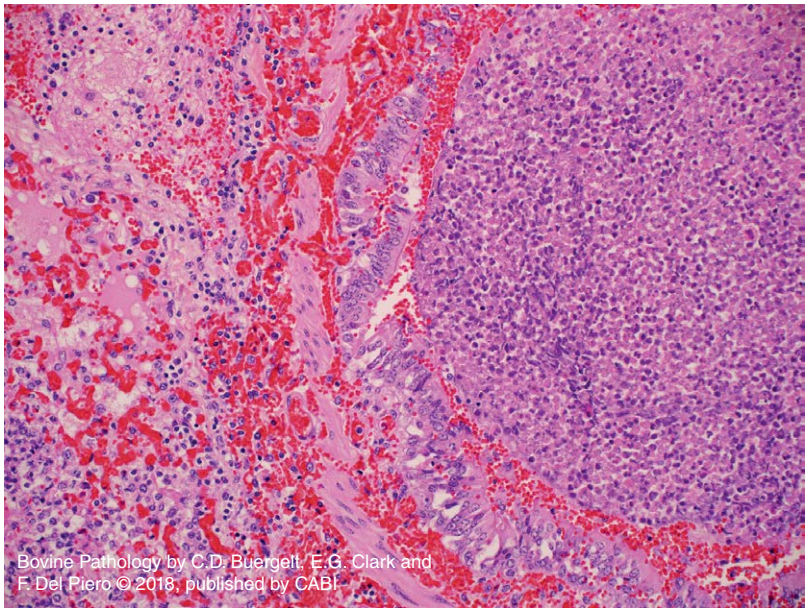

Fig. 1.23. Ox. Lung. Coronavirus pneumonia. Fibrinolymphocytic bronchitis. The bronchus is obstructed by a plug composed of fibrin, neutrophils and lymphocytes, many of which are degenerate. Adjacent alveoli are filled with a proteinaceous exudate and some lymphocytes. Coronavirus can be found in healthy animals in the respiratory tract (H&E). (Courtesy of Dr J. Caswell, University of Guelph, Canada.)

Fig. 1.22. Ox. Lung. Cranioventral necrosuppurative bronchopneumonia. Focal apical areas of consolidated lung lobules are characterized by purple discoloration. A variety of viral agents and some bacterial pathogens are responsible for the lesion.

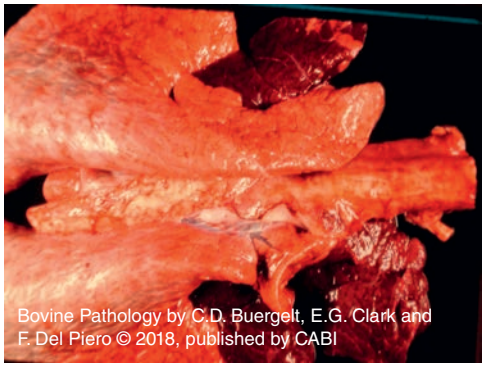

Fig. 1.25. Ox. Lung. Cranioventral suppurative bronchopneumonia. There is consolidation of both apical lobes. Adenovirus was isolated. The virus also causes enteritis in calves, resulting in watery diarrhea. If both organs are involved, the condition is called pneumoenteritis.

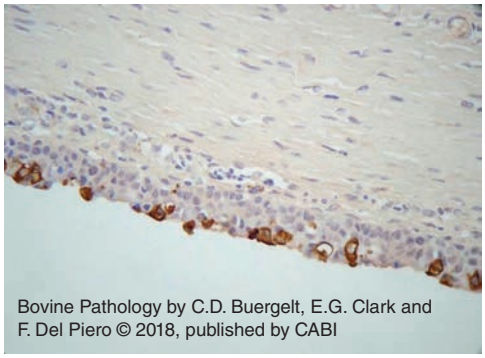

Fig. 1.26. Ox. Trachea. Adenovirus. Identification of viral antigen by indirect immunohistochemistry in the nuclei of epithelial respiratory cells (IHC).

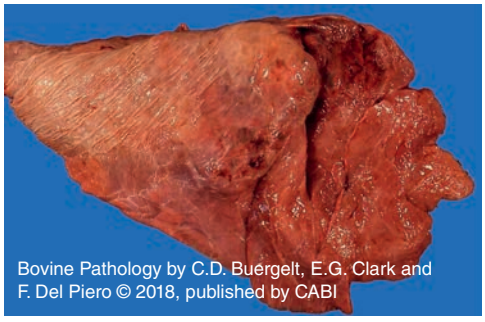

Fig. 1.27. Ox. Lung. Parainfluenza-3. Necrosuppurative bronchopneumonia. Apical, middle and diaphragmatic lobes are affected by brown consolidation.

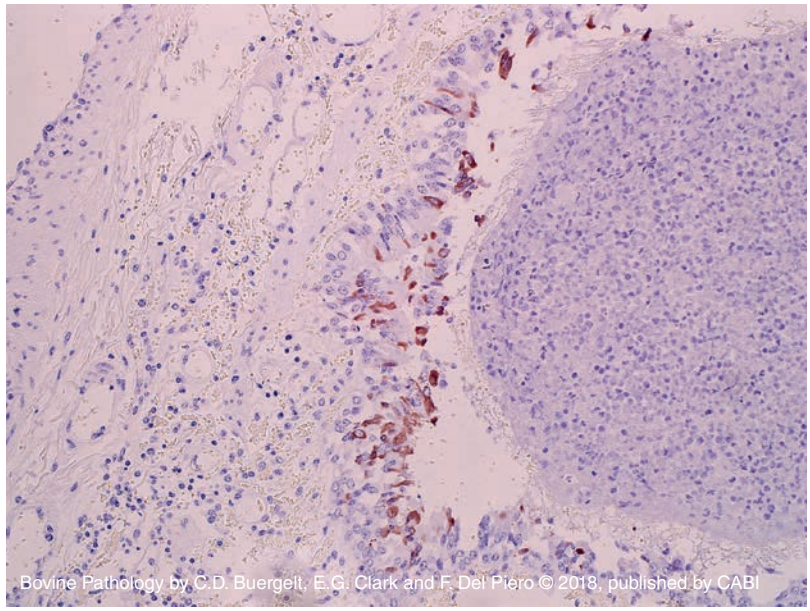

Fig. 1.24. Ox. Lung. Coronavirus pneumonia. Detection of viral antigen in the cytoplasm of respiratory epithelial cells (IHC). (Courtesy of Dr J. Caswell, University of Guelph, Canada.)

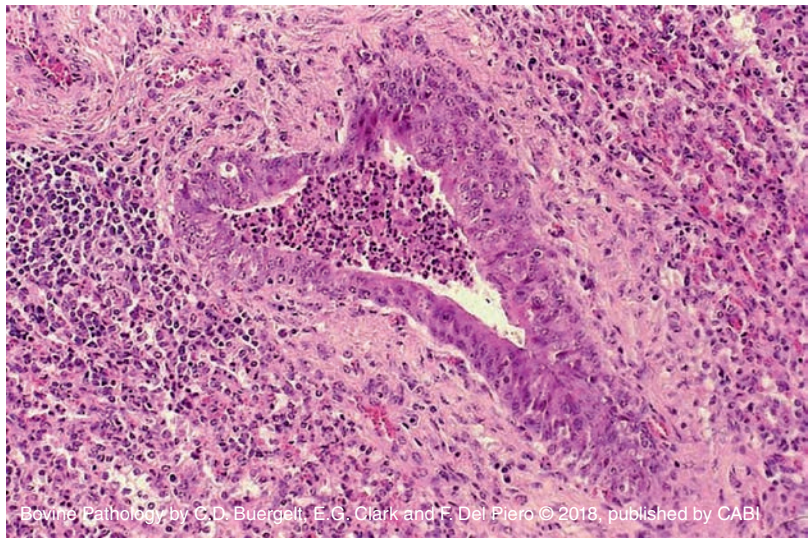

Fig. 1.28. Ox. Lung. Parainfluenza-3. Necrolymphocytic bronchopneumonia. Microscopically, the bronchiole is characterized by degeneration, sequestration and hyperplasia of respiratory epithelial cells. Fibrin and degenerate lymphocytes are present in the lumen. Adjacent alveoli are distended by lymphocytes (H&E).

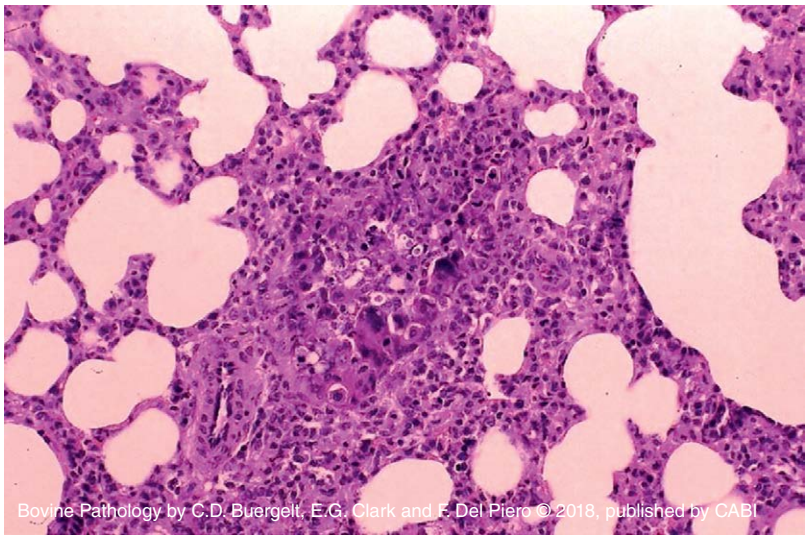

Fig. 1.29. Ox. Lung. Parainfluenza-3. Alveoli contain a focus of lymphocytes mixed with occasional multinucleated giant cells in the center (H&E).

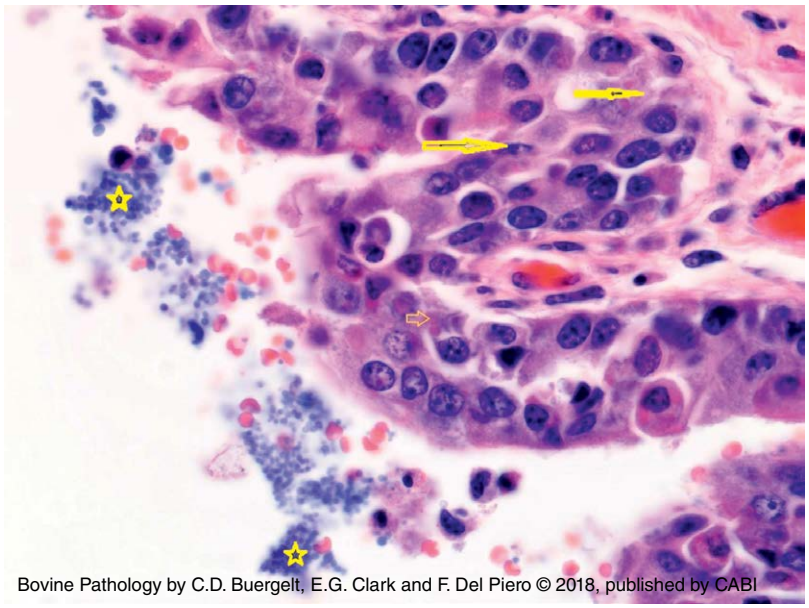

Fig. 1.30. Ox. Lung. Parainfluenza-3. Bronchus. Several eosinophilic viral inclusions (arrows) are visible in the cytoplasm of the respiratory epithelial cells. The lumen contains clusters of *Candida* yeast (asterisk) (H&E).

Fig. 1.32. Ox. Lung. Bovine respiratory syncytial virus (BRSV). Suppurative bronchopneumonia. Cranial and middle lung lobes are consolidated. Lobular distribution, diffuse edema and emphysema are characteristic of BRSV. More examples are demonstrated in Chapter 3: Diseases of the Respiratory System.

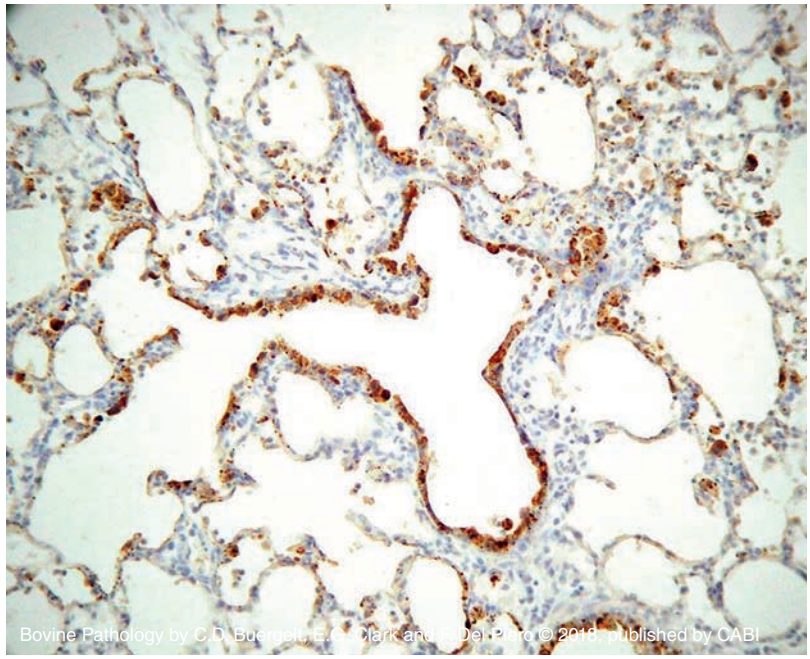

Fig. 1.31. Ox. Lung. Parainfluenza-3. Viral antigen is identified in the cytoplasm of bronchiolar lining cell, bronchi, multinucleate giant cells and macrophages (IHC).

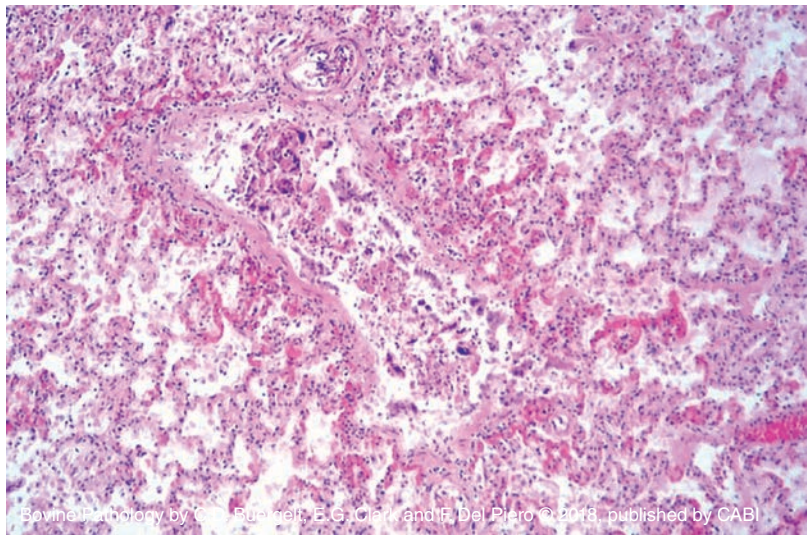

Fig. 1.33. Ox. Lung. Bovine respiratory syncytial virus (BRSV). Necrotizing bronchiolitis. Respiratory epithelium has sloughed into lumen, so are endothelial cells in the upper blood vessel. Alveoli are filled with fluid and loosely arranged, variably sized mononuclear cells (H&E).

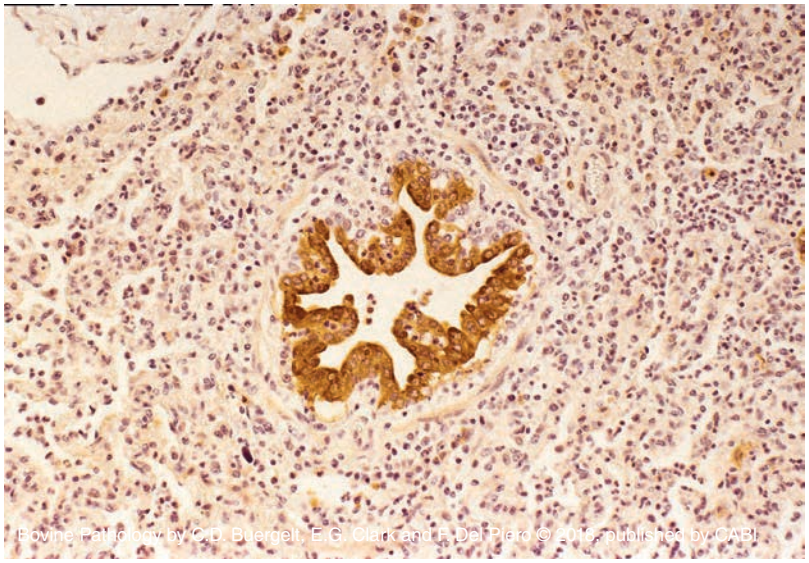

Fig. 1.34. Ox. Lung. Bovine respiratory syncytial virus (BRSV). The virus replicates in epithelial lining cells of the airways and viral antigen is demonstrated in the cytoplasm (IHC).

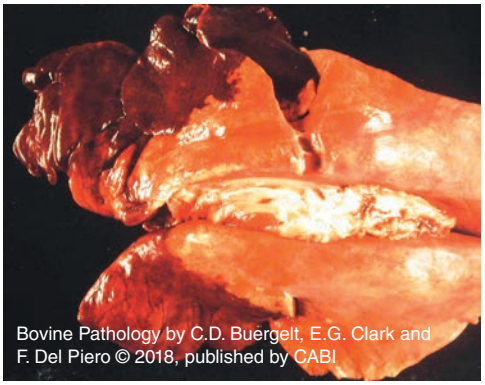

Fig. 1.35. Ox. Lung. Pasteurellosis. Anteroventral suppurative bronchopneumonia. An example of secondary bacterial infection to preceding viral pneumonia, it leads to consolidation without associated overlying pleural fibrin. *Pasteurella multocida* can sometimes be involved as the primary pathogen.

Fact Sheet: Enzootic Calf Pneumonia

Definition
Outbreak of pulmonary infection on dairy premises covering a broad spectrum of pulmonary pathogens and risk factors. The usual age range of affected dairy calves is up to 4 months.

Pathogens

- Primary
  - Coronavirus
  - Adenovirus
  - Parainfluenza-3 virus
  - Bovine respiratory syncytial virus
  - Bovine viral diarrhea virus (BVDV) (immunosuppressive)
- Secondary
  - *Pasteurella multocida*
  - *Mannheimia haemolytica*
  - *Trueperella pyogenes*
  - *Bibersteinia trehalosi*

Risk Factors
Overcrowding
Poor ventilation
Lack of ventilation
Pre-exposure to BVD virus
Wide fluctuation of weather

Another virus occasionally involved in enzootic calf pneumonia is bovine herpesvirus type 1 (BHV-1), which will be discussed in Chapter 3: Diseases of the Respiratory System.

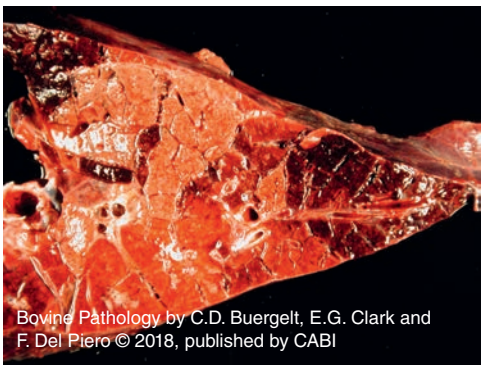

Fig. 1.36. Ox. Lung. Pasteurellosis. Necropurulent bronchopneumonia. The mottled appearance of the lobules on the cut section reflects various phases of inflammation and is a good indicator of *Pasteurella multocida* infection.

Fig. 1.37. Ox. Lung. *Mannheimia haemolytica*. Fibrinous pleuropneumonia. Severe consolidation of all lung lobes, with fibrin covering the pleura.

Fig. 1.38. Ox. Lung. *Mannheimia haemolytica*. Hemorrhagic bronchopneumonia with venous thrombosis. Marked necrosis and hemorrhage secondary to vasculitis are indicated by deep red discoloration of multiple lobules, with locally extensive distribution.

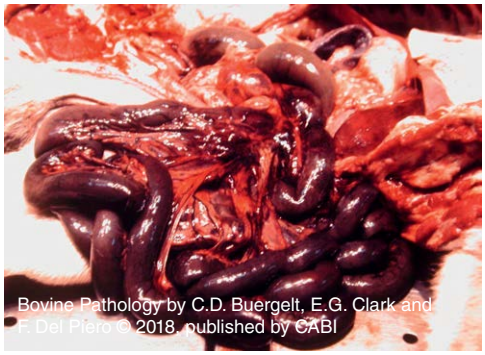

Fig. 1.39. Ox. Small intestine. Volvulus. Hemorrhagic jejunitis. Loops of jejunum are severely reddened due to a twist around the mesenteric attachment.

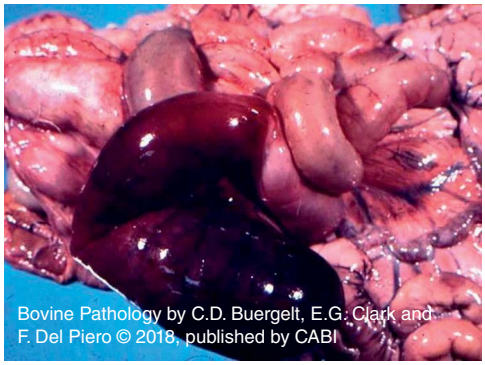

Fig. 1.40. Ox. Cecum. Torsion. Hemorrhagic typhlitis. The entire cecum has rotated around its longitudinal axis and is devitalized.

1.4 GASTROINTESTINAL DISORDERS

1.4.1 Displacements

Introduction. These are sporadic events and involve small and large intestines. Rotations (volvulus, torsion) or intestinal telescoping (intussusception) result in severe toxic necrosis of the tissue segments involved, due to obstruction of blood flow.
Clinical signs. Obstruction, pain, shock.
Differential diagnosis. Acute enteric pathogens (clostridial disease).

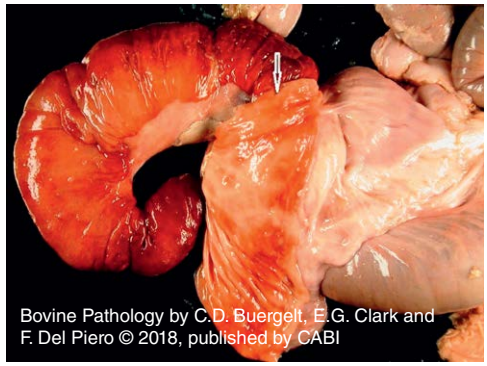

Fig. 1.41. Ox. Cecum. Intussusception. Necrohemorrhagic typhlitis. The entire cecum has telescoped into the colon. Point of invagination is depicted by arrow.

1.4.2 Inflammation

Introduction. Gastrointestinal inflammation in calves is frequently the result of infection. Infectious pathogens are of significant economic consequences and comprise virus, bacteria, protozoa, and occasional fungi and endoparasites. Dual etiology is common as one type of agent predisposes to another.
Clinical signs. Diarrhea, weight loss, dehydration, perineal pasting.
Differential diagnoses. Intestinal displacements, starvation.

Fig. 1.43. Ox. Rumen. Candidiasis. Parakeratosis. Multifocal dry nodules on the mucosa develop from yeast infection. Similar changes can be encountered on the tongue and in the mucosa of the esophagus. Affected animals are usually immunocompromised. Excessive use of antibiotics can cause ruminal candidiasis as well.

Fig. 1.44. Ox. Rumen. Infectious bovine rhinotracheitis (IBR). Exudative and proliferative rumenitis. Curdled exudate and parakeratosis are features of the alimentary form of IBR in the calf (BHV-1). With this form, lesions would also be expected in the oral cavity and esophagus.

Fig. 1.42. Ox. Rumen/reticulum. Toxic rumenitis/reticulitis. The condition is known as 'ruminal drinking' of suckling calves when a faulty esophageal groove reflux results in the ingestion of milk into the reticulum/rumen. The formation of toxic metabolic fermentation products leads to tympany, mucosal hyperemia, necrosis, and ulceration

Calf scours

Acute enteric diseases of calves are a major cause of mortality in the first weeks of life, and of economic importance for producers replacing livestock with their own progeny. A variety of pathogens are involved, single or in concert with each other. The failure of passive transfer, lack of vaccination, poor hygiene, crowding, inadequate nutrition, and wet, cold conditions are major risk factors. Profuse diarrhea and dehydration are the clinical signs of pathologic intestinal damage. Responsible pathogens can be classified into viral, bacterial, and protozoal.

1.4.2.1 Viruses

Introduction. The viruses responsible for outbreaks of enteritis with severe clinical complications are rotavirus (*Reoviridae*) and coronavirus (*Coronaviridae*). Most infections occur during the first weeks of life. Diagnosis is made via enzyme-linked immunosorbent assay (ELISA), fluorescense microscopy (FM), PCR, immunohistochemistry (IHC), or detection of viral particles in negatively stained feces by transmission electron microscopy (TEM). Other enteric viruses with lesser clinical impact are enteric parvovirus, adenovirus and torovirus (Breda virus) infecting older calves. The role of bovine viral diarrhea virus (BVDV) and IBR in primary calf enteritis is less defined.

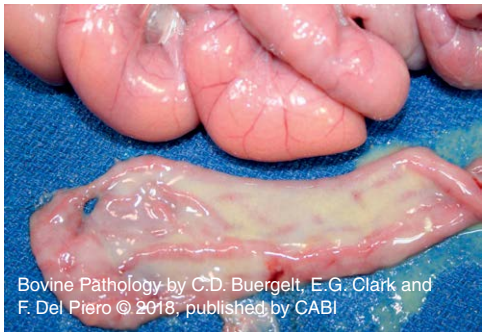

Fig. 1.45. Ox. Jejunum. Coronavirus. Catarrhal enteritis. Loops of jejunum are dilated and extended by milky fluid. When opened, the mucosa is covered by a mucous exudate. Rotavirus infection grossly induces similar changes. Also, intestinal content is identical in a very young calf on a high milk diet.

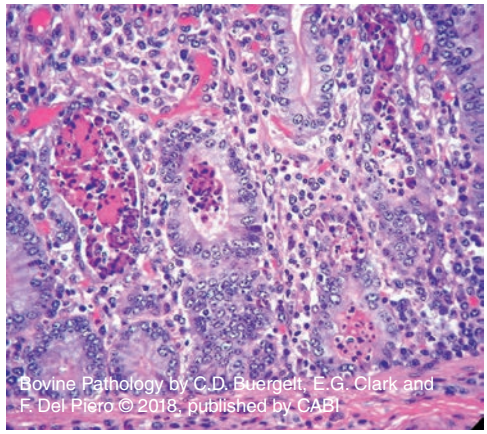

Fig. 1.46. Ox. Jejunum. Coronavirus. Crypt abscesses. Necrotic cellular debris with some fibrin is located within crypts. The lamina propria is infiltrated moderately by lymphocytes and some plasma cells (normal) (H&E).

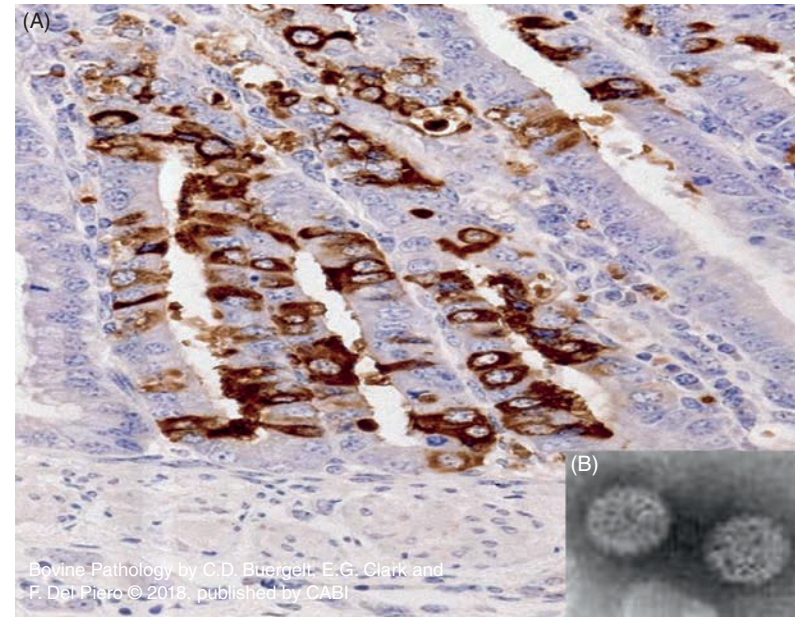

Fig. 1.47. Ox. Colon. Coronavirus. (A) Viral antigen is identified diffusely in the cytoplasm by indirect immunohistochemistry (IHC). (B) (inset) Negatively stained coronavirus particles demonstrated by transmission electron microscopy (TEM). (Courtesy of Dr M. Hines II, University of Georgia, USA.)

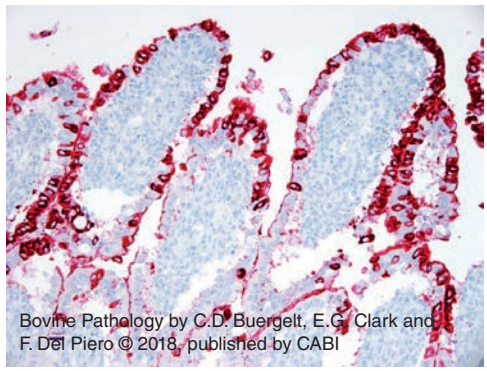

Fig. 1.49. Ox. Small intestine. Rotavirus. Viral antigen is located diffusely in cytoplasm of superficial enterocytes (IHC). (Courtesy of Dr B. Brodersen, University of Nebraska-Lincoln, USA.)

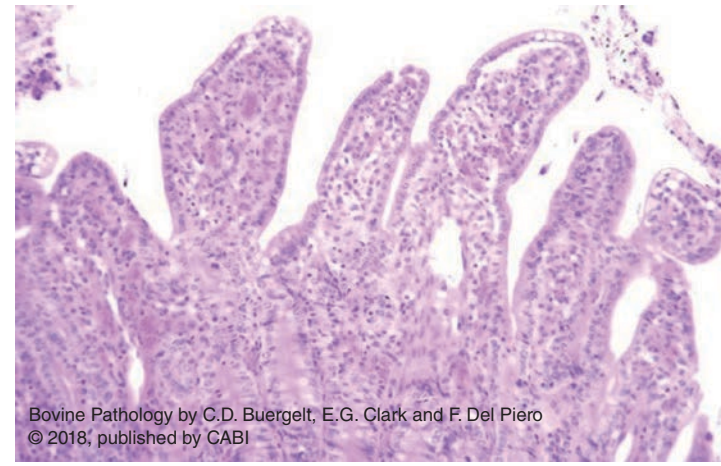

Fig. 1.48. Ox. Small intestine. Rotavirus. Villous atrophy. Villi are slightly shortened and clubbed. The lamina propria contains a normal number of lymphocytes (H&E).

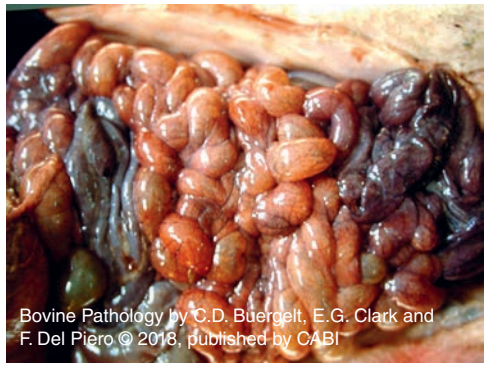

Fig. 1.50. Ox. Small intestine. Colibacillosis. Catarrhal enteritis. Loops of small intestine are markedly distended by watery fluid; some segments have a reddened wall.

1.4.2.2 Bacteria

Introduction. The main types of contagious bacterial enteric pathogens are *Escherichia coli* and *Clostridium perfringens* in the first 2 weeks and *Salmonella enterica* in the first 1–3 months of life. The highly virulent pathogens are associated with high mortality, and in the case of *E. coli*, lead to sepsis (septicemic colibacillosis).

Clinical signs. Watery diarrhea, dehydration, depression, sunken eyes, recumbency.

Differential diagnoses. Protozoa, viruses.

Escherichia coli (E. coli). Various strains of *Escherichia coli* are involved in disease outbreaks, with some of them often fatal (enterotoxigenic *E. coli*). *E. coli* bacteria occur as non-virulent and virulent strains. Non-virulent strains are part of the normal intestinal flora. Virulent strains are classified as to serotypes, composition of antigens and the damage they cause in the intestinal tract. Major strains involved in enteric colibacillosis are the enterotoxigenic (ETEC) strain, the enterohemorrhagic (EHEC) strain, the attachment and effacing (AEEC) strain, a subset of the enteropathogenic, enteroinvasive (EPEC) strain, and the necrotoxigenic strain (NTEC), producing cytotoxic necrotizing factors. The enterohemorrhagic strain produces a Shiga-toxin (verotoxin), damaging intestinal epithelium and vascular endothelium, causing erosive fibrinohemorrhagic enterocolitis. The strain contributes to foodborne illness in humans. Enterotoxigenic *E. coli* produces enterotoxin and colonizes the intestinal epithelium via fimbriae, also known as pili. Immunohistochemical stains for fimbrial adhesin is helpful for the diagnosis, especially of K99 *E. coli*. Culture can be attempted if the animal has not been treated extensively with antibiotics. Microscopic lesions are usually minimal for this strain. Enteroinvasive strains disseminate systemically and are responsible for calf sepsis (septicemic colibacillosis).

Enterotoxemia. Clostridial disease, caused by *Clostridium perfringens*, type A (90% of cases) and type C (5% of cases), secretes enterotoxins, alpha in the case of type A and alpha and beta in the case of type C, causes hemorrhage and ischemic necrosis of small intestinal tissue, leading to gas production, bloat, colic, toxic shock and death.

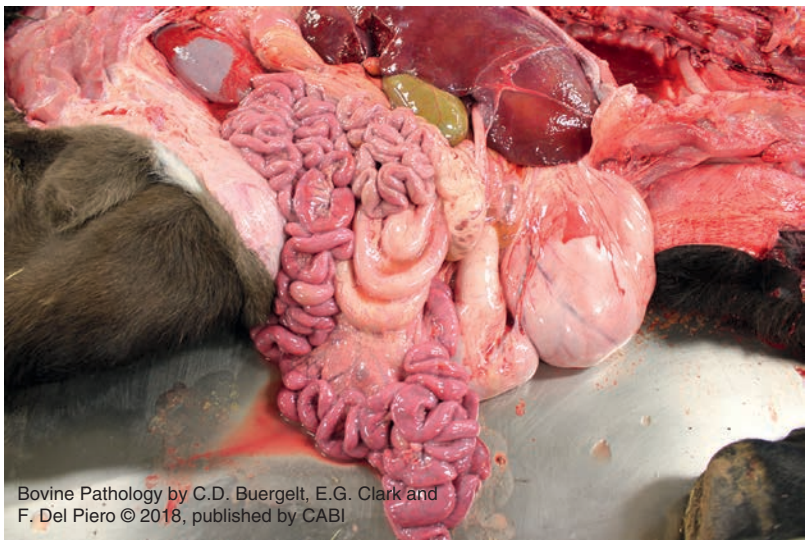

Fig. 1.52. Ox. Small intestine. Clostridial disease. Catarrhal, hemorrhagic enteritis. 'Purple gut'. Distinct purple discoloration of intestine. *Clostridium perfringens* type C is usually isolated. (Courtesy of Dr D. O'Toole, University of Wyoming, USA.)

Fig. 1.51. Ox. Small intestine. Clostridial disease. Catarrhal hemorrhagic enteritis. Excessive blood-tinged watery fluid is released from the intestinal lumen. *Clostridium perfringens* sp. (Courtesy of Dr D. O'Toole, University of Wyoming, USA.)

Fig. 1.53. Ox. Small intestine. Clostridial disease. Necrotizing, fibrinous, hemorrhagic enteritis. What looks like digesta is an adherent exudate with a distinctive green tinge which does not wash off. (Courtesy of Dr D. O'Toole, University of Wyoming, USA.)

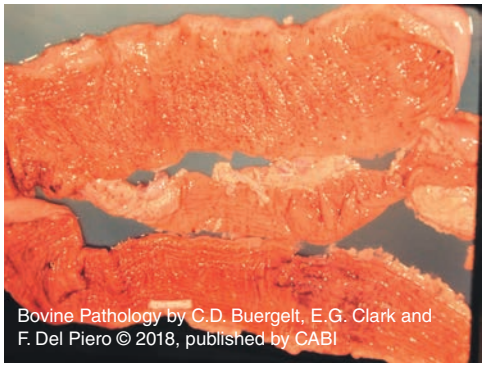

Fig. 1.54. Ox. Small intestine. Acute salmonellosis. Multifocal hemorrhagic enteritis. Petechiae cover the mucosa.

Fig. 1.55. Ox. Colon. Subacute salmonellosis. Transmural necrohemorrhagic enteritis. The entire wall of the intestine is friable, discolored and contains multiple pale foci of necrosis.

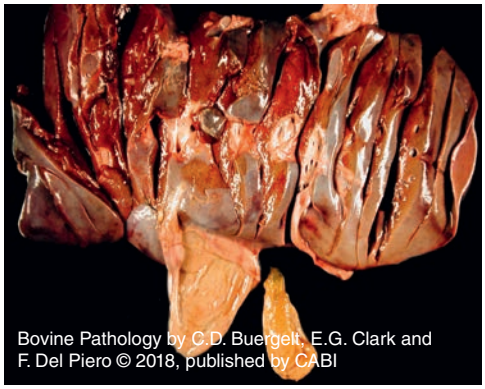

Fig. 1.57. Ox. Gall bladder. Salmonellosis. Fibrin plug. A yellow fibrin cast contained within the gall bladder is highly suggestive of infection with *Salmonella* spp.

Salmonellosis. Salmonella is an intracellular, gram-negative pathogen with zoonotic potential, and infects both calves and adult cattle. Infection phases range from peracute to chronic, with a pathologic spectrum ranging from minimal punctuate focal hemorrhage in the intestinal mucosa in acute cases to inspissated diphtheritic membranes covering the mucosa of small and large intestines in chronic cases. The species of Salmonella involved in the majority of infections is *Salmonella enterica* with six subspecies. Two capabilities allow *Salmonella* spp. to cause inflammation: penetration of the intestinal epithelium (M-cells); survival in macrophages.

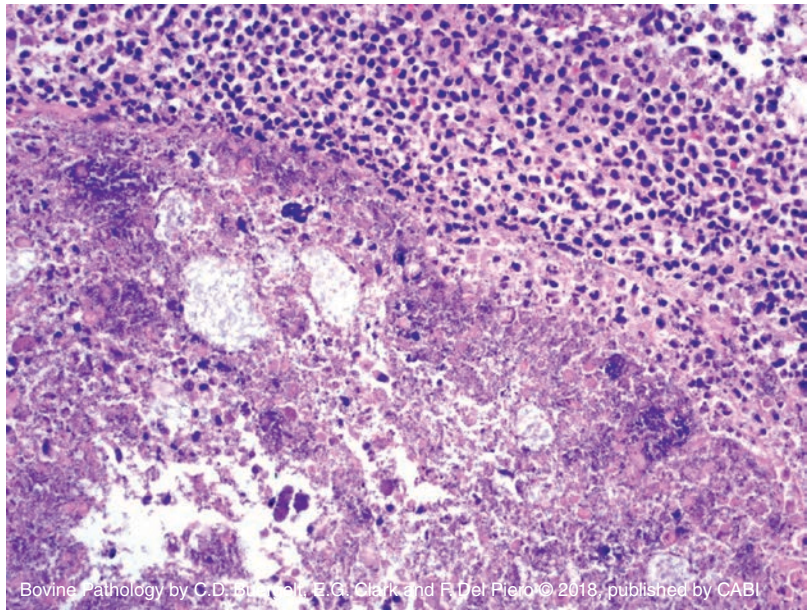

Fig. 1.56. Ox. Intestine. Salmonellosis. Fibrinonecrotic enteritis. The mucosa denuded from its enterocyte lining is covered by fibrin and degenerate, necrotic inflammatory cells. Intermingled are numerous bacterial colonies. Fibrin thrombi in small lamina propria capillaries are suggestive indicators of the disease, although clostridiosis has also been associated with thrombi (H&E).

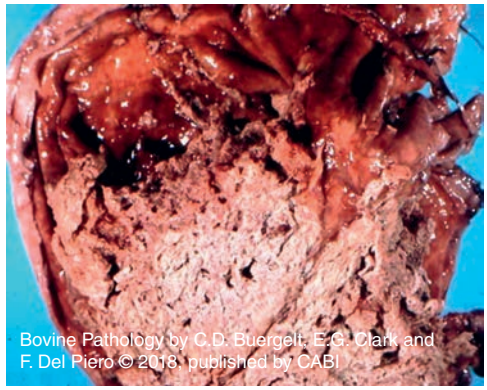

Fig. 1.58. Ox. Abomasum. Salmonellosis. Fibrinous abomasitis. The mucosa is covered with sheaths of fibrin, resembling curdled milk. Differential diagnosis should include infection with the bovine rhinotracheitis virus (IBR), clostridial abomasitis.

1.4.2.3 Protozoa

Introduction. Cryptosporidiosis, giardiasis and coccidiosis are all enteric diseases involved in calf scours and morbidity.
Clinical signs. Profuse watery or bloody diarrhea.
Differential diagnoses. *E. coli*, *Salmonella* spp., bovine rotavirus, bovine coronavirus.

Cryptosporidiosis. Two species are prevalent in calves, *Cryptosporidium parvum* and *Cryptosporidium andersoni*. *C. andersoni* resides in the abomasum, whereas *C. parvum*, a zoonotic pathogen, resides within the intestinal brush border. The agents are intracellular and extracytoplasmic. Transmission is via the oral–fecal route from contaminated environments, water or sewage. Grossly, the intestinal mucosa may be mildly reddened and may be covered by mucous fluid.

Giardiasis. Caused by *Giardia duodenalis*, the flagellated organism attaches to the microvilli of the intestinal epithelium with a ventral sucker interfering with membrane function to induce malabsorption. There are usually no gross changes in infected animals.

Coccidiosis. Infestation in calves with *Eimeria bovis*, but more often with *Eimeria zuernii*, causes severe profuse bloody diarrhea associated with tenesmus, dysentery, rectal prolapse, and weight loss. Small and large intestine are involved. The nervous form of coccidiosis has been reported from northern America due to a potential toxin produced by the parasite.

Fig. 1.61. Ox. Colon. Coccidiosis. Hemorrhagic colitis. Segments of the colonic mucosa are deeply red and thickened, and contain a luminal necrotic cast and/or fibrin.

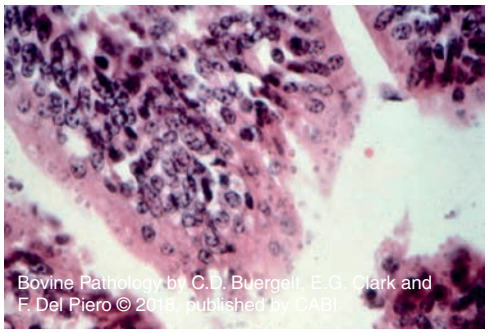

Fig. 1.59. Ox. Jejunum. Cryptosporidiosis. Ruptured brush border. The brush border is discontinuous and contains multiple intracellular, small basophilic structures, 1–2 microns in diameter (H&E).

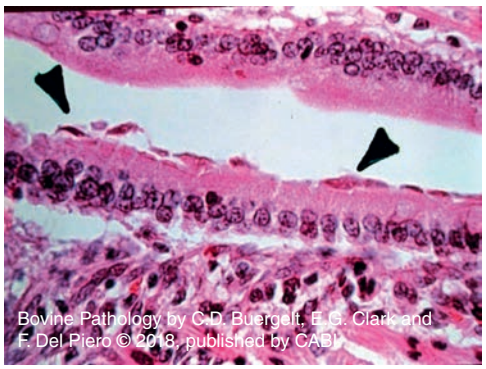

Fig. 1.60. Ox. Jejunum. Giardiasis. Protozoal attachment. Flagellated, banana-shaped structures (arrow heads) phagocytize the intestinal brush border (H&E).

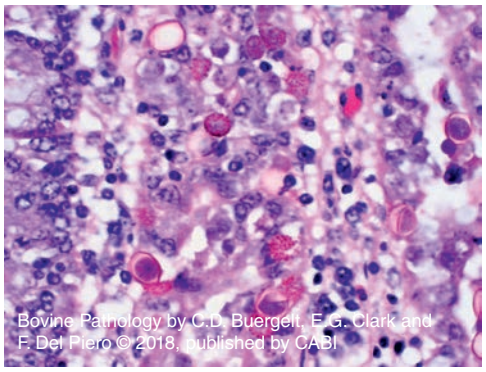

Fig. 1.62. Ox. Colon. Coccidiosis. Lymphocytic colitis with gametogony. Various forms of gametes are lodged within the villous tips of the mucosa (H&E).

1.4.2.4 Fungi

Introduction. Mycotic enteritis is a sporadic individual event and can be predisposed by excessive antibiotic treatment. It needs to be considered in the differential diagnosis of diarrhea when individual animals are affected.

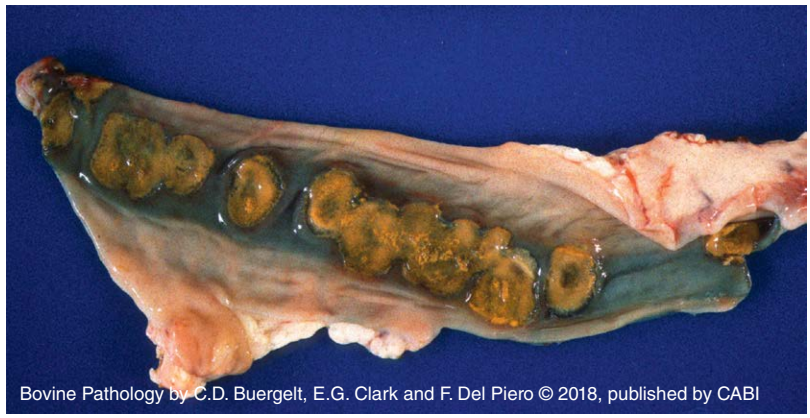

Fig. 1.63. Ox. Jejunum. Mycosis. Multifocal angiocentric fibrinonecrotic enteritis. Elevated plaques with a tan, friable center and dark rim suggest fungal involvement. (Courtesy of the Government of Alberta, Canada.)

1.4.2.5 Parasites

Introduction. Infestation with endoparasites in calves is usually asymptomatic and their presence an incidental finding at necropsy. Endoparasites involved are nematodes and cestodes.

Fig. 1.64. Ox. Ascarids. Adults of *Toxocara vitulorum* collected from the small intestine.

1.5 ADDITIONAL GASTRIC CONDITIONS

1.5.1 Abomasal foreign body formations

These are known as trichobezoars (pilobezoars, hairballs) and incidental findings at necropsy. They develop from self-suckling and swallowing of hair and become entrapped in the abomasum without causing clinical signs. This is particularly common in veal calves. Calves may occasionally be born with hairballs. If the conglomerate is made of plant material, the formations are called phytobezoars. The combination of plant and hair material is called phytotrichobezoars.

Fig. 1.65. Ox. Abomasal trichobezoars. Multiple, different-sized hairballs were retrieved from the abomasum of an individual or multiple calves.

Fig. 1.66. Ox. Whole body. Abomasal tympany (bloat) due to perforated abomasal ulcer. The abdomen is markedly distended by severe antemortem gaseous accumulation (bloat) in the abomasum.

1.5.2 Abomasal ulceration

Introduction. Commonly seen in the abomasum in feedlot cattle. May perforate, especially in young beef calves 2–4 months of age on pasture. In dairy calves, they develop secondarily to stress, poor milk quality, coarse feed, grain overload and subsequent overgrowth of *C. perfringens* type A, rarely type C. A preceding infection with BVDV cannot be confirmed. A small outbreak of abomasal ulcers in dairy calves has been associated with rotavirus infection.

A research project in Canada devoted to finding a cause in pastured beef calves failed to do so.

Clinical signs. Bloat, colic, shock.

Differential diagnoses. Intestinal displacement, peritonitis.

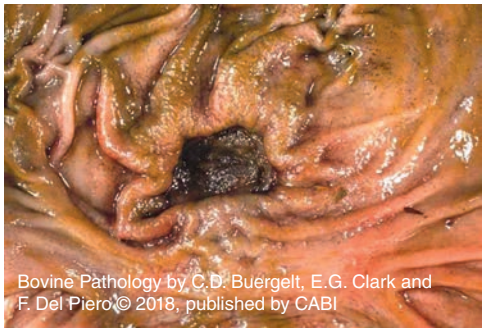

Fig. 1.67. Ox. Abomasum. Chronic, non-perforating ulcer. The ulcer bed is covered by blood and fibrin. The rounded mucosal edges suggest chronicity.

1.5.3 Hemorrhagic abomasitis

Introduction. Sporadic cases have been incriminated to be caused by infectious agents, such *Clostridium septicum* (braxy), *C. perfringens* type A, *Sarcina ventriculi* or *Salmonella* spp.

Clinical signs. Abdominal pain, tympany, shock.

Differential diagnosis. Abomasal torsion.

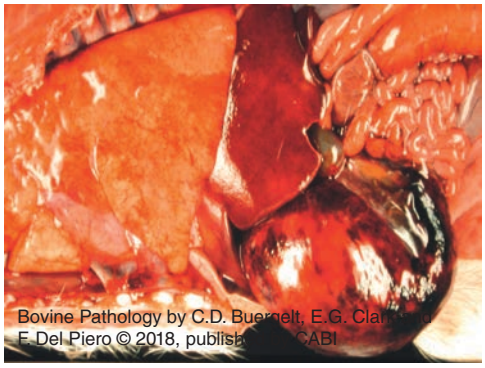

Fig. 1.68. Ox. Abomasum. Braxy-like hemorrhagic abomasitis. Transmural hemorrhage in slightly distended abomasum. *Clostridium septicum* was isolated. The condition in sheep is known as braxy.

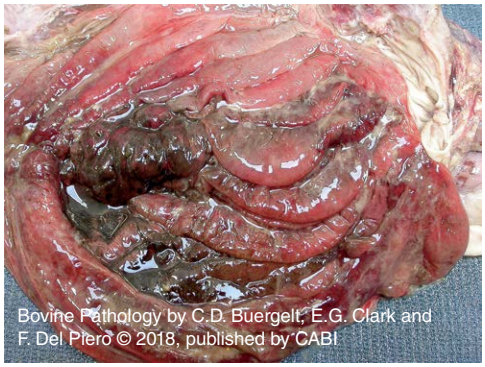

Fig. 1.70. Ox. Abomasum. Braxy-like hemorrhagic necrosis. The abomasal wall is markedly thickened by edema and hemorrhage. The weakened wall is susceptible to perforation. *Clostridium* spp. and *Sarcina* spp. are possible etiologic agents.

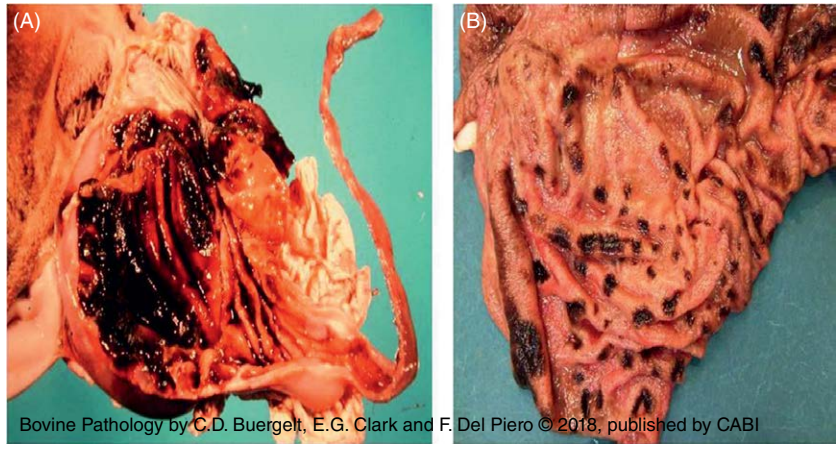

Fig. 1.69. Ox. Abomasum. Multifocal hemorrhagic abomasitis. (A) Large foci of a thickened mucosa are affected by severe hemorrhage. *Clostridium perfringens* type A was isolated. (B) Punctuate foci of hemorrhage are scattered throughout the mucosa. *Salmonella* spp. was isolated.

Fig. 1.72. Ox. Abomasum. Mycosis. Angiocentric hemorrhagic abomasitis. Patches of red rings cover the mucosa, suggesting vascular invasion by systemic fungi such as *Aspergillus* spp. Fungal invasion is frequently a sequel to excessive antibiotic treatment or salmonellosis.

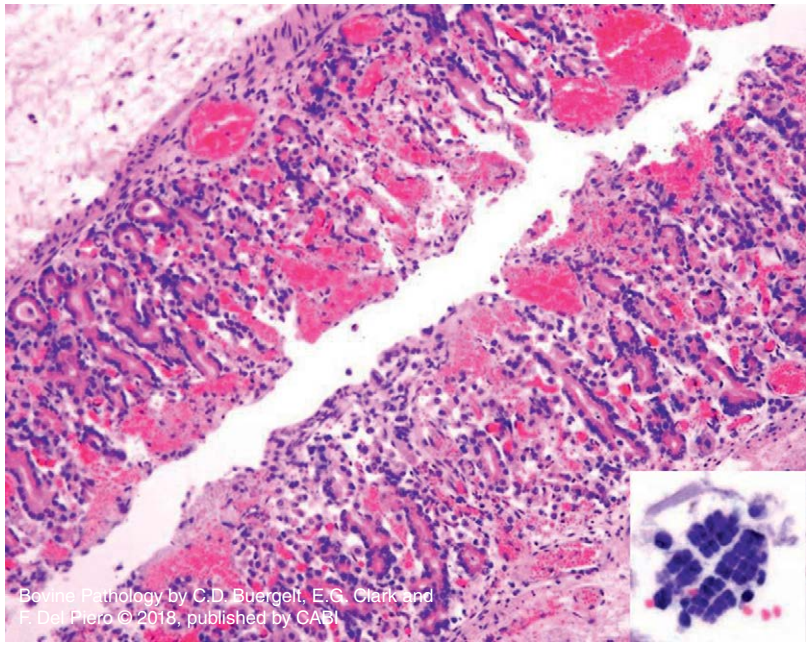

Fig. 1.71. Ox. Abomasum. Fibrinonecrotic hemorrhagic abomasitis. Superficial mucosal cells are necrotic with some fibrin on the surface. There is some lymphocytic infiltration in the lamina propria which is of no significance. Lamina propria blood vessels are markedly congested, partially thrombosed and have perivascular lymphocytic inflammation (H&E). Inset: packet-forming *Sarcina ventriculi* cocci. These may be found on the mucosal surface in calves with abomasal bloat and abomasitis (Giemsa stain).

1.5.4 Calf sepsis (formerly septicemia)

Introduction. Neonatal calves are most susceptible to sepsis following infection with *E. coli*, *Salmonella* spp. and other gram-negative bacteria. Risk factors are weather fluctuations, minimal shelter, lack of colostrum, poor birth hygiene, and umbilical infection as the portal of entry. The term 'septicemia' is now rarely used and has been replaced with sepsis. Sepsis is a life-threatening condition that arises when the body's response to infection injures its own tissues and organs. Sepsis is defined as the systemic inflammatory response syndrome (SIRS) in response to an infectious process. SIRS constitutes the presence of two or more of the following: abnormal body temperature, heart rate, respiratory rate or blood gas, and white blood cell count.

Clinical signs. Fever, depression, anorexia, disorientation, lameness, sudden death.

Differential diagnoses. Congenital abnormalities affecting individual vital organs, cold (freezer) artefact, BVDV infection (see Chapter 15: Diseases of Eye and Ear).

Figures 1.74–1.78 exhibit the frequent gross pathologic changes encountered with septic calves.

Fig. 1.73. Ox. Umbilicus. Suppurative omphalophlebitis. The umbilicus and ductus venosus are markedly distended by pus, serving as the perfect portal of entry for opportunistic pathogens to translocate hematogenously to various organ systems such as the liver, which contains multiple abscesses.

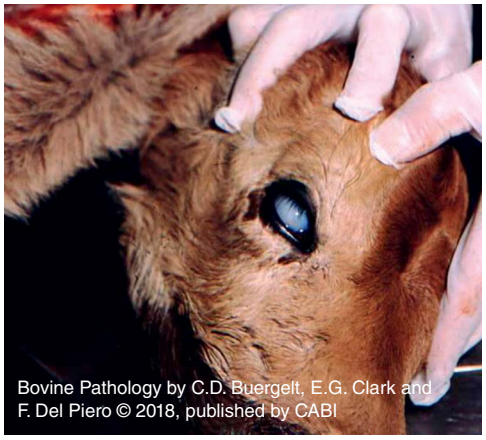

Fig. 1.76. Ox. Eye. Calf sepsis. Hypopyon. A cloudy fibrinosuppurative exudate occupies the anterior eye chamber.

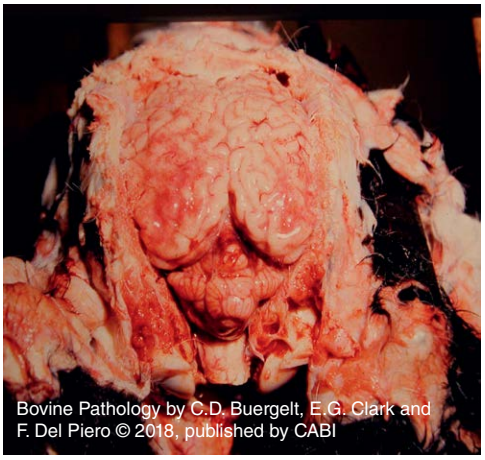

Fig. 1.74. Ox. Brain. Calf sepsis. Hemorrhagic meningitis. The meningeal surface exhibits ecchymotic hemorrhage over occipital lobes and cerebellum.

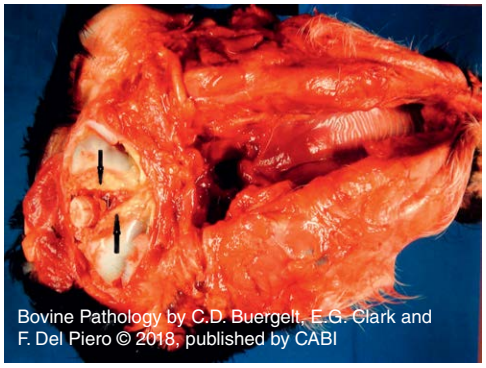

Fig. 1.77. Ox. Atlanto-occipital joint. Calf sepsis. Fibrinous arthritis. Fibrin plugs (arrows) close to the condyles are good indicators for the diagnosis of calf sepsis.

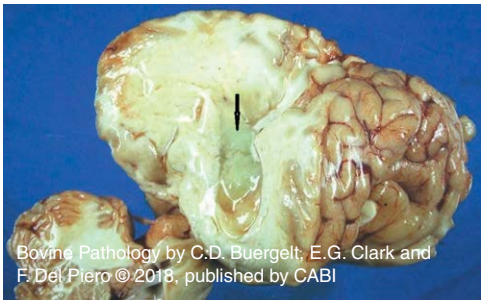

Fig. 1.75. Ox. Brain. Calf sepsis. Purulent ventriculitis. The lateral ventricle (arrow) is filled with viscous pus.

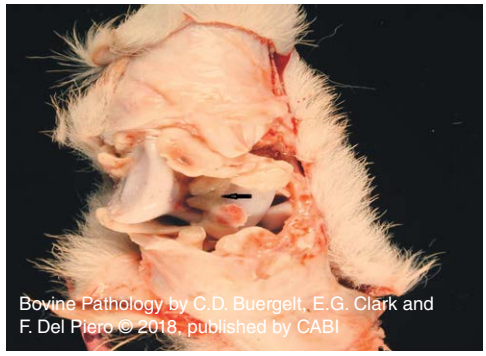

Fig. 1.78. Ox. Hock joint. Calf sepsis. Fibrinous arthritis. Fibrin casts (arrow) cover the articular cartilage. They should not be confused with fat. The hock joints should always be cultured in cases of calf sepsis, with the intent to isolate the pathogen. An antibiotic resistance test should be performed at the same time.

Fact Sheet: Calf Sepsis

Definition

Inflammatory response of various organs to embolic dissemination of gram-negative bacteria from a primary source (nidus) or to toxins (sepsis) produced by the gram-negative bacteria.

Primary source

- Umbilicus
- Digestive tract
- Tonsil

Organ systems involved in gross pathology

- Joints
- Meninges
- Brain
- Eyes
- Gall bladder

Infectious agents isolated

- Coliforms
- *Salmonella* spp.
- *Clostridium perfringens*

Co-infection

- Rotavirus
- Coronavirus
- *Cryptosporidium parvum*
- *Giardia duodenalis*

Risk factors

- Poor umbilical hygiene
- Failure of passive transfer of immunoglobulins
- Nutritional imbalance
- Management practices

1.6 MUSCULOSKELETAL DISORDERS

1.6.1 Muscular system

1.6.1.1 White muscle disease (WMD)

Introduction. The disorder is an example of a nutritional myodegeneration and the result of vitamin E/selenium deficiency observed in many vertebrates. It is responsive to selenium treatment. It typically occurs in fast-growing animals and affects multiple striated muscle groups, principally the most active muscles, and the heart. Sources of nutrient deficiency are selenium-deficient soil or moldy feed (hay). In newborn calves, severe vitamin E deficiency (drought) with no selenium deficiency cause white muscle disease. There is more on WMD in Chapter 8: Diseases of the Musculoskeletal System.

Clinical signs. Lameness, stiff gait, myoglobinuria, aspiration pneumonia, respiratory distress.

Differential diagnoses. Toxic myodegeneration (plants, ionophore antimicrobial), tetanus, hemolytic disorders.

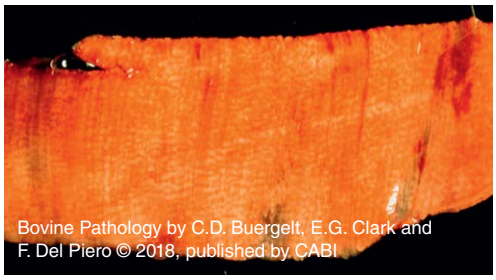

Fig. 1.79. Ox. Diaphragm. White muscle disease (WMD). Myodegeneration. A paintbrush linear arrangement of streaks is distributed along the length of muscle fibers.

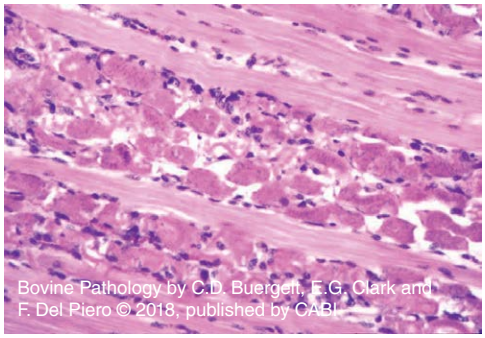

Fig. 1.80. Ox. Skeletal muscle. White muscle disease (WMD). Myonecrosis with mineralization. Swollen muscle fibers have undergone coagulative necrosis with heavy mineralization. Mild lymphocytic inflammation is present adjacent to dense sarcolemmal cells (H&E).

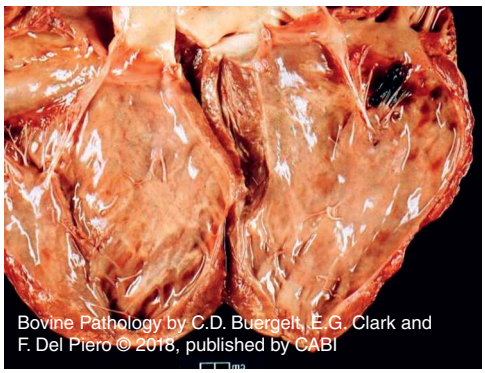

Fig. 1.81. Ox. Heart. White muscle disease (WMD). Cardiac myodegeneration. There is diffuse pallor affecting a thin left ventricle. The presentation of cardiac WMD is unusually extensive in this case.

1.6.2 Skeletal system

Introduction. Skeletal abnormalities may be genetic, teratogenic (viruses), or nutritional in origin.

Clinical signs. Lameness of various degrees; stunted limbs; disproportionate body parts; recumbency.

Differential diagnosis. Acquired locomotor disorders

1.6.2.1 Congenital chondrodysplasia

The condition occurs mainly in beef cattle breeds and produces disproportionate calves (bovine dwarfism). Depending on the breed, a genetic etiology is often involved, with the mode of inheritance either autosomal dominant or autosomal recessive. In some cases, mineral deficiency (zinc, manganese) is hypothesized as being the cause of the deformities. The deformity involves various anatomic parts of the skeletal system (see Chapter 8: Diseases of the Musculoskeletal System).

Fig. 1.82. Ox. Head. Chondrodysplasia. A shortening of the cervical vertebral column of the neck with a relatively large head has created the phenotypical appearance of a 'bull nose'.

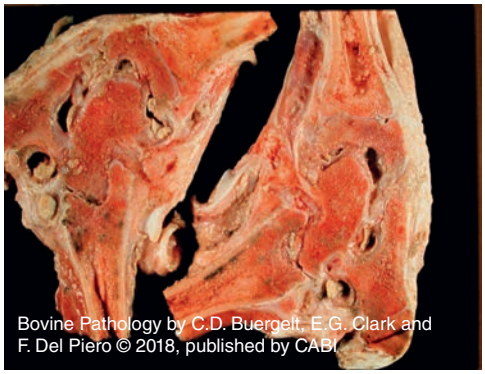

Fig. 1.84. Ox. Joint. Tarsal joint. Septic arthritis. The joint is filled with purulent exudate. Trauma and subsequent infection can be considered a cause if a single joint only is involved.

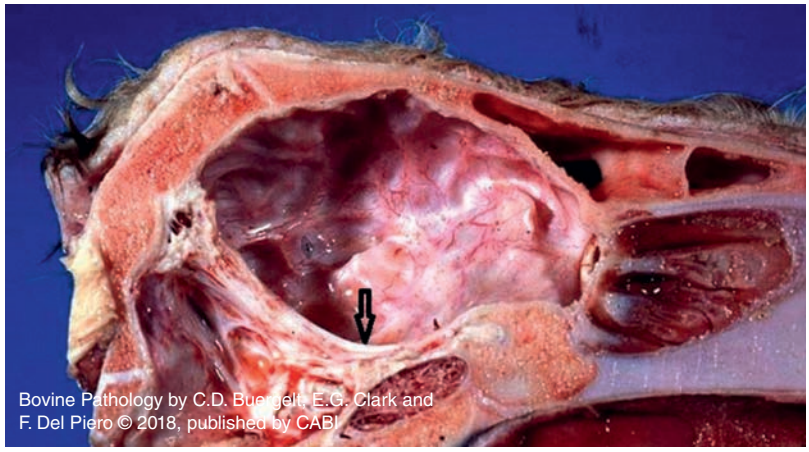

Fig. 1.83. Ox. Skull. Bovine dwarfism. Wing of basisphenoid bone. The accentuation of a sharp, bony proliferation (arrow) is a good anatomic indictor of bovine dwarfism. (Courtesy of Dr J.M. King and Section of Anatomic Pathology, Cornell University, USA.)

1.6.2.2 Arthritis

More examples will be presented in Chapter 8: Diseases of the Musculoskeletal System.

Fig. 1.85. Ox. Peritoneum. Mesothelioma. The serosal surface of the rumen and the abdominal peritoneum are covered by white to yellow, nodular neoplastic growths, many of which are confluent. Calves may be born with the condition.

1.7 NEOPLASIA

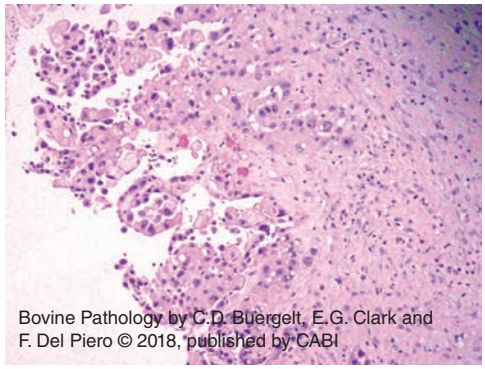

Fig. 1.86. Ox. Peritoneum. Mesothelioma. The mesothelioma is characterized histologically by clusters of pleomorphic cells with large hyperchromatic nuclei. The neoplastic cells form papilla projections and rest on a fibrovascular stroma. The histologic and immunohistochemical identification of mesothelioma and differentiation from carcinoma historically has always been challenging (H&E).

1.8 MISCELLANEOUS

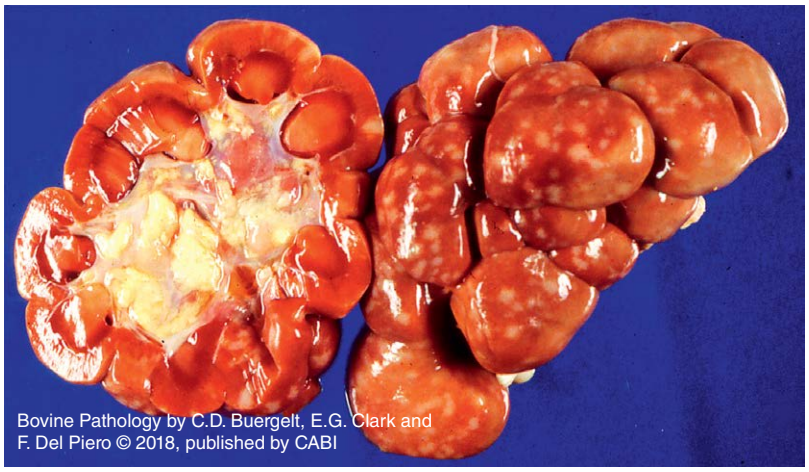

Fig. 1.87. Ox. Kidney. White spotted kidney (WSK). Thromboembolic glomerulonephritis. The cortical surface is studded by small white foci of inflammation. It is hypothesized that bacteria from a preceding enteric infection, especially *Escherichia coli*, hematogenously shower the glomeruli, inciting a purulent inflammation. When the primary inflammatory changes resolve, chronic interstitial nephritis develops (see Chapter 7: Diseases of the Urinary System).

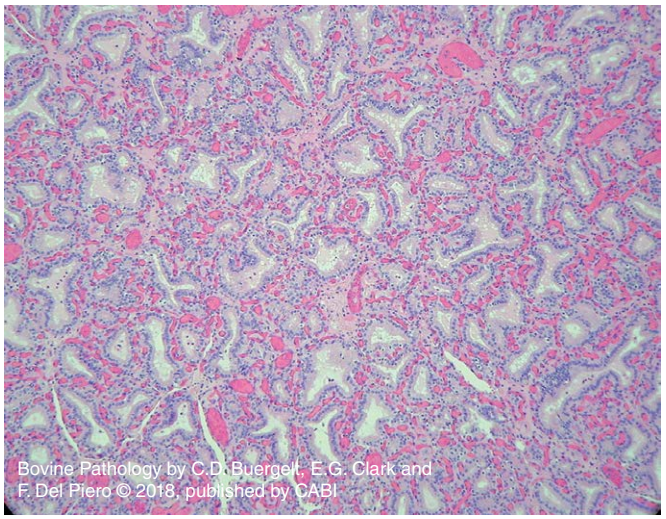

Fig. 1.89. Ox. Thyroid. Stillborn. Hyperplasia with little colloid (H&E).

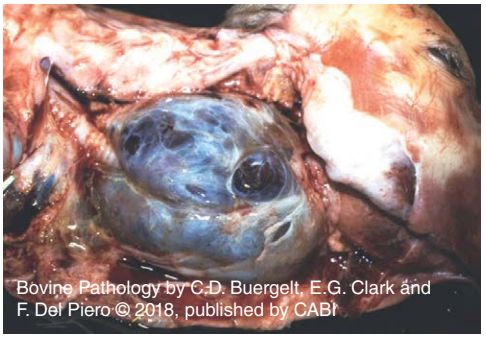

Fig. 1.88. Ox. Thyroid. Neonatal goiter. Hypotrichosis. Bilateral thyroid hypertrophy. The affected calves are born to iodine-deficient dams. In drought conditions with poor-quality feed, the thyroid glands should be collected for histologic examination, especially in stillborn and non-viable neonate calves. These will often show very little colloid production, but the cause for this condition is unknown. Vitamin E deficiency has been proposed. These affected animals grossly will not have goiter-type changes. For adult goiter, see Chapter 9: Diseases of the Endocrine System.

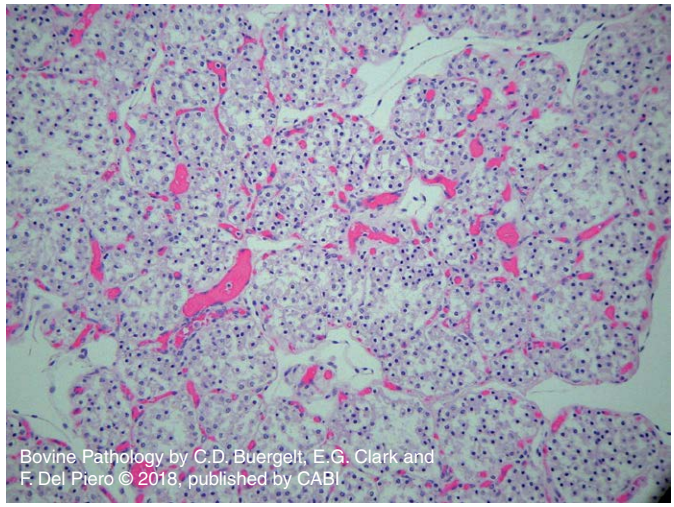

Fig. 1.90. Ox. Thyroid. Stillborn. Severe degeneration. No colloid (H&E).

1.8.1 Bovine neonatal pancytopenia (BNP)

Introduction. A hemorrhagic diathesis syndrome affecting neonatal calves emerged on the continent of Europe in 2008. The syndrome was characterized by pancytopenia and internal and external bleedings. The clinical signs were observed after the ingestion of colostrum. The history included that the dams of the affected calves received a killed vaccine against BVDV containing a novel adjuvant. Maternal alloantibodies produced against adjuvant antigen were secreted into the colostrum and, when ingested by calves, resulted in opsonization of leukocyte surface antigen, cytophagocytosis by macrophages, cytotoxicity to megakaryocytes and trilineage bone marrow hypoplasia.

Clinical signs. Dermal, nasal and rectal bleedings**.** Petechiae in visible mucous membranes.

Differential diagnoses. Bracken fern toxicity, nitrofurazone toxicity, infection with the thrombocytopenic strain of BVDV.

Fig. 1.91. Ox. Neck. Bovine neonatal pancytopenia (BNP). Dermal bleeding. Trauma to the cervical skin resulted in exudation of blood (unclotted). (Courtesy of Dr E. Lepri, University of Perugia, Italy.)

Fig. 1.92. Ox. Bovine neonatal pancytopenia (BNP). Oral mucous membranes. Hemorrhagic diathesis. Multiple petechiae are visible. Mucous membranes are pale. (Courtesy of Dr E. Lepri, University of Perugia, Italy.)

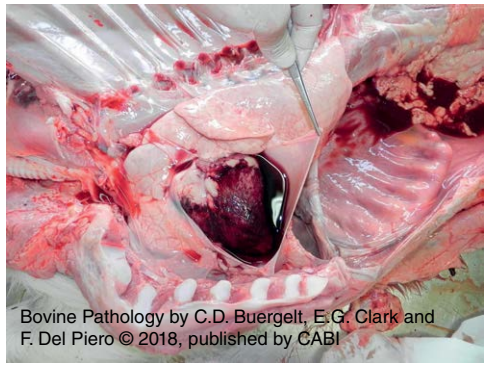

Fig. 1.93 Ox. Bovine neonatal pancytopenia (BNP). Epicardium. Effusive hemorrhage. The heart sac contains unclotted blood. The epicardial surface is tinged diffusely by blood. Tissue is pale. (Courtesy of Dr E. Lepri, University of Perugia, Italy.)

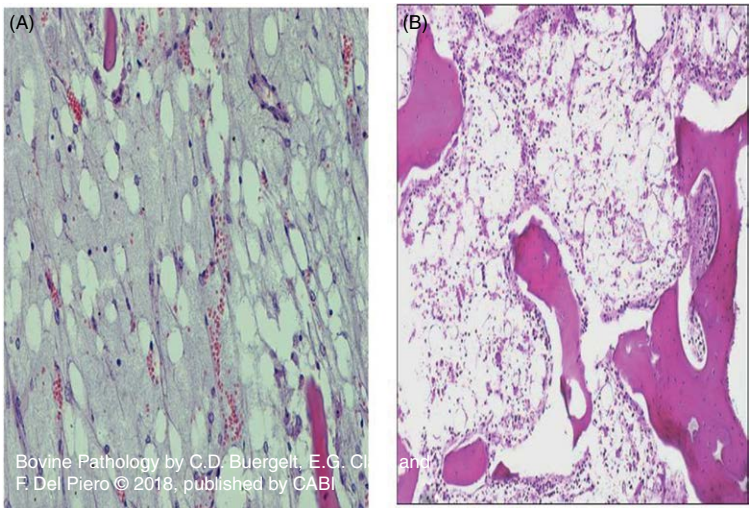

Fig. 1.94. Ox. Bovine neonatal pancytopenia (BNP). Bone marrow. Hypoplasia of hematopoietic cells. (A). All lineages are affected. (B) Normal bone marrow of unaffected animal (H&E). (Courtesy of Dr E. Lepri, University of Perugia, Italy.)

Fig. 1.95. Ox. Ears. Floppy ear syndrome. Calf with droopy ears.

1.8.2 Floppy ear syndrome

Introduction. Infection of the middle ear and tympanic bullae in pre-weaned dairy calves fed waste milk from cows suffering from *Mycoplasma bovis* mastitis (see Chapter 15: Diseases of Eye and Ear).

Clinical signs. Drooping ear, head tilt, strabismus, stiff neck, opisthotonos, purulent aural discharge.

Differential diagnoses. Meningitis, cerebellar hypoplasia, ear mites (*Raillietia auris*).

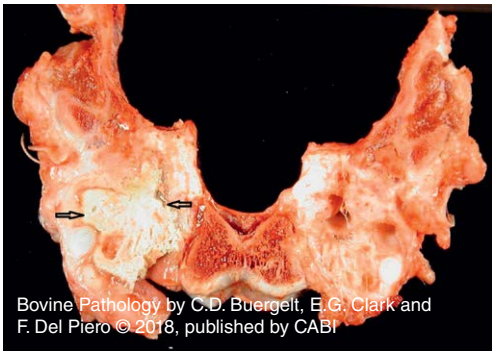

Fig. 1.96. Ox. Tympanic bulla. Floppy ear syndrome. Unilateral purulent otitis media. Arrow denotes purulent exudate in middle ear. *Mycoplasma bovis* is cultured from exudate.

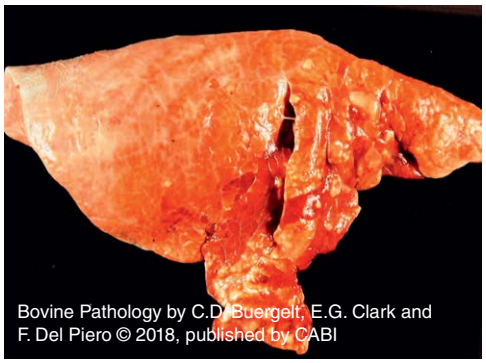

Fig. 1.98. Ox. Lung. Floppy ear syndrome. Cranioventral bronchopneumonia. Frequently, the floppy ear syndrome is associated with *Mycoplasma bovis* bronchopneumonia. It is postulated that exhaled tracheal exudate reaches the middle ear via the eustachian tube from the oral cavity. Oral ingestion of *Mycoplasma bovis* contaminated milk can also result in the colonization of tonsils and subsequent ascending infection to the middle ear.

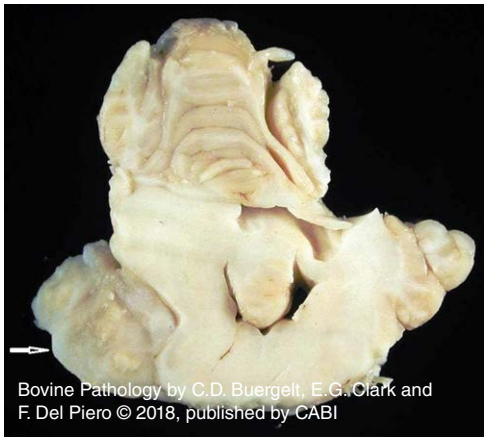

Fig. 1.97. Ox. Brainstem. Floppy ear syndrome. Purulent meningitis (arrow). The otitis media infection ascended to the meninges, producing an abscess.

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