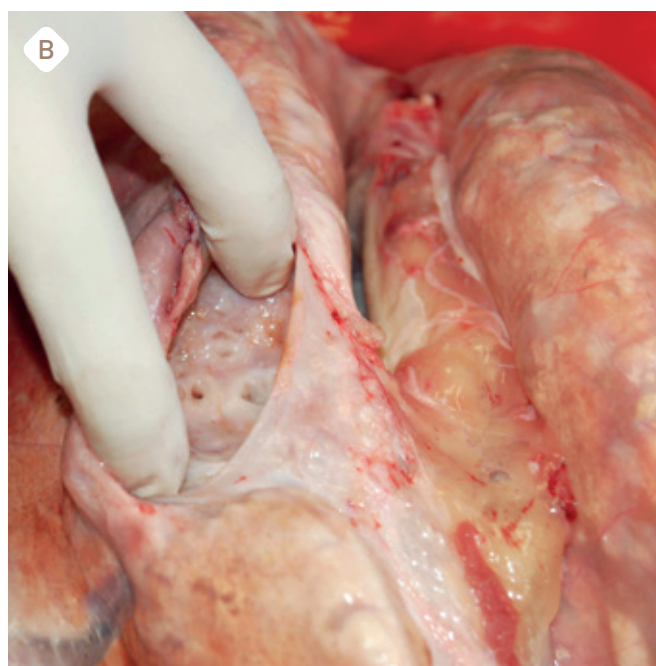
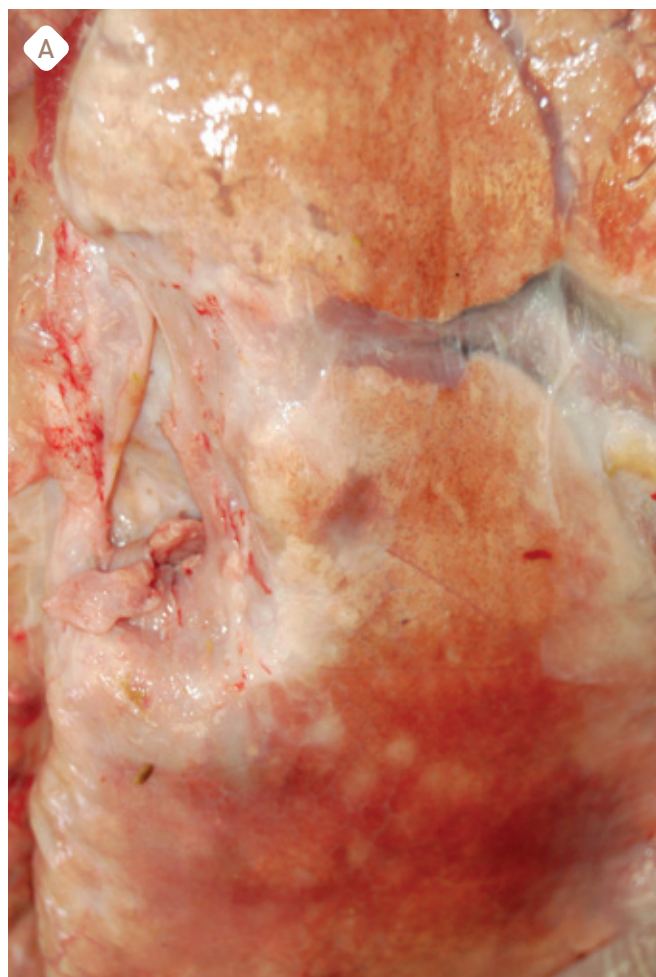


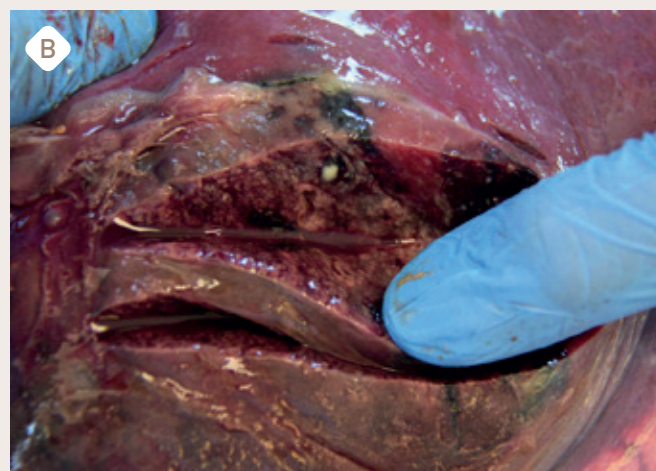
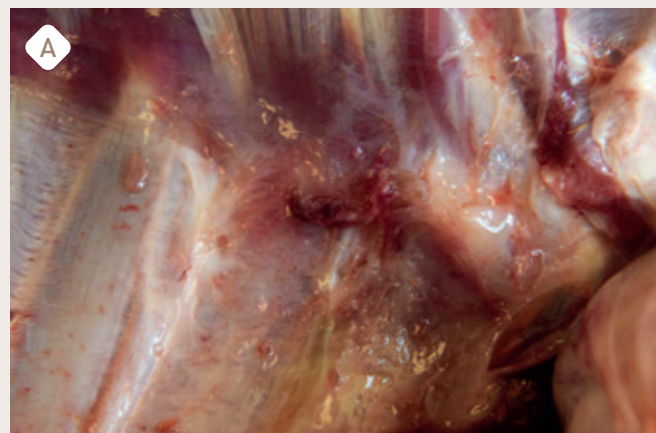


3.139. **Caverns.** It is also possible to find clean, empty, aseptic caverns of unknown aetiology (A and B).

IATROGENIC PNEUMONIA DUE TO VACCINE INJECTION IN THE LUNG



3.140. **Acute pneumonia.** The accidental injection of a vaccine directly into the lung produces acute pneumonia and abundant fibrinous pleurisy in a few hours.



3.141. **Vaccination of the injection tract.** Confirmation is easy after locating the entry point through the costal wall (A) and the pulmonary tract (B).



Digestive system disorders



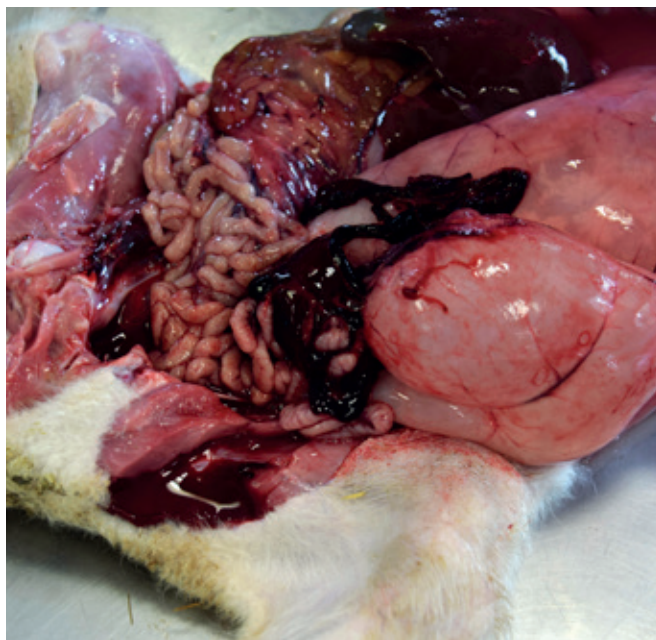
YOUNG ANIMALS

TRAUMA

Traumatic lesions commonly happen in the first days of life, especially if there is an overpopulation of animals. These traumas can cause bleeding and even perforation of the abomasum or intestine or a ruptured liver.



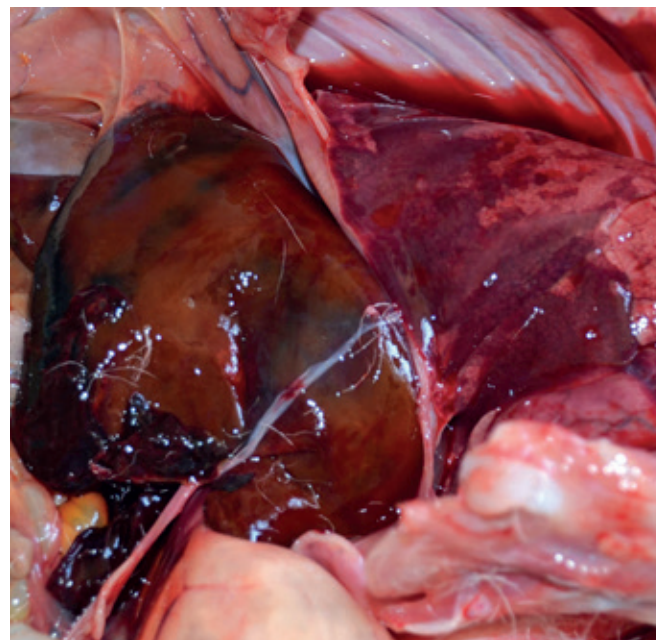
4.1. Rupture of the abdominal wall. A tear of the abdominal wall without breakage of the skin. This process is relatively frequent due to blows with the head or the horns. Less frequent by stomping.



4.2. Haemorrhages. Bleeding haemorrhage of traumatic origin in the abdominal cavity with extravasated blood and clots.



4.3. Great traumatic haemorrhage. Significant extravasation of partially clotted blood after trauma.



4.4. Liver rupture. Coagulated blood in the suprahepatic area in a lamb after a trauma by trampling of the mother.

HIGH MILK CONSUMPTION DIARRHOEA

Diarrhoea can be caused by excess milk intake, especially in single lambs born from high-producing ewes. It is also common to find this kind of diarrhoea associated with the incorrect use or poor quality powdered milk.



4.5. Alimentary diarrhoea. Alimentary diarrhoea does not impact the vitality of the affected animals in the first stages of the disease. Nursing lambs keep their appetite.



4.6. Alimentary diarrhoea. Despite diarrhoea, the animals follow their dams and continue to suckle.



4.7. Complicated alimentary diarrhoea. Contaminated and wet ground where newborns are kept can cause digestive disorders in lambs and kids. The rain has turned the soil into a quagmire, favouring bacterial growth and diarrhoea.



4.8. Faeces. The stool becomes pasty to runny, and the colour is greenish-yellow.



4.9. Abomasal overload and bacterial contamination. Lambs with abomasal overload suffer from paralytic ileum, leading to abnormal growth of the pathogenic flora found in the intestine (*E. coli* and *C. perfringens*). If the growing bacteria are gas producers, we can find abomasal bloat (A and B).



4.10. Intestinal bloat. Sometimes these secondary contaminations can also give rise to intestinal bloat.

HYPOGLYCAEMIA AND ENDOTOXIC COLIBACILLOSIS (WATERY MOUTH SYNDROME)



4.11. Poor colostrum intake. Premature lambs, especially in multiple lambings, may have difficulty getting a correct and abundant colostrum intake.



4.12. Consequences of inadequate colostrum consumption. Hypothermia also happens with weak lambs, especially in cold seasons.



4.13. Hypersalivation. Animals are hypoglycemic and show salivary hypersecretion (drooling).



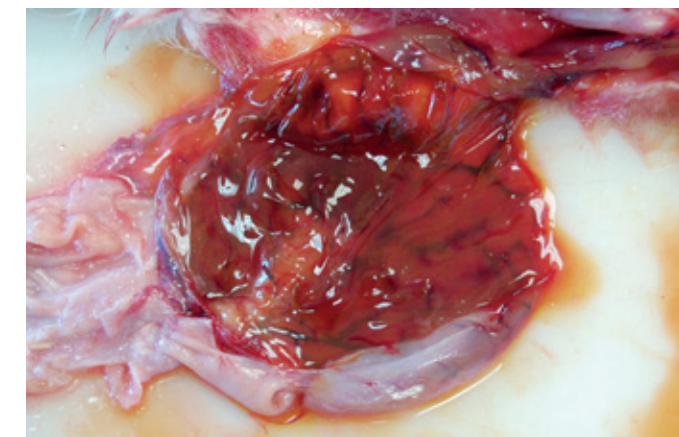
4.14. Lethargic lambs. Affected lambs are dull, lethargic, depressed and reluctant to suck.



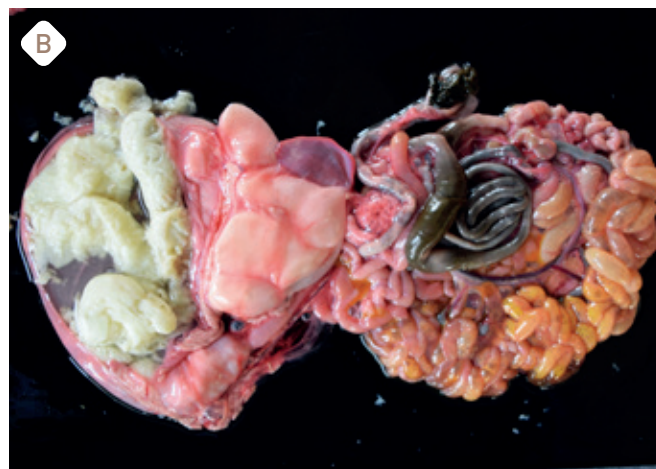
4.15. Wet perioral area. Death occurs in the first 24-72 hours of life. The perioral area and the floor where the lamb rests are damp from drooling.



4.16. Abomasum. At necropsy, we can find completely empty abomasum.



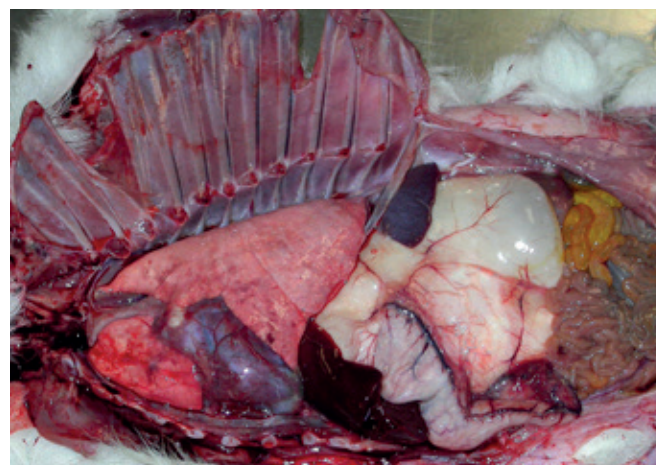
4.17. Abomasitis. The abomasum may show inflammation, with mucous content (saliva) and absence of colostrum.



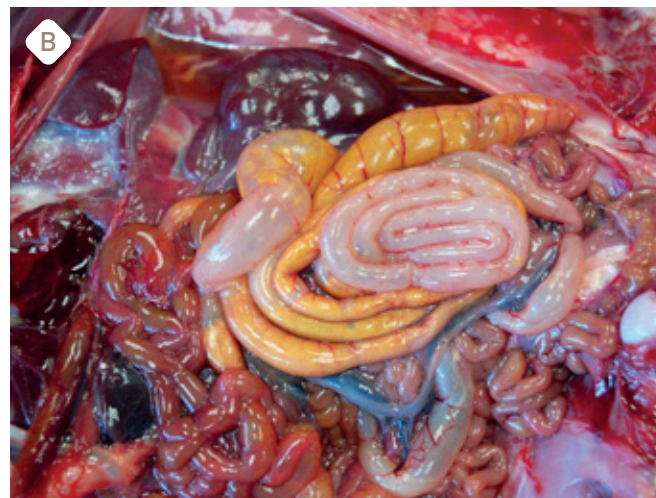
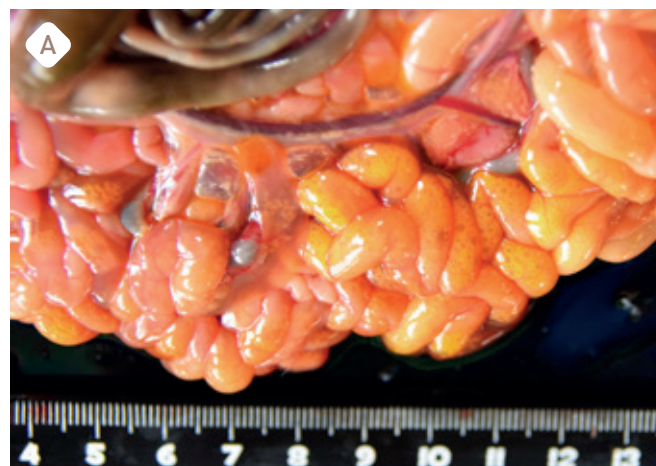
4.18. Lack of sufficient colostrum intake. A certain amount of milk can be found in the abomasum at necropsy (A), but in this case it was insufficient to ensure a correct elimination of meconium (B).



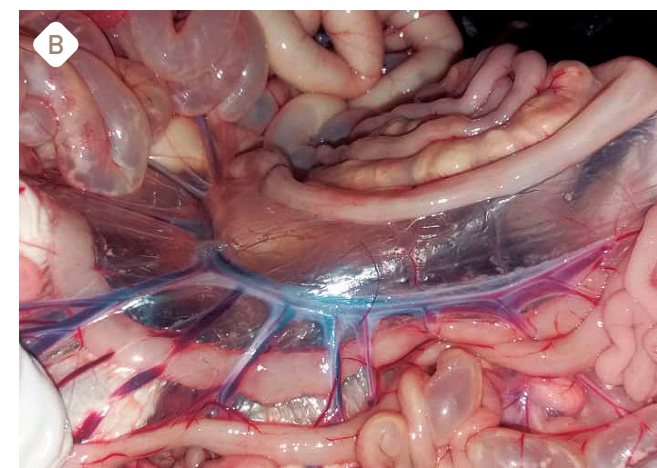
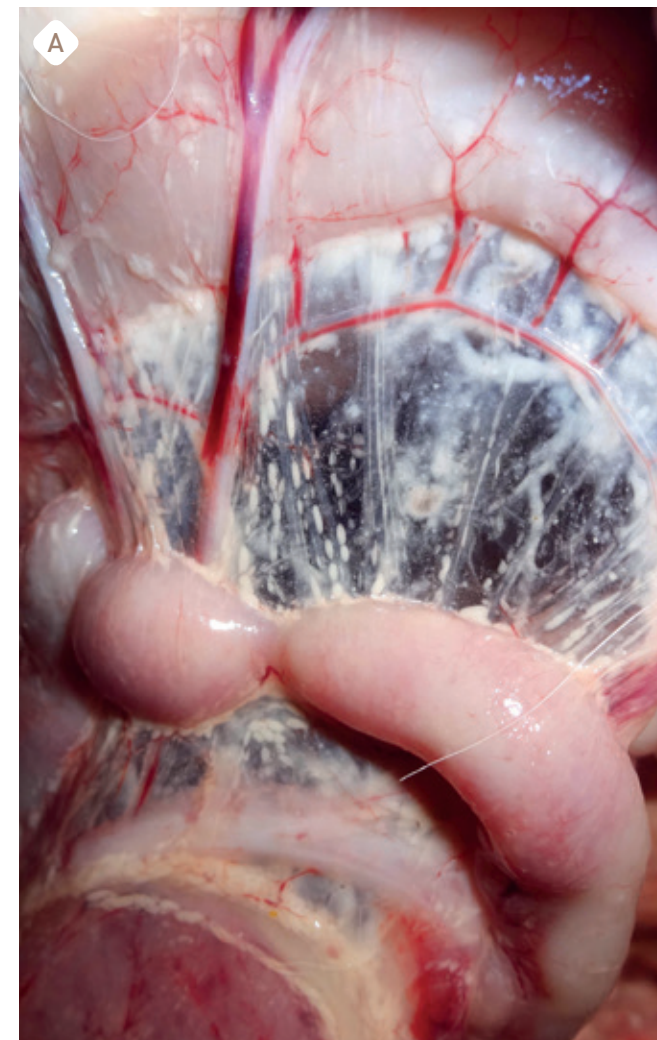
4.19. Milk in the abomasum. Sometimes we find the abomasum full of milk, but the lamb has developed enteritis due to insufficient colostrum intake.



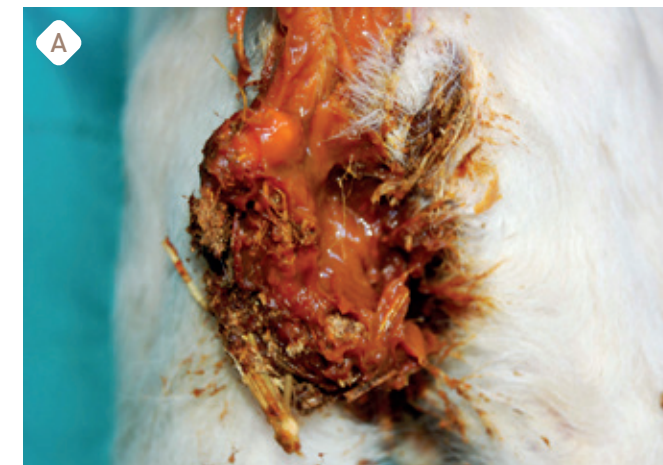
4.20. Enteritis due to *E. coli*. Lambs with insufficient colostrum intake look for food on the floor, and they acquire new infections that cause digestive disorders such as enteritis.



4.21. Catarrhal enteritis. The intestinal loops affected by enterotoxigenic *E. coli* show catarrhal enteritis (A and B).



4.22. Hypoglycaemia and lack of reserves. There is insufficient absorption of nutrients, and the animal stays without energy reserves. Cloudy mesenteric appearance in a lamb which is caused by milk absorption (A) and the clear mesenteric appearance in a lamb that has died of starvation (B).



4.23. Right colostrum intake. After a correct colostrum intake, the lambs show pasty orange-coloured stools that sometimes remain attached to the perianal area (A and B).



4.24. Meconium making defecation difficult. Occasionally, the adhered meconium makes it challenging to defecate correctly.



ENTERIC COLIBACILLOSIS



4.25. **High mortality.** Colibacillary diarrhoea is commonly the first cause of death in lambs and kids in the first week of life (A and B).



4.26. **Colibacillary diarrhoea.** It produces a yellow diarrhoea (A and B) that can appear individually or collectively.



4.27. **Collective colibacillary diarrhoea.** The collective form is the most frequent presentation of this process.



4.28. **Faeces aspect.** The appearance of the faeces is pasty and of intense yellow colour.



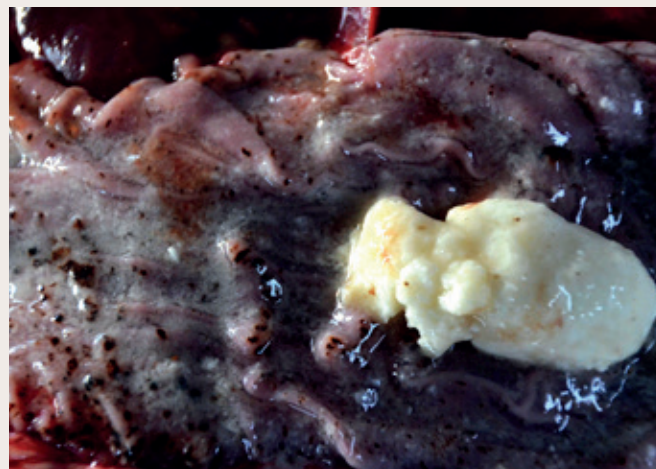
4.29. **Colibacillary diarrhoea after several days of the clinical picture.** If the animal is not treated and does not die, enteritis can evolve into intestinal bleeding, giving the stool a dark brown colour.



4.30. **Overloaded abomasum.** Postmortem findings of animals affected by colibacillary enteritis usually show a dilated abomasum with high milk content (A and B).



4.31. **Abomasum with large milk clots.** The opening of the abomasum shows abomasitis and, usually, large milk clots that are difficult to be digested.



4.32. Abomasal petechiae. When removing abomasal content, petechiae can be seen in the surface of the abomasum that stains its content. Sometimes, large milk clots can also be found together with the petechiae.



4.34. Intestinal fluid in colibacillary diarrhoea. The opening of the intestine lets the bright yellow contents flow.



4.33. Catarrhal enteritis. The intestine shows catarrhal enteritis, with reactive Peyer's patches, with very marked blood vessels and yellowish contents (A and B).



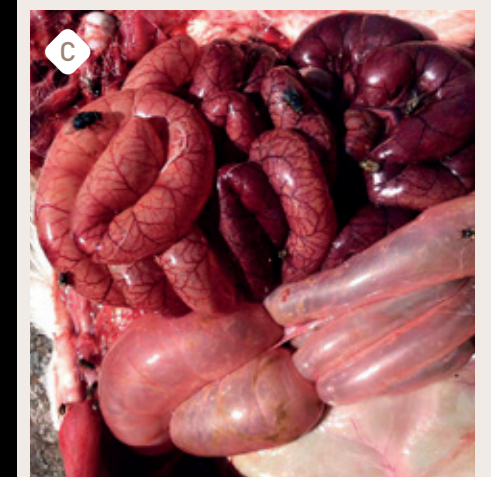
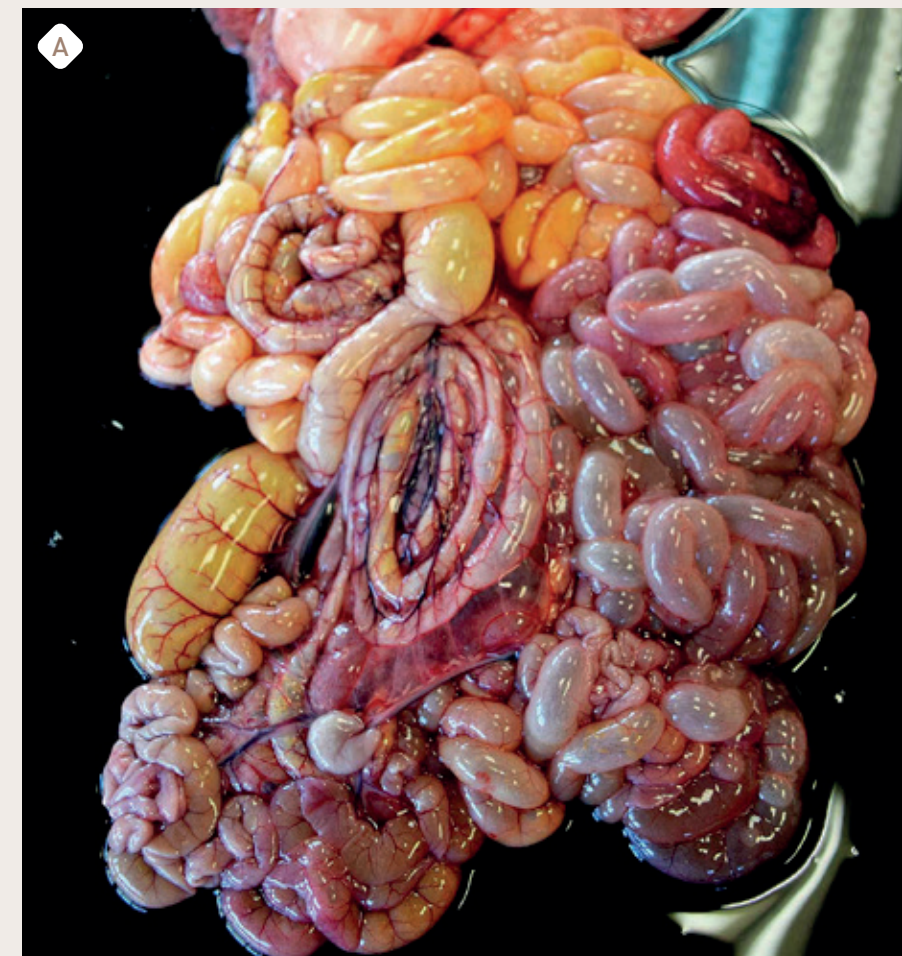
4.35. Intestinal loops affected by catarrhal and haemorrhagic enteritis. In some intestinal loops, catarrhal enteritis evolves to haemorrhagic, with more bloody content that stains the previous yellow (A and B).



4.36. Catarrhal-haemorrhagic enteritis. Haemorrhagic enteritis can affect almost the entire digestive package, especially at the end of the first week of life and the beginning of the second. At this time, *Escherichia coli* and *Clostridium perfringens* type B predominate in bacterial isolates.



4.37. Mixed diarrhoea caused by *E. coli* and *Cryptosporidium*. Throughout this period, the lamb may be simultaneously infected by *Cryptosporidium* sp. In these cases, the affected intestinal loops contain a more greenish material, easily visible when the intestine is opened.



4.38. Haemorrhagic enteritis. Over time, enteritis found is predominantly haemorrhagic, and *E. coli* is less frequently isolated and *Cl. perfringens* type B is usually found, and even type D in animals that reach the third week of life (A-C).



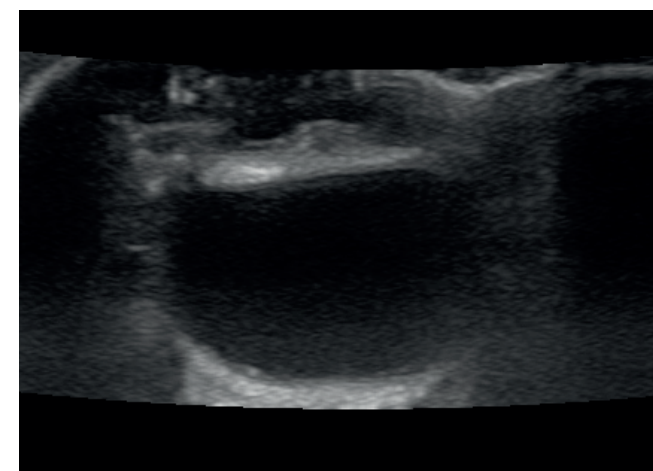
SEPTICAEMIC COLIBACILLOSIS



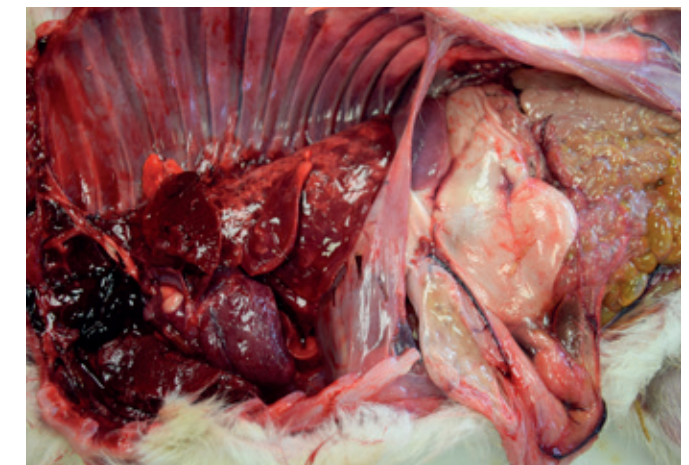
4.39. Clinical signs. Some animals affected by enteric colibacillosis may subsequently suffer from septicaemic colibacillosis. These animals show depression, incoordination and stagger (A), sometimes suffer colic pain (B), recumbency (C) and can die in a short time (D).



4.40. Fibrin deposition in the anterior chamber of the eye. In some cases of colibacillary septicaemia, it is possible to find a floc of fibrin floating in the anterior chamber of the eye (A and B). This is a very characteristic clinical sign of this disorder.



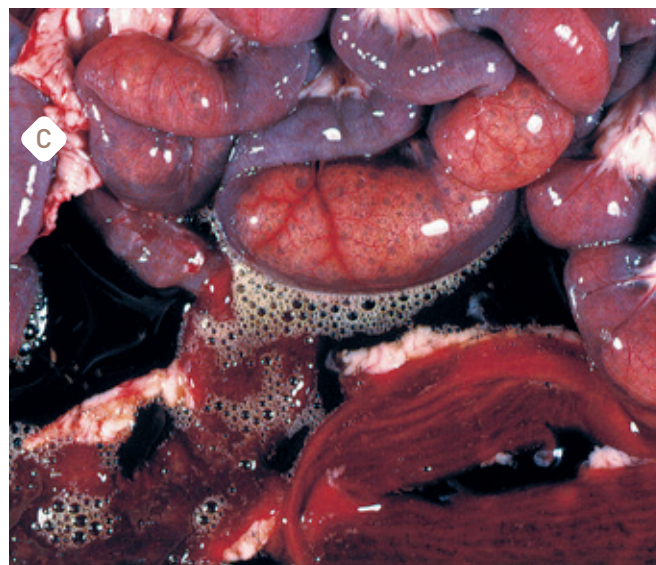
4.41. Ultrasound of the eye with fibrin deposition. Ocular ultrasound shows the fibrin floc floating in the anterior chamber. Microbiological analysis confirms the result.



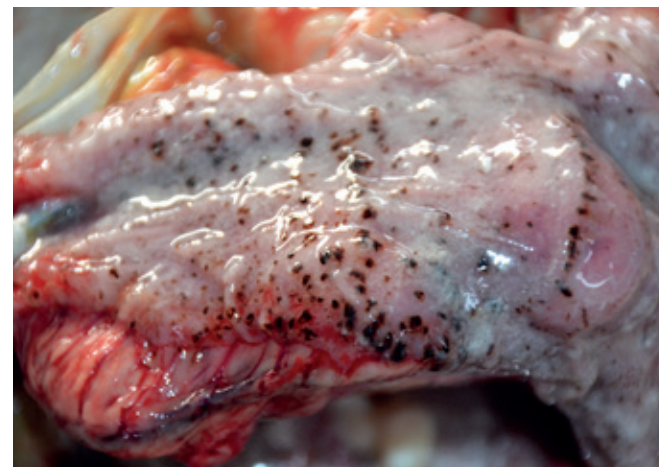
4.42. Catarrhal enteritis and pneumonia. Sometimes, postmortem findings may show a digestive system with catarrhal enteritis coexisting with a pneumonic disorder. In this case, a more acute and intense reaction in the lymph nodes can clarify the digestive or pulmonary origin of septicaemia.



4.43. Catarrhal enteritis in a twelve-days-old lamb. Occasionally, in slightly older animals, we find enteritis but without the yellow content of colibacillary diarrhoea (A and B). The microbiological or toxin study clarifies the colibacillary or clostridial origin of the lesion.



4.44. Digestive haemorrhagic content. Haemorrhagic fluid content in the abomasum (A and B) and intestine (C) can also be found.



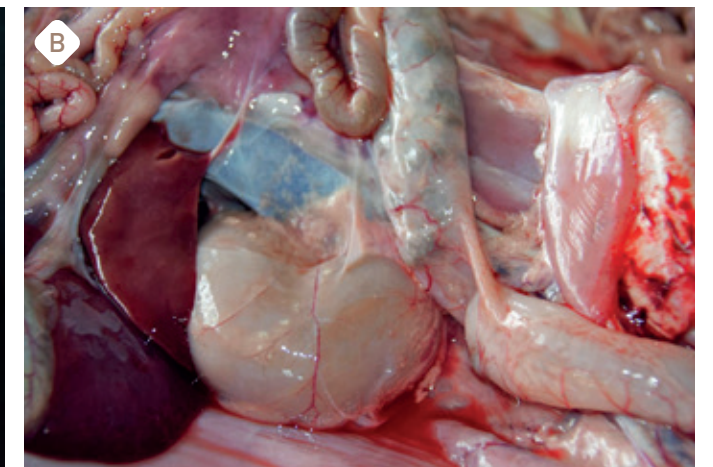
4.45. Petechiae in abomasum. The abomasum can also be full of milk and with more or less abundant petechiae.



4.46. Disseminated petechiae. Petechiae can also be found in other locations such as the rumen (A), thymus, kidneys or heart. In addition, congestion, oedema and even some petechiae can be seen in the brain (B). The brain is the chosen location to take samples to confirm septicaemic colibacillosis.

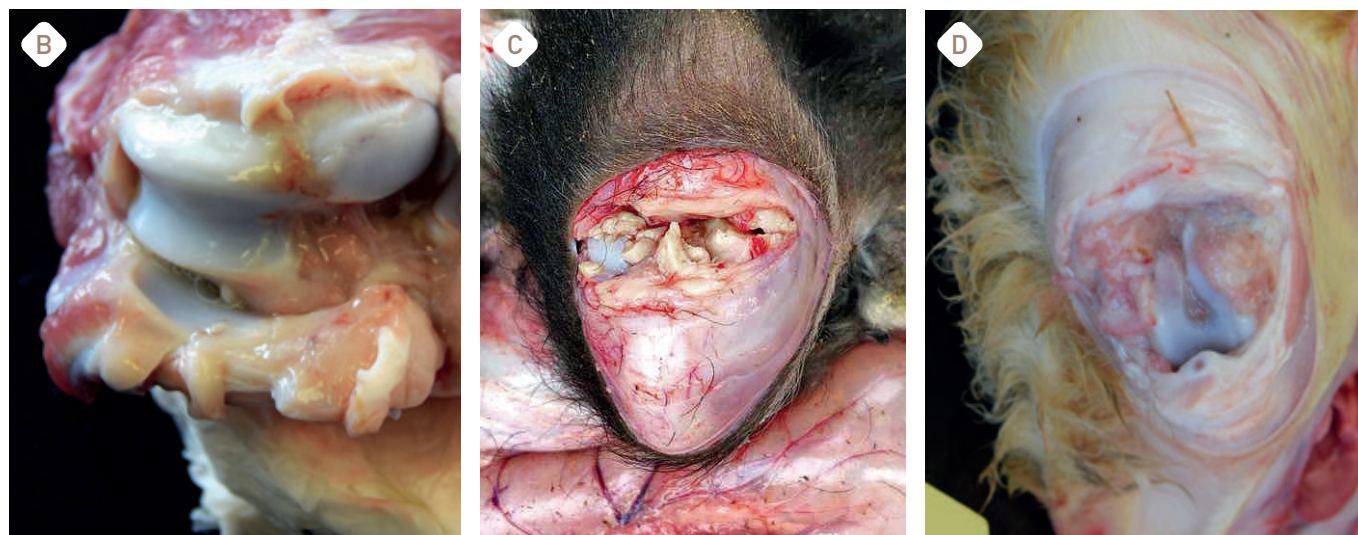


4.47. Nephrotic kidney. Enlarged clear kidneys can be seen in some cases of long-term colibacillosis or after having overdosed treatments (A and B).



4.48. Polyarthritis. Some lambs that survive the process end up presenting polyarthritis (A and B).





4.49. Fibrinous polyarthritis. Postmortem findings show abundant clean and clear yellowish synovial fluid (A) or different amounts of fibrin in the affected joints (B-D). Pus is not usually present.

HAEMORRHAGIC ENTEROTOXAEMIA

Affected lambs die from haemorrhagic enteritis. The perianal area is stained with red (acute) or dark (subacute) blood. Dehydration can be seen in the skin and the sunken eyes. Postmortem findings show haemorrhagic enteritis. It usually affects one to three-days-old lambs.



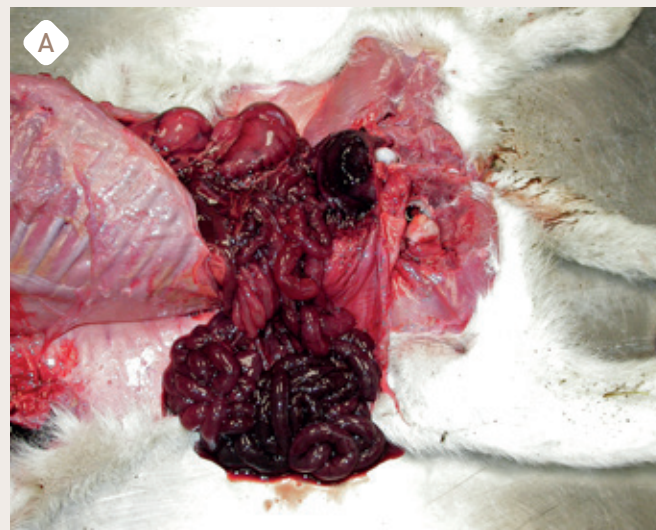
4.50. Hyperacute haemorrhagic enterotoxaemia. Occasionally, *C. perfringens* type A and C can be isolated. These recumbent lambs affected by *Clostridium perfringens* type C can evolve rapidly, even without showing haemorrhagic diarrhoea.



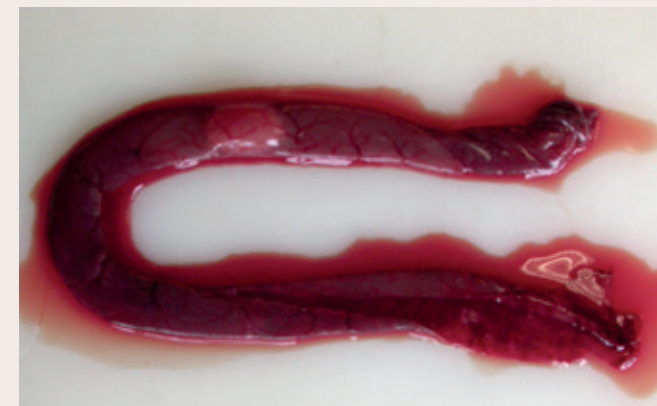
4.51. Death from acute haemorrhagic enterotoxaemia. Mortality in haemorrhagic enterotoxaemia tends to be high, and the animals show the perianal area stained with bloody stools, red in the acute form (A) and black when they survive some more days (B and C).



4.52. Dehydration. Dehydration is usually rapid and severe (A-C).



4.53. Digestive postmortem findings. The necropsy shows a bloody digestive tract (A), both in the lumen (B) and in the serosa of the intestine (C).



4.54. Haemorrhagic content. Haemorrhagic fluid in the intestinal lumen.



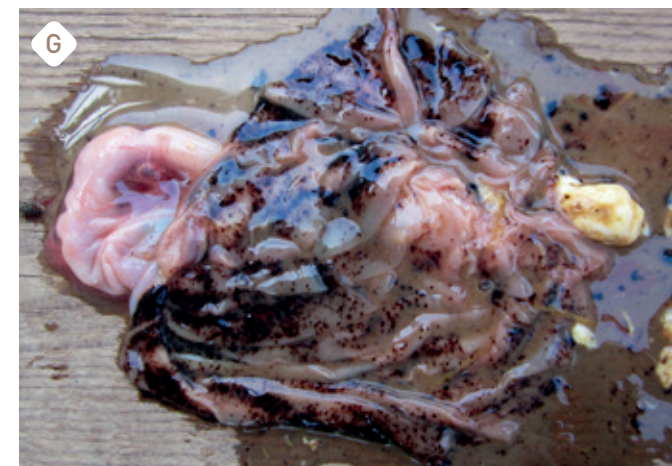
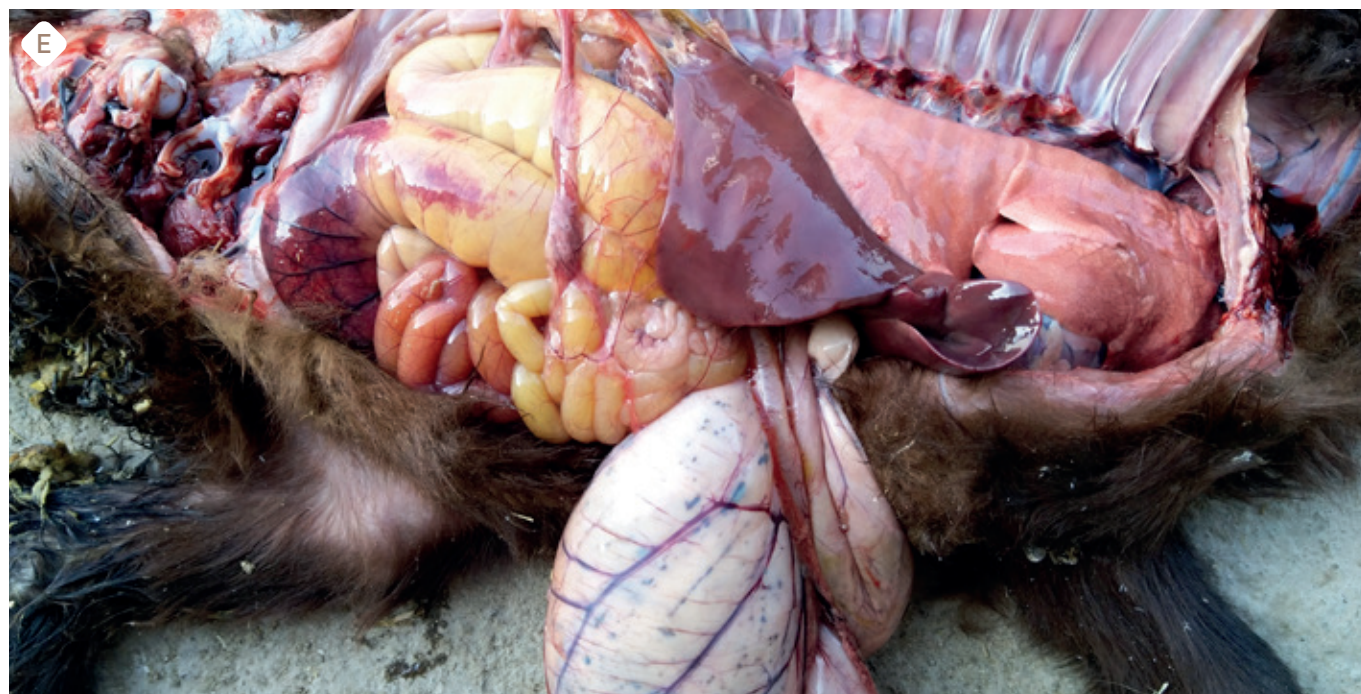
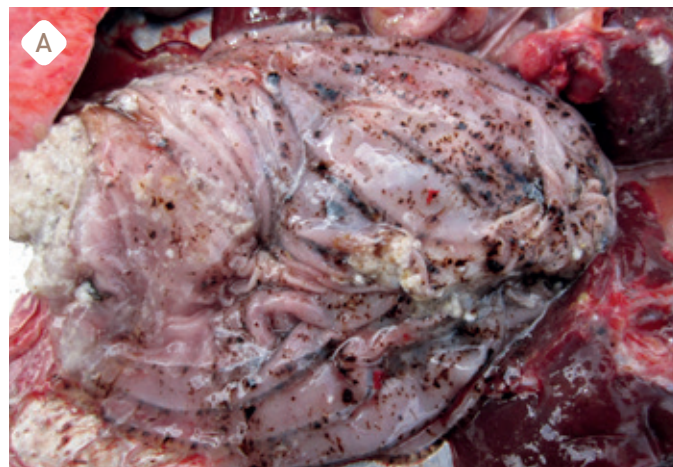
4.55. Lymphadenitis of the mesenteric lymph nodes. Acute lymphadenitis in the mesenteric lymph nodes.

SALIVARY ABOMASUM DISEASE (SAD)

Salivary abomasum disease affects lambs and kids from 3 to 20 days of age and neonates born in the second half of the lambing season are most commonly affected.



4.56. Clinical signs. SAD is characterised by lethargy, absence of sucking, hindlimbs weakness, increasing abomasal/abdominal distension, dehydration and frequent recumbency. Without treatment, the condition progresses to lateral recumbency with abdominal pain, bruxism and death within one to four days.



4.57. Postmortem findings. At necropsy, multiple small abomasal haemorrhages with blood clots ('coffee grains') (A and B) are visible on both the serosal (C-E) and mucosal surfaces (F-J).



4.58. Abomasal haemorrhages and milk clots. Sometimes, milk clots and mild or severe haemorrhages are found in the abomasum (A-D).

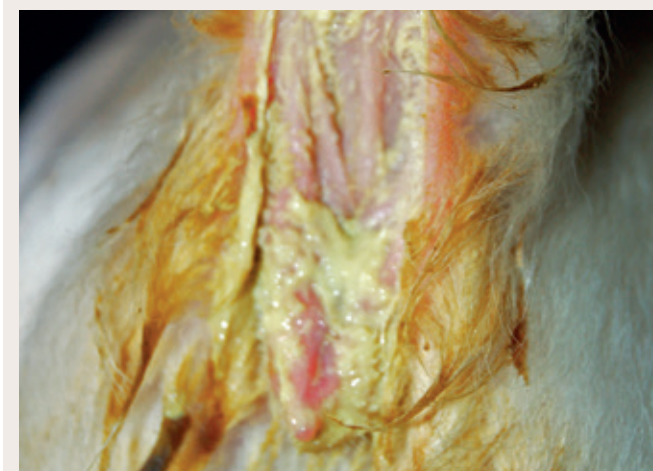


LAMB DYSENTERY

Along with colibacillary diarrhoea, lamb dysentery is one of the disorders that causes the highest mortality rate in young animals. Lamb dysentery is caused by *C. perfringens* type B.



4.59. Lamb dysentery. Lamb dysentery affects animals of the second and third week of life. It usually occurs collectively, affecting a large number of animals (A and B).



4.60. Acute lamb dysentery. At the beginning of the process, white diarrhoea staining the perianal area can be seen.



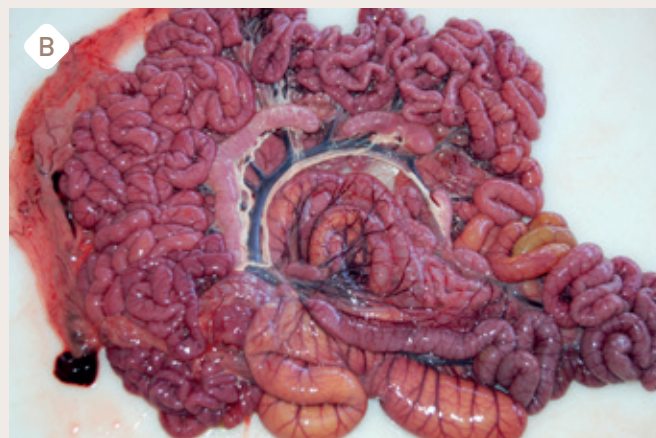
4.61. Subacute lamb dysentery. When the lambs are not recovered after the first stage of the disease, they are seen as dull, depressive and dehydrated. Then diarrhoea begins to turn dark in colour due to the presence of digested blood in faeces.



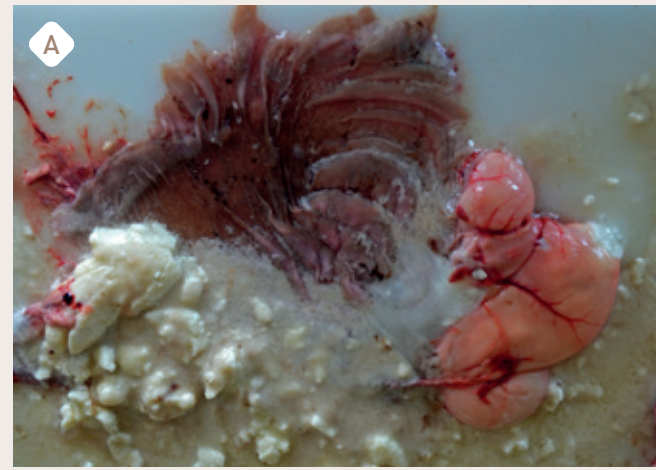
4.62. White pasty faeces. If we visit the farm at the beginning of the process, white pasty faeces can be seen on the pen's ground, which looks like drops of plaster (A and B).



4.63. Abomasum overload. As in colibacillosis, lamb dysentery usually begins with the abomasal overload that favours the growth of *Clostridium perfringens* type B and the production of β toxin.



4.64. Abomasum and intestine. In addition to the abomasum, the intestinal loops are dilated and full of whitish content. In one to two-week-old animals, this white content can coexist with yellowish coloured loops, affected by colibacillary diarrhoea (A and B). At this stage, enteritis still does not present haemorrhages.



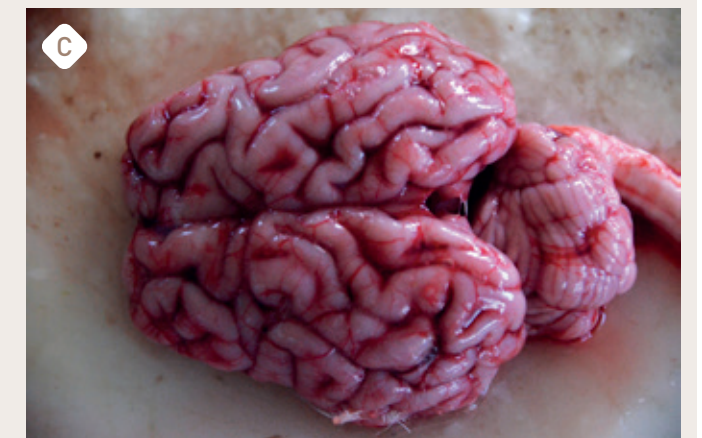
4.65. Haemorrhagic abomasitis. The abomasum usually contains large milk clots and small petechiae at the early stages (A). If the process continues, the abomasal content becomes completely black and bloody (B).



4.66. Haemorrhagic enteritis. As the disorder evolves, catarrhal enteritis turns into haemorrhagic enteritis (A and B).



4.67. Blood clots in the intestine. The intestinal content can have so much blood that it forms clots inside.

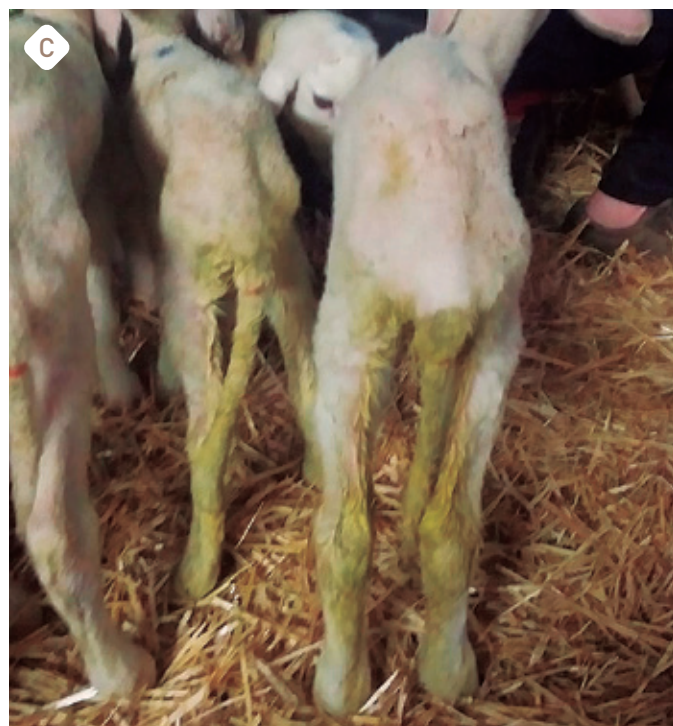
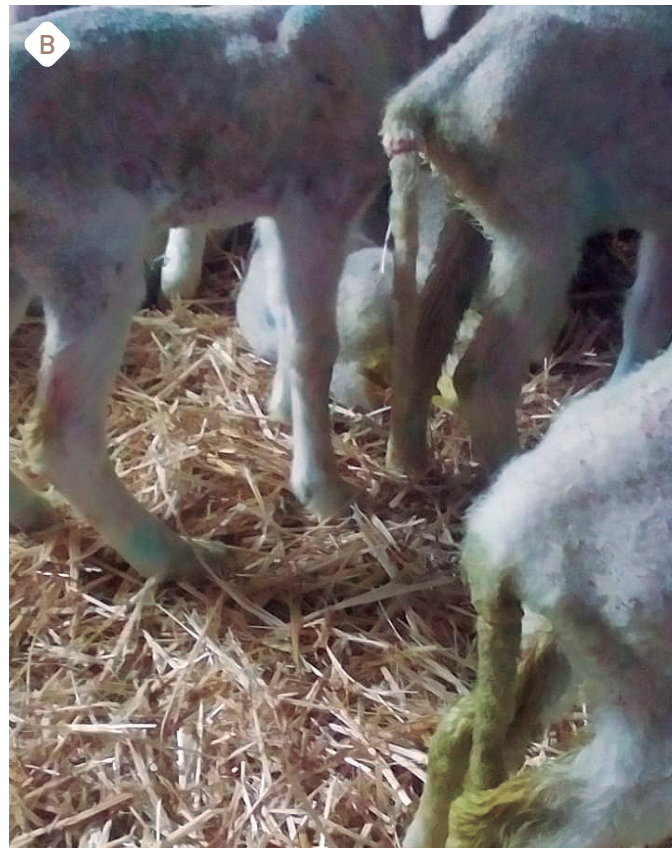
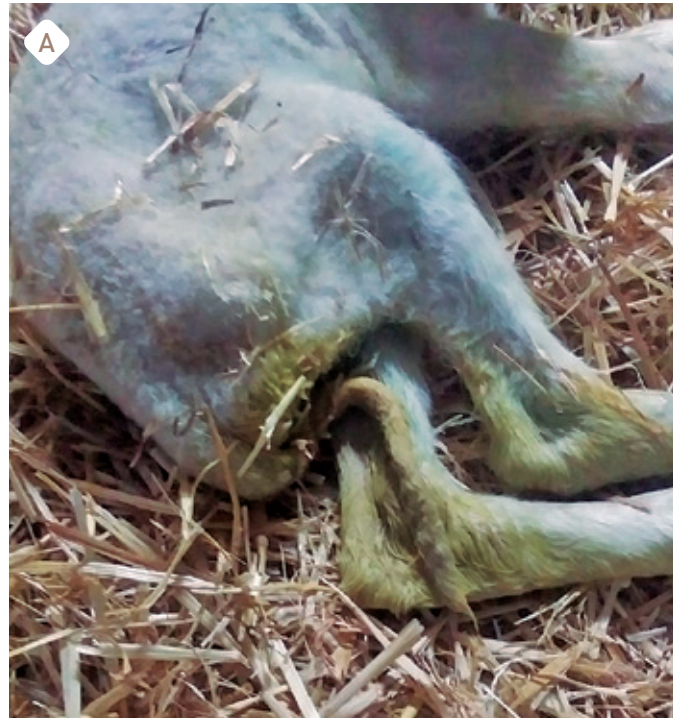


4.68. Older than two-three-week-old lambs. When dysentery affects lambs older than 2-3 weeks, *Clostridium perfringens* type B can act simultaneously with type D and present dilated and air-filled abomasum (A) and intestinal loops (B), pulpy kidney and congestion and cerebral oedema (C).



CRYPTOSPORIDIOSIS

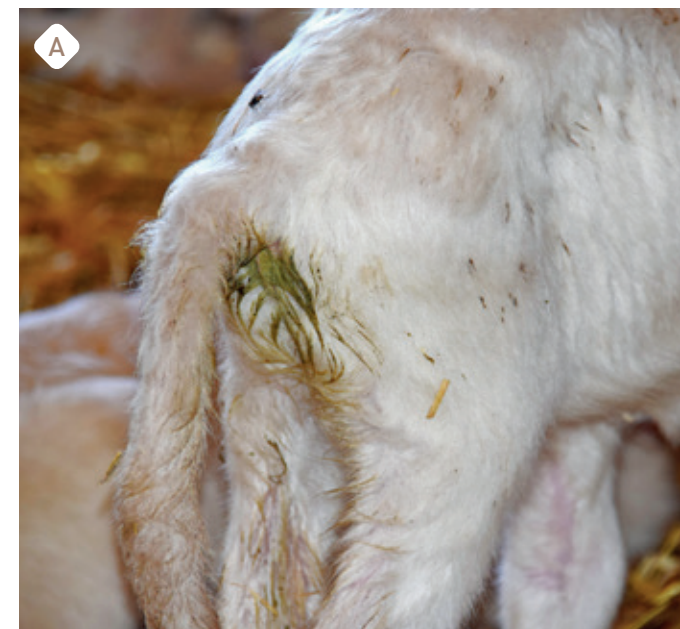
Caused by *Cryptosporidium parvum*.



4.69. Clinical signs. *Cryptosporidium parvum* affects lambs of any age and even adult animals. In young animals, it causes fluid or watery diarrhoea that leads to dehydration. Diarrhoea usually affects collectively. When other microorganisms such as *Clostridium perfringens* or *Escherichia coli* act together, *C. parvum* can enhance its pathogenic power and increase mortality (A-D).



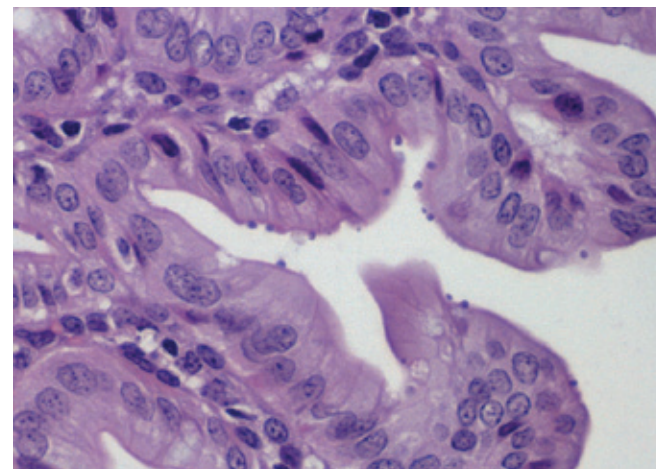
4.70. Watery diarrhoea. *Cryptosporidium parvum* damages the intestinal wall, making water absorption challenging and favouring the development of very fluid diarrhoea, usually light greenish.



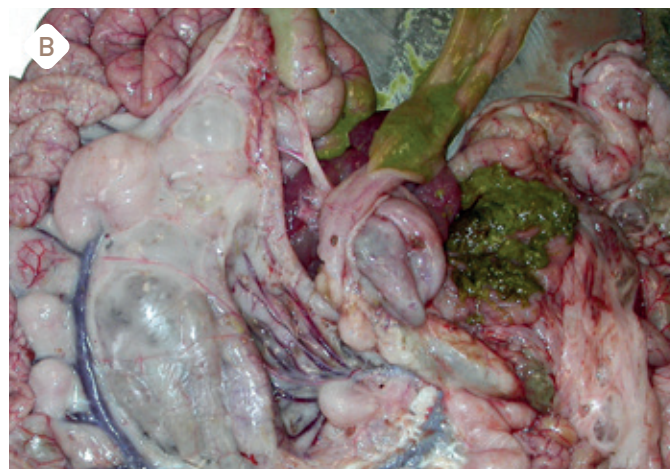
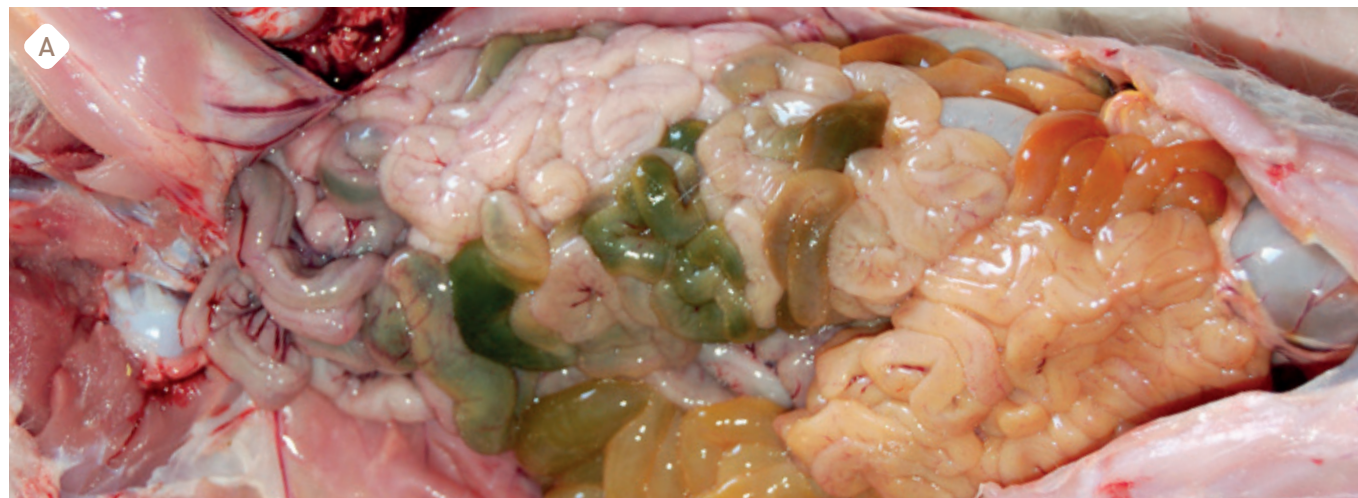
4.71. Diarrhoea caused by *Cryptosporidium parvum*. Lambs affected by cryptosporidiosis suffer from diarrhoea that stains the perianal area and hocks of a light green colour (A-E).



4.72. Appearance of faeces. Sometimes, fluid greenish diarrhoea can be seen on the farm floor. This may contain mucus, poorly digested milk and even remnants of the mucosa.



4.74. Microscopic image of *Cryptosporidium parvum* in the intestine. Cryptosporidia line the surface of intestinal villi (H&E).



4.73. Enteritis caused by *Cryptosporidium parvum*. Acute catarrhal enteritis with loops affected to varying degrees and very fluid and greenish content can be seen at necropsy (A). In chronic processes or accompanying coccidiosis, we can find chronic enteritis with greenish content (B and C).

COCCIDIOSIS

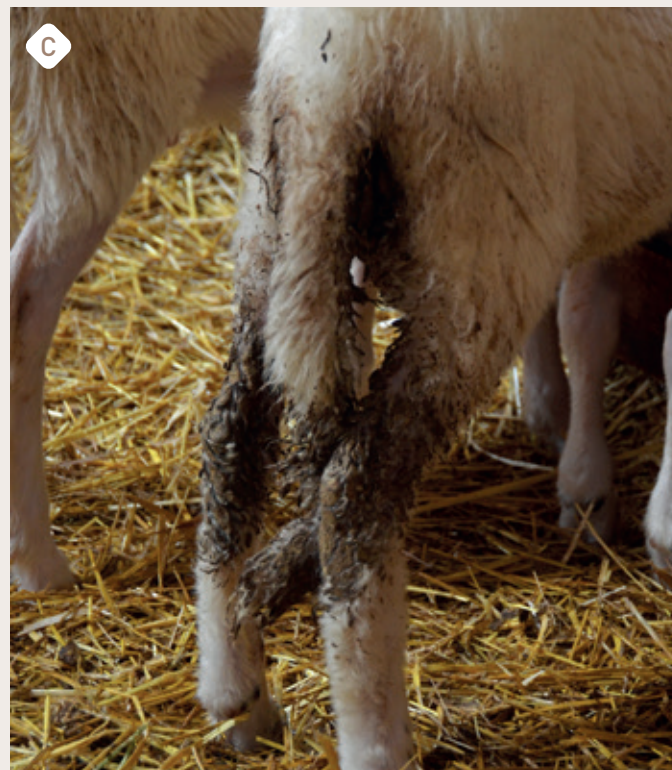
Coccidiosis is one of the most critical diseases in fattening period, causing clinical and subclinical pictures. Although it does not lead to high mortality in most farms, coccidiosis produces relevant economic losses associated with poor production rates of subclinically affected lambs and kids. As this parasite produces a relevant immunosuppressor effect, it potentiates the presence of other diseases such as small ruminant respiratory complex (SRRC).



4.75. Clinical signs. When a group of lambs show signs of coccidiosis, almost all the animals are affected, although some of them will not show clear clinical signs (A and B coccidiosis in lambs, C and D coccidiosis in kids).



4.76. Number of affected lambs. When we explore a group of lambs, a good strategy to diagnose the number of affected lambs is to clap and make them run. When they start running, the lambs lift their tails, and we can see the spotted perianal area.

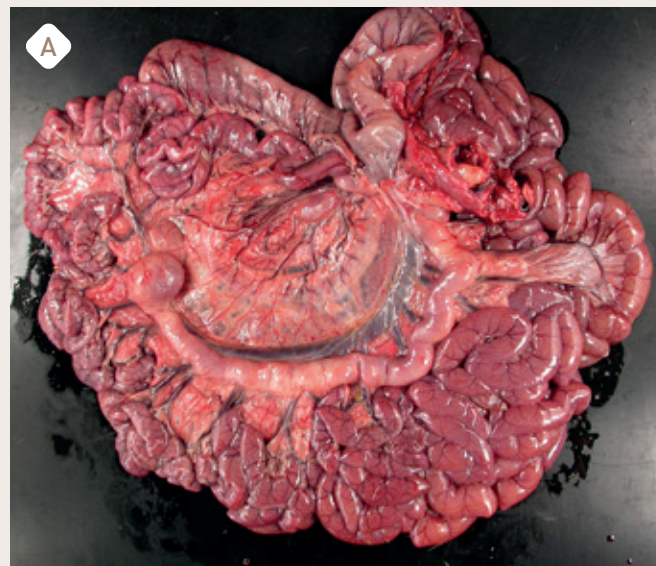


4.77. Clinical variability of coccidiosis. Lambs more severely affected show a worse growth and clinical appearance than the least affected (A and B). Wool or hair show poor appearance. Pasty, greenish-brown to greyish stools stain the perianal area down to the hocks (C), even forming large concretions on the sides of the perianal area (D and E). The moisture of faeces and friction from concretions can inflame the perineum, especially the vulva (F). Occasionally, coccidiosis can be the primary cause of death in animals (G).





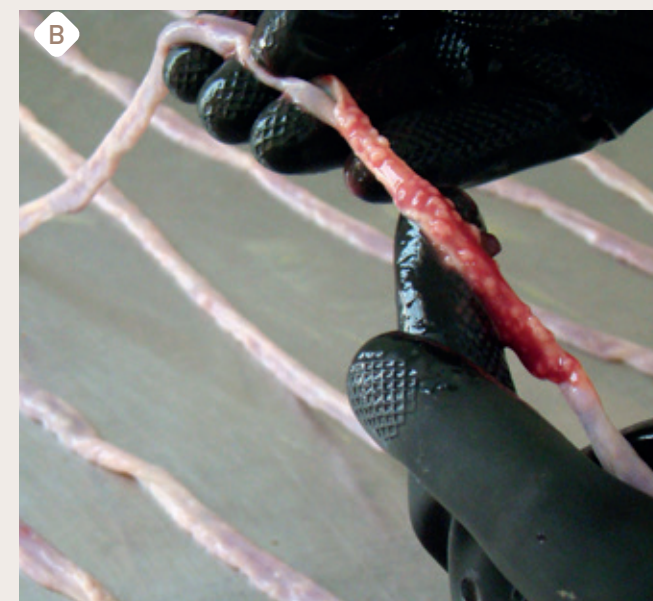
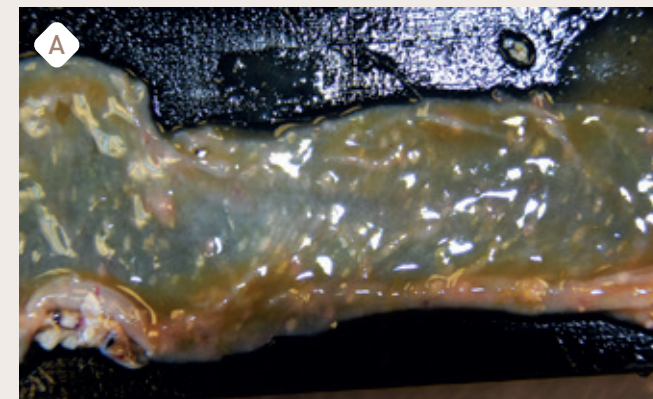
4.78. Faeces appearance. The faeces of affected animals can vary according to the evolution of the parasite in the intestinal mucosa. We can find normal to pasty or diarrheal stools (A-C). The pasty faeces, which stick to the sole of the boots (D), together with the clinical presentation and the lamb age, give a relatively straightforward diagnosis of the process.



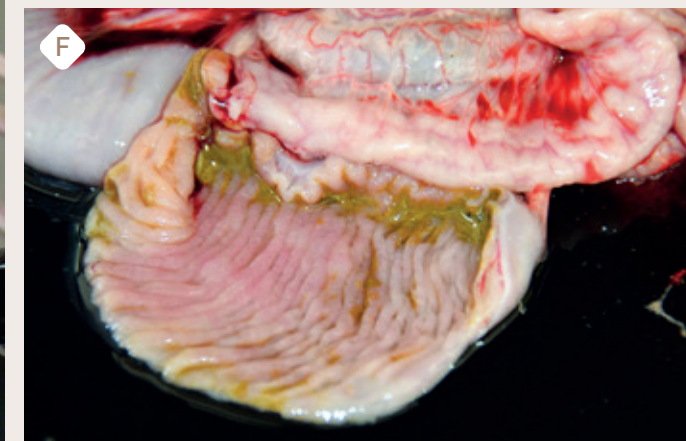
4.79. Subacute catarrhal enteritis. Coccidiosis causes subacute catarrhal enteritis (A-C) with enlargement of mesenteric lymph nodes.

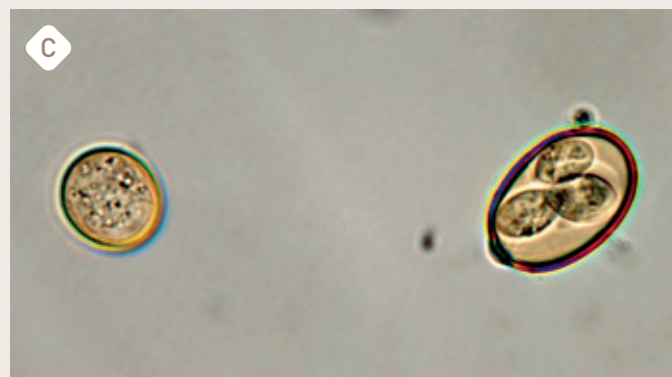
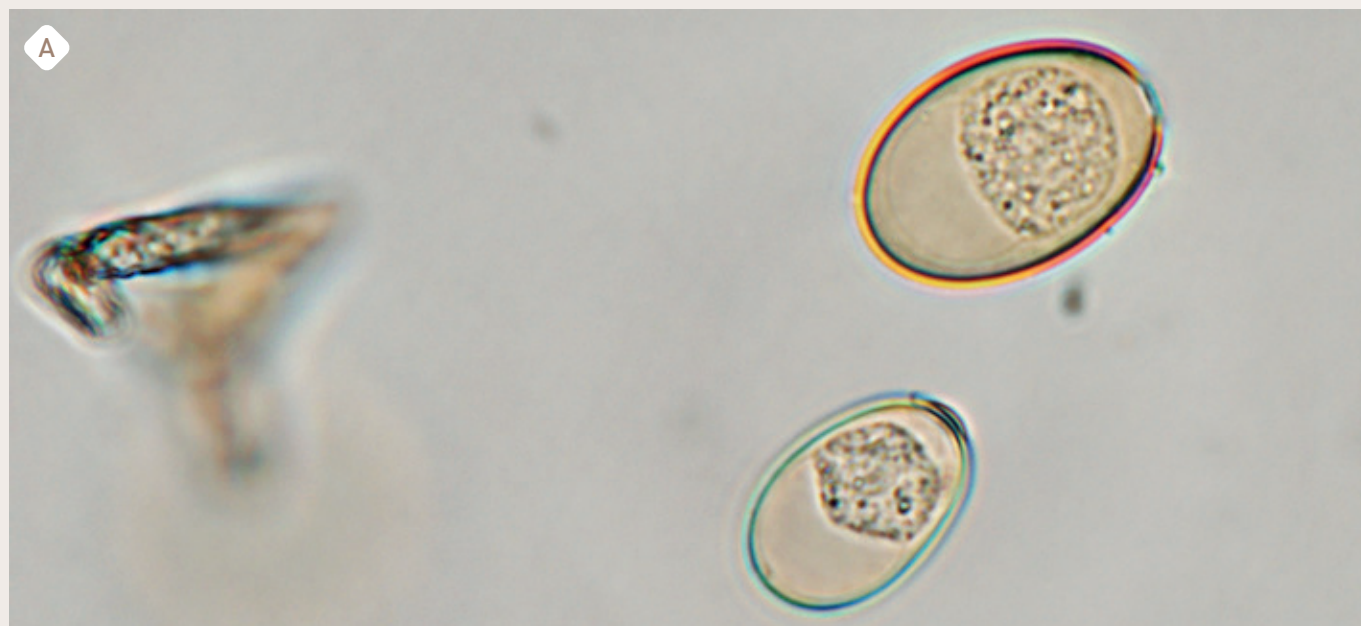


4.80. Chronic catarrhal enteritis. The evolution of the disease leads to chronic enteritis, with hypertrophied lymph nodes as in the previous clinical presentation.



4.81. Intestinal mucosa. As in the clinical findings, the degree of affection of the intestinal mucosa can vary widely from atrophic enteritis (A) with an almost transparent intestinal wall, sometimes dotted with small nodules visible through the serosa (B and C), to a hypertrophic intestinal mucosa (D-F).





4.82. Oocysts. The rapid confirmation of the process is completed with the observation under the microscope of the oocysts obtained in a scraping of the intestinal mucosa or by flotation of the faeces (A-C). Commonly, different species with different pathogenic power are found in the same animal.

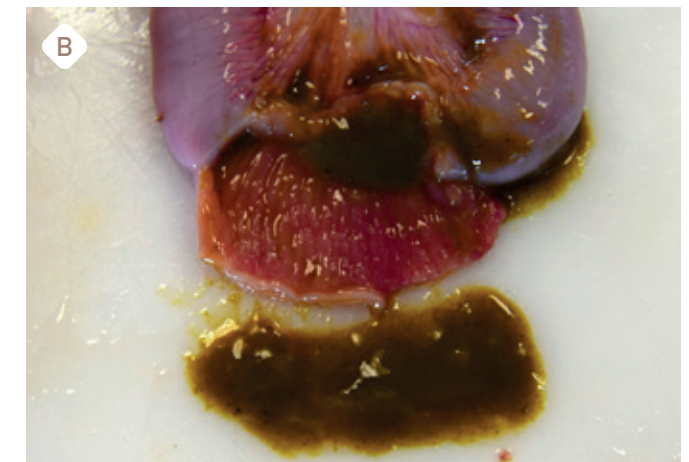


4.83. Complications of coccidiosis. As a result of increased digestive peristalsis and perianal irritation, the animal may present rectal prolapse (A) and even some intestinal torsion (B).

CAMPYLOBACTERIOSIS



4.84. Campylobacteriosis. Diarrhoea caused by *Campylobacter* sp. is dark and pasty to fluid, and it does not cause a high mortality rate (A and B). Clinical differentiation of coccidiosis is challenging, affecting lambs of the same age.



4.85. Campylobacteriosis postmortem findings. The necropsy shows a digestive tract with very enlarged lymph nodes (A) and dark intestinal content (B), especially in the final sections of the small intestine (C). In the coprological test of these animals, an absence of coccidian oocysts is observed.



TERMINAL ILEITIS

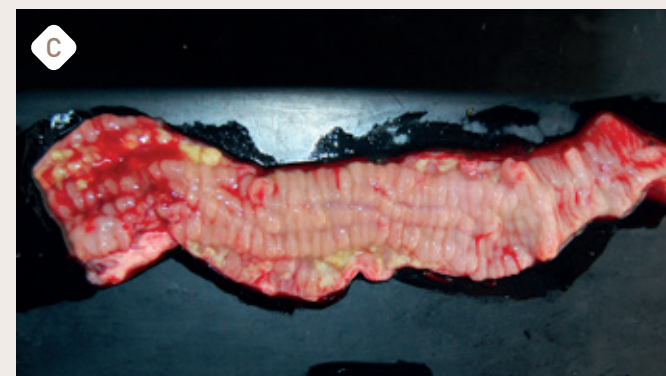
The aetiology of terminal ileitis is unknown, although it has been associated with the presence of *Campylobacter* sp. in lambs affected by the border disease virus (BDV).



4.86. Diarrhoea in terminal ileitis. In terminal ileitis, lambs usually do not show much diarrhoea (A), although if it appears, this is similar to coccidiosis (B).



4.87. Poor growth rate in terminal ileitis. The lamb's growth rate is very poor, and the difference with the other lambs in the same lot becomes more and more evident as the lambs grow. They have a poor wool appearance, bad general aspect, and abdominal distension when they get older. Terminal ileitis affected lamb is compared to a healthy lamb of the same age (A). When they reach puberty, their appearance and size indicate a lack of normal development (B).



4.88. Postmortem findings. Thickening of the intestinal wall in the terminal portion of the ileum with an intense enlargement of the mesenteric lymph nodes are the key findings of this disorder (A and B). In addition, thickening and folding of the mucosa and patchy diphtheria-like enteritis are commonly found (C-E).





OTHER DIGESTIVE CLOSTRIDIOSIS

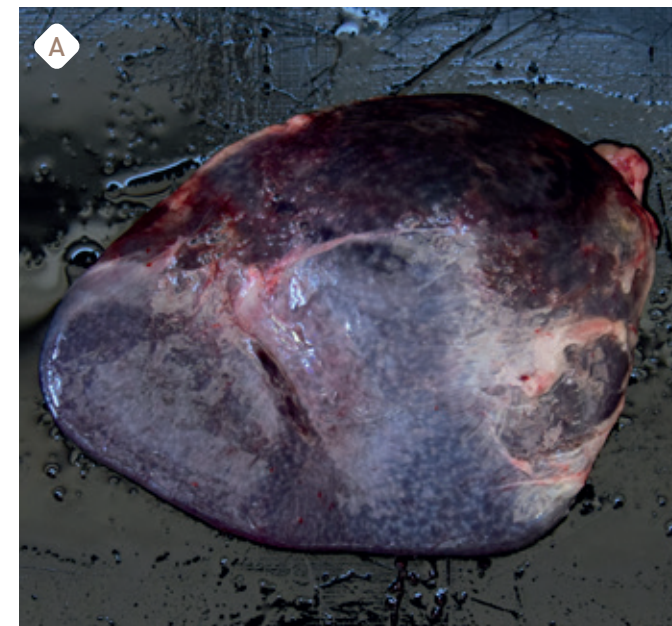
Other species of clostridia can affect the digestive system of lambs, causing severe lesions.



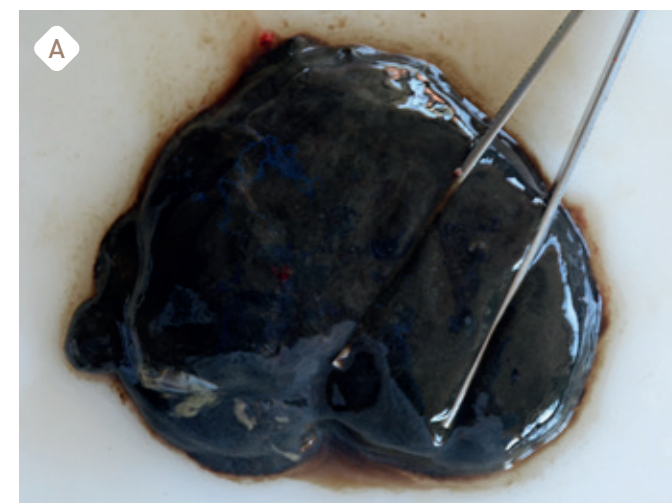
4.89. Enterotoxaemia caused by *Clostridium perfringens* type A. It is also called icteric enterotoxaemia or yellow lamb disease. It receives this name because it causes an acute disease with sudden death, rarely subacute (12-24 h) and generally asymptomatic, with the appearance of yellow carcasses (A) or cadavers (B), at sacrifice or death of the lamb, increasing the yellow colour during the airing of the carcasses.



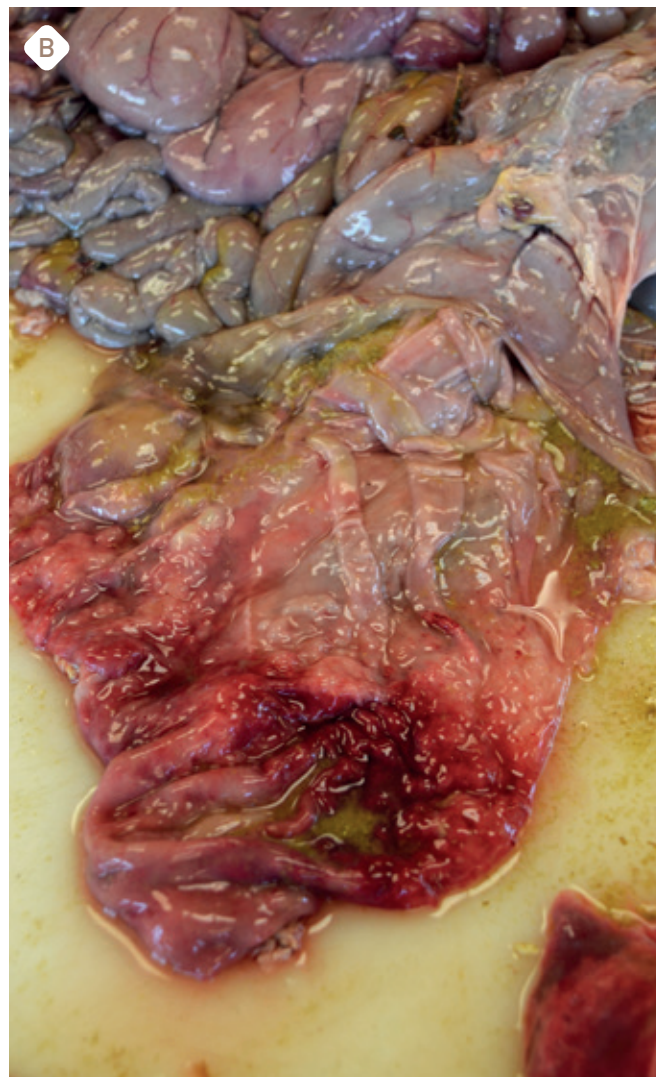
4.90. Yellow lamb disease. The necropsy or the viscera in the slaughterhouse shows a yellowish colouration (A and B), and some intestinal loops present catarrhal enteritis with fluid content (C).



4.91. Yellow lamb disease. Splenomegaly is frequently found (A), as well as ascites with amber fluid (B) and enlarged, friable and yellowish liver (C).



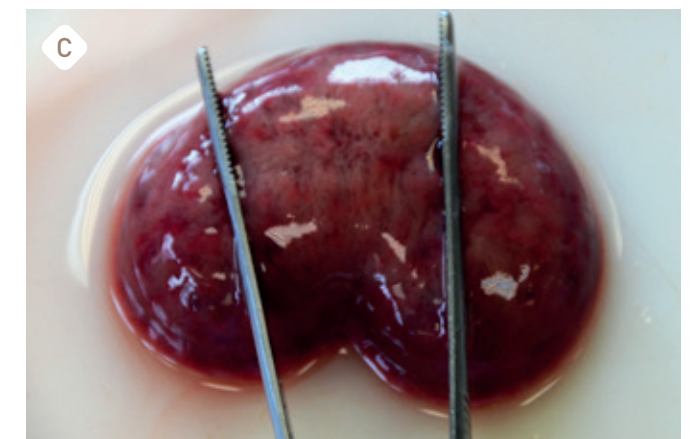
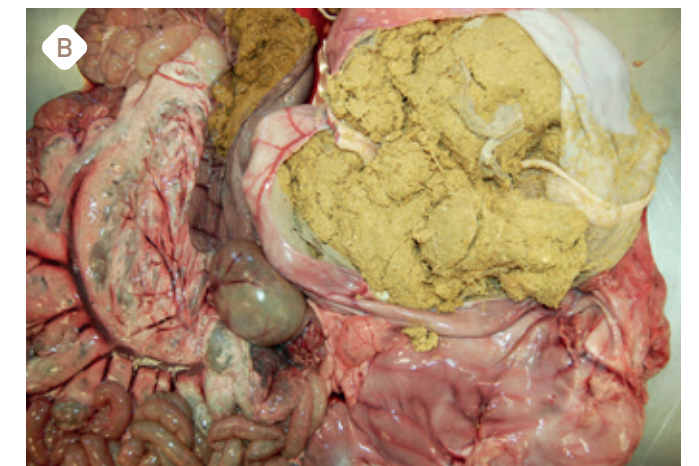
4.92. Yellow lamb disease. The kidneys are soft, enlarged and blackish due to the haemoglobinuric nephrosis they suffer (A), and the bladder is usually distended and contains dark red urine (B).



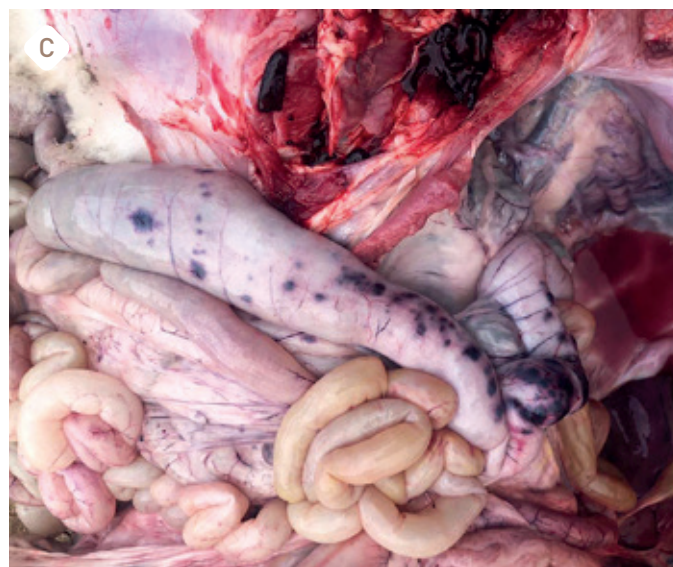
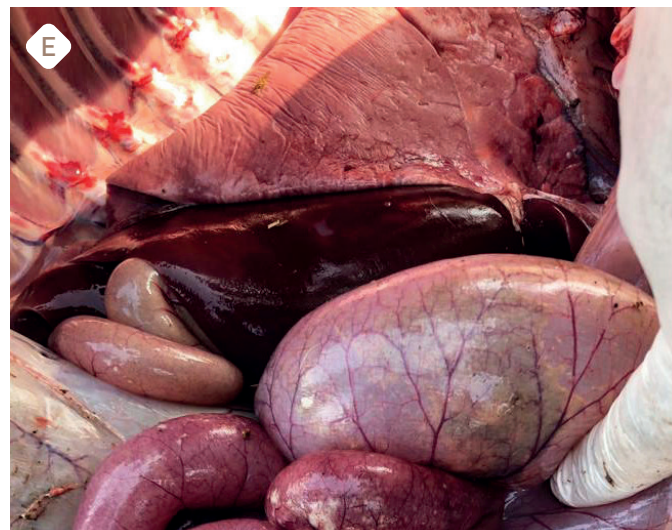
4.93. Clostridiosis caused by *Clostridium sordellii*. *Clostridium sordellii* produces abomasitis with the presence of gas (A and B).



4.94. Clostridiosis caused by *Clostridium chauvoei*. In this case, haemorrhagic disorders are predominant, especially abomasitis with different degrees of severity of bleeding (A-E).



4.95. Classic enterotoxaemia or pulpy kidney disease. It is caused by epsilon toxin produced by *Clostridium perfringens* type D and although it is more commonly found in adults, pulpy kidney disease can also affect older than three weeks lambs, especially during intensive fattening periods. Animals suddenly die or show nervous symptoms (pedalling) (A). At necropsy, paralytic ileum and stomachs full of dry content can be observed (B). Softening and accelerating autolysis of the kidneys (pulpy kidney) (C) is also a common finding.

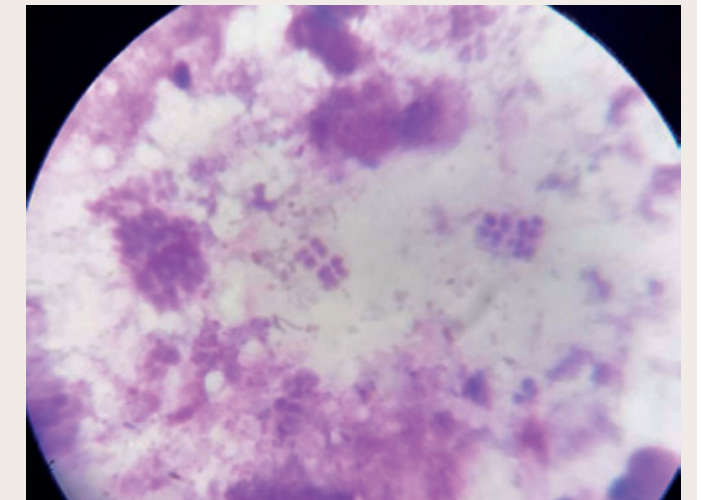


4.96. Necrotic enteritis. Necrotic enteritis associated with *Clostridium perfringens* type D (A-E). This process will be seen more extensively in the adult digestive chapter.

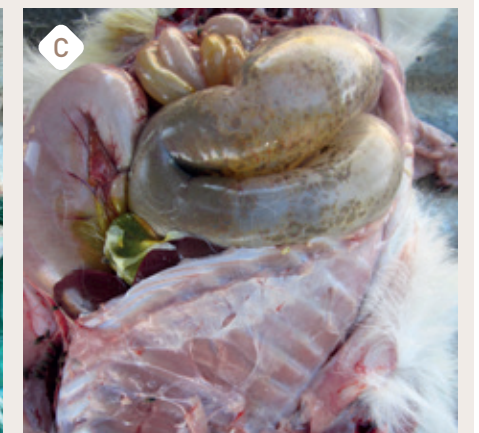
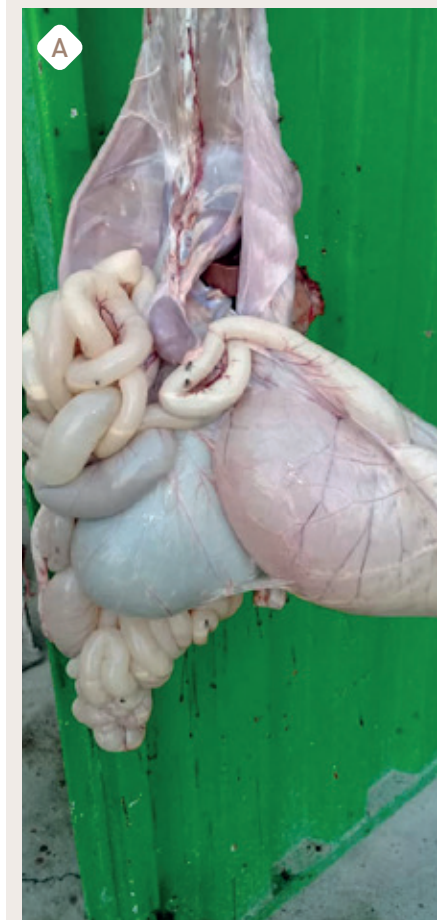
ABOMASITIS



4.97. Acute deaths with accumulation of gas. There are other digestive disorders that cause acute death with accumulation of gas in the digestive system.



4.99. Sarcina. In some of these cases with gas accumulation and of unknown aetiology, *Sarcina* sp. is detected in a direct stain forming tetrads. Picture courtesy of Dr. Mario Belaro.

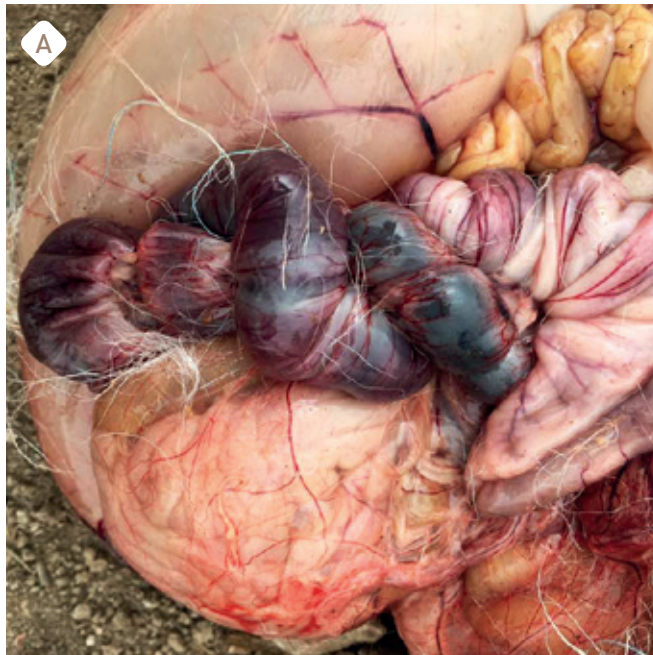


4.98. Acute deaths with accumulation of gas. The gas can be found free inside the viscera (A-C) or inside the abomasum walls forming emphysematous abomasitis (D). Picture D courtesy of Dr. Mario Belaro.



INTESTINAL STRANGULATION, VOLVULUS AND INVAGINATION

Strangulation often occurs in kids when they are fed powdered milk, especially if the temperature and volume of the milk are not well adjusted.



4.100. Intestinal volvulus or torsion. Intestinal torsion or “red gut” is a rapidly fatal condition of twisting of the intestinal mass around the mesenteric blood supply in the long axis of the mesentery and subsequent occlusion of the cranial mesenteric artery and vein. The area of the intestine above the obstruction continues to function and fills with food, fluids and gas (A and B).



4.101. Consequences. The volvulus results in intestinal distension, vascular compromise, intestinal necrosis, and eventually death unless surgically corrected.



4.102. Consequences. There is hyperemia and congestion in some intestinal areas.



4.103. Invagination. Excessive peristalsis can cause a segment of the intestine to fold and penetrate the lumen of the adjacent anterior segment. Consequences are similar to those of the intestinal volvulus.

RUMINAL DISORDERS

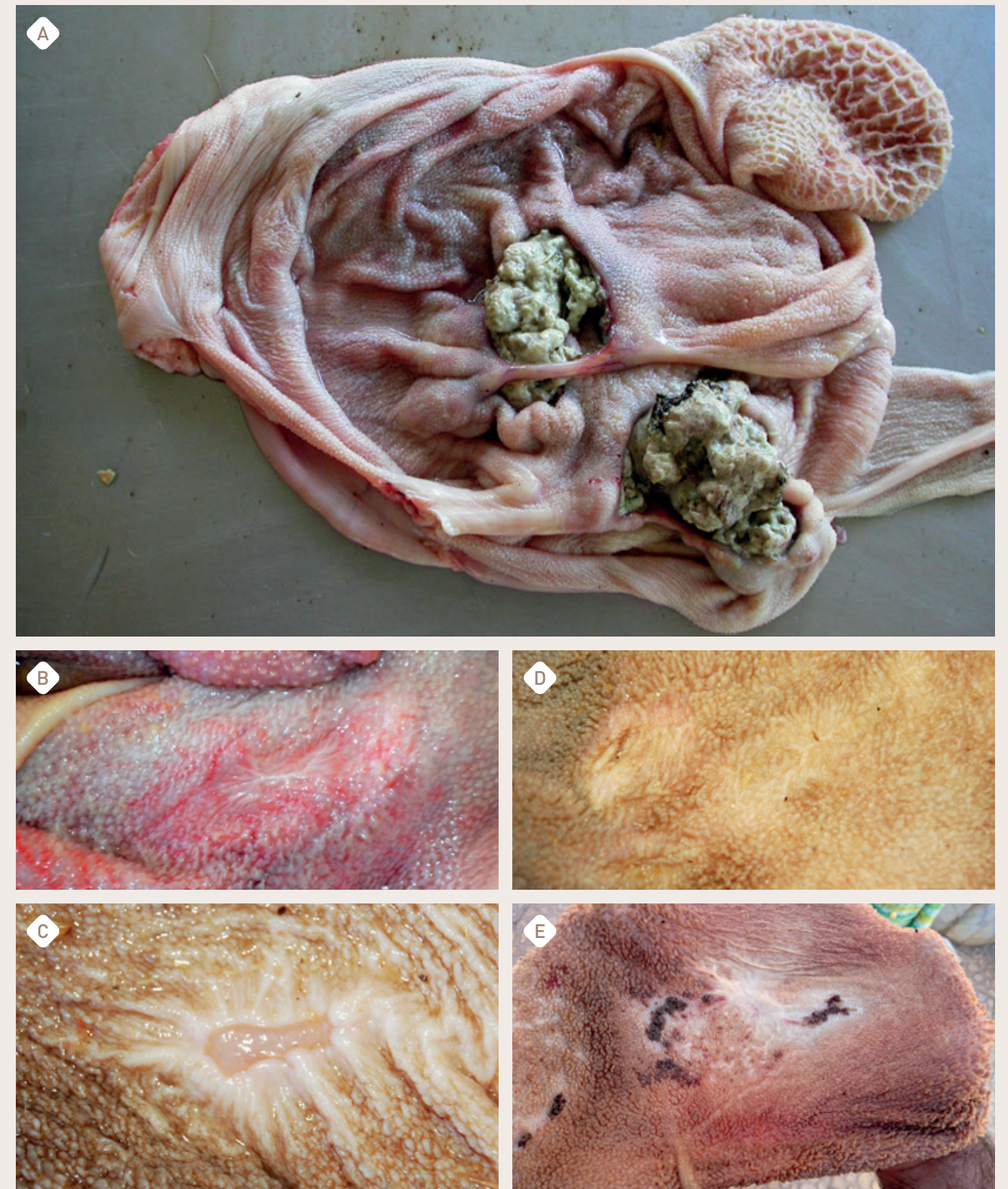
The consumption of excessively energetic, poorly balanced or low-fibre feed in the ration during the fattening period usually leads to the development of acidosis. This can be presented as subacute or chronic disorders and, many times, no clinical signs are observed.



4.104. Acidosis. Sometimes excess carbohydrate intake can trigger diarrhoea (A). The pH of the rumen is usually shown as equal to or less than 5 (B).



4.105. **Acute acidosis.** After a massive and prolonged intake of carbohydrates (A and B), various transformations can be observed in the mucosa of the pre-stomachs derived from a process of acidosis. The images above show ruminitis (C-F), loss of villi (G) and areas of hyper and parakeratosis (H).



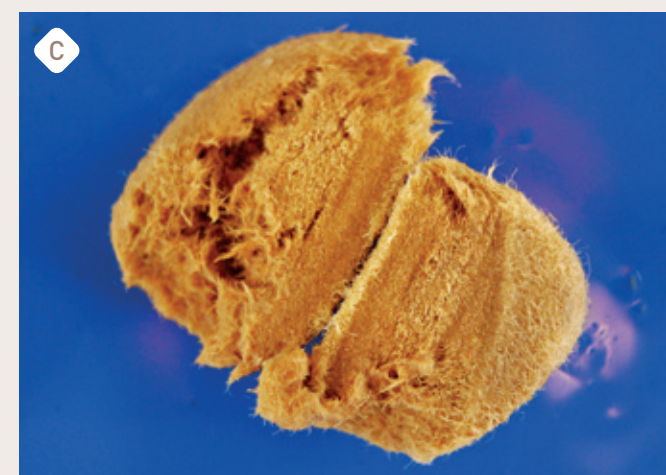
4.106. **Acute scarred ruminal ulcers.** Many times, acidosis induces ulcers that can be seen in the ruminal wall in different degrees of affection (A-E).



4.107. Acidosis and abomasitis. Abomasitis (A) and various abomasal ulcers (B) can derive from a process of ruminal acidosis.



4.108. Latent hydrochloric acidosis. Abomasal ulcers close to the pyloric area (A) may develop a posterior vagal syndrome with food retention in the abomasum and a rumen with low content (B). In this situation, a latent hydrochloric hyperacidity occurs due to the reflux of hydrochloric acid towards the rumen, causing varying degrees of damage to the ruminal mucosa (C).



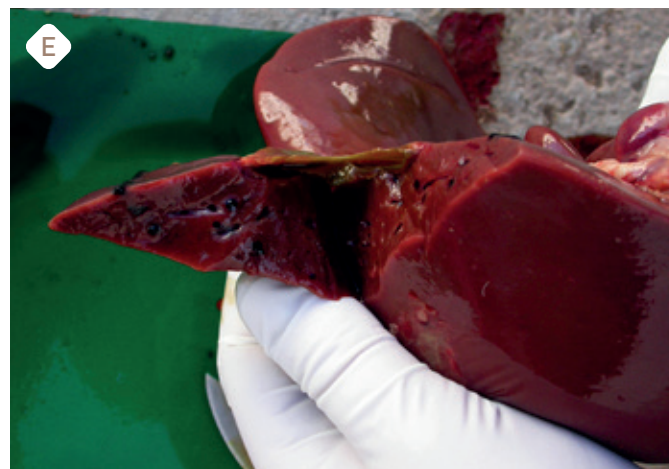
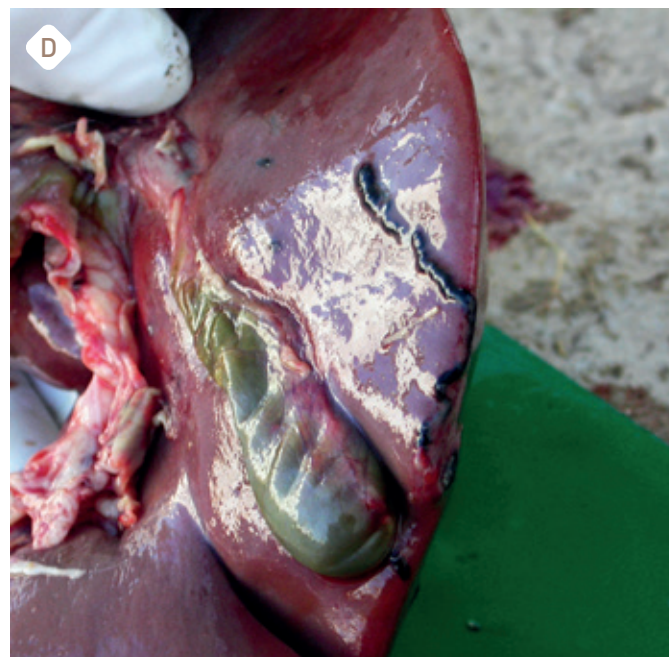
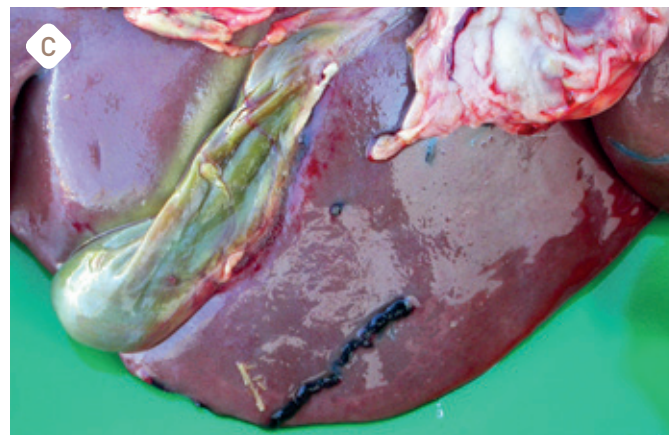
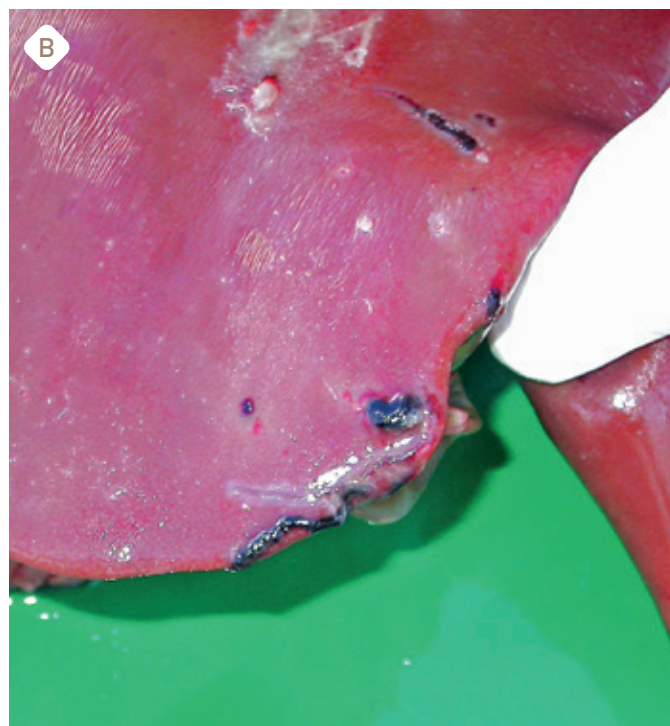
4.109. Bezoars. High-energy, low-fibre diets induce animals to look elsewhere for fibre. One of the most frequent is the licking and pulling of hair or wool from their mothers or other lambs. Consumption of hair or wool generates consistent trichobezoars (A-C). The consumption of poorly digestible vegetable fibers forms phytobezoars (D and E).



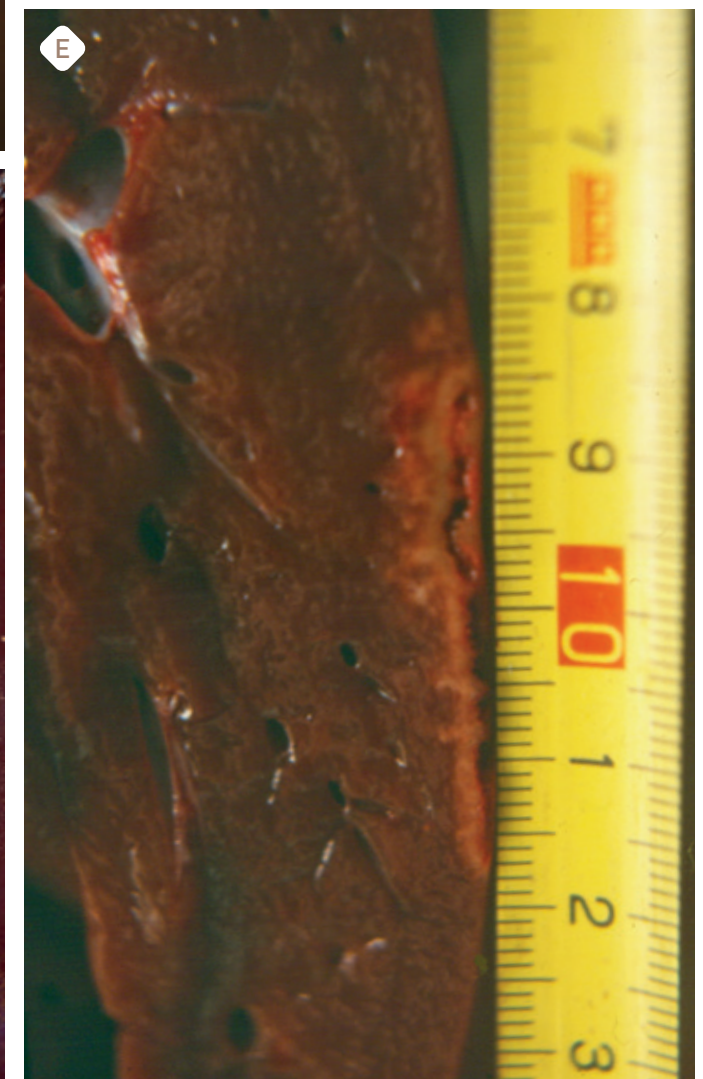
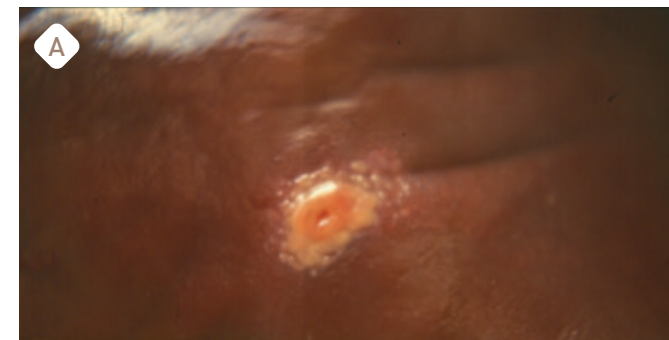
VISCERAL CYSTICERCOSIS

Ovine visceral cysticercosis is caused by the larval stages of *Taenia hydatigena*.

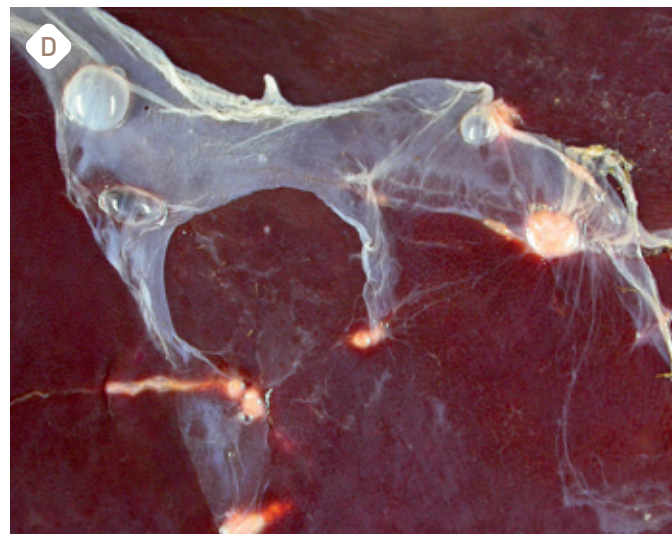
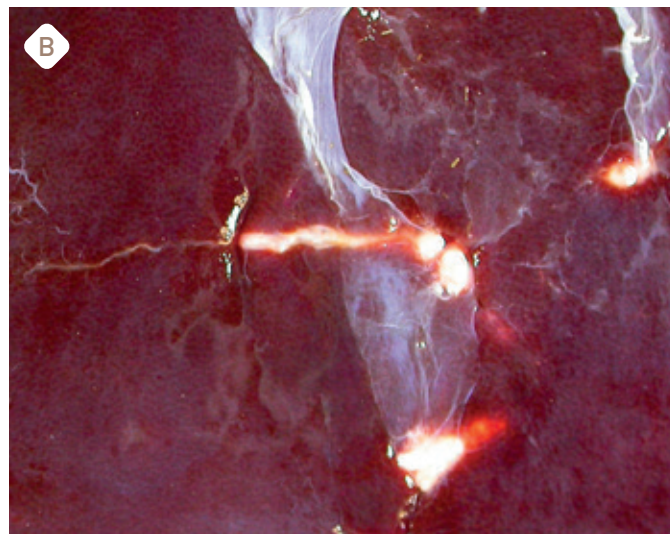
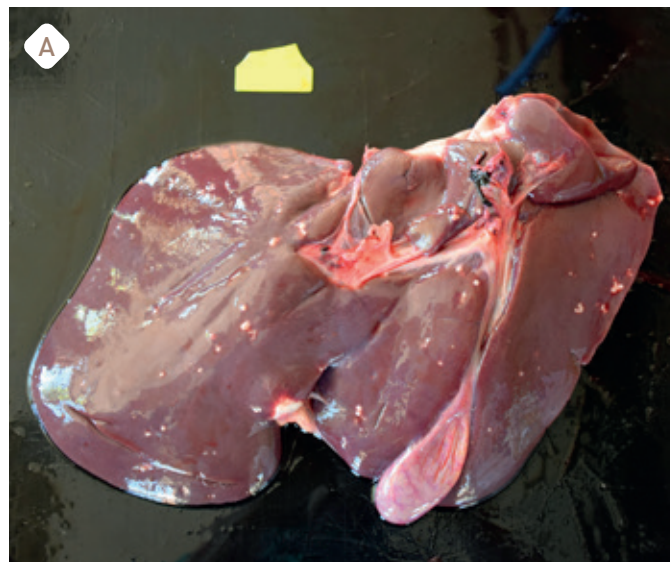
Two different clinical presentations can be found in lambs: the paths caused by the migration of *Taenia hydatigena* oncospheres through the liver parenchyma and the implantation of the vesicles in the omentum and the peritoneal surfaces of different viscera.



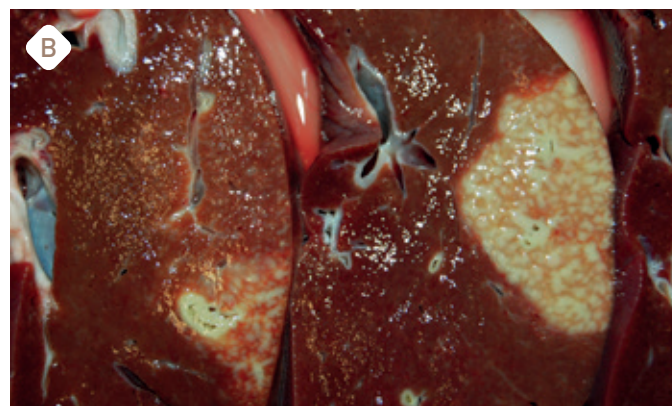
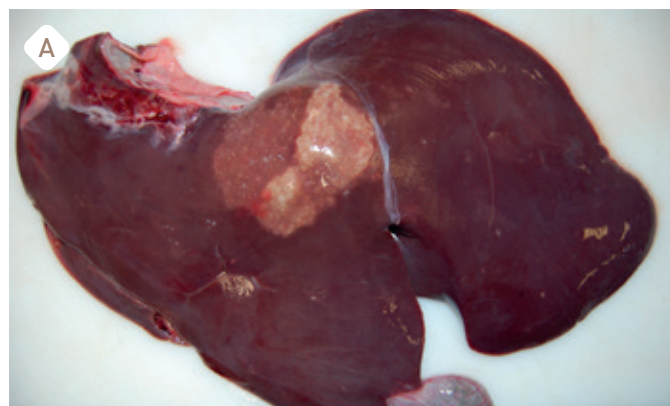
4.110. Visceral cysticercosis. Haemorrhagic tracts in the liver. *Taenia hydatigena* oncospheres cross the liver parenchyma, perforating the capsule and emerging at one point to the surface. Sometimes they make small subcapsular tracts, leaving a point or small haemorrhagic tracts (A and B). Occasionally, these haemorrhagic subcapsular trajectories are long and sinuous (C and D). The section of the liver shows the haemorrhagic paths in its parenchyma (E).



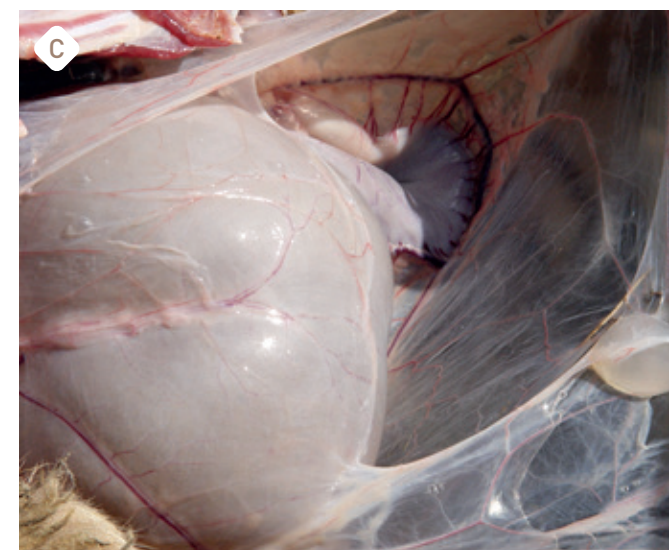
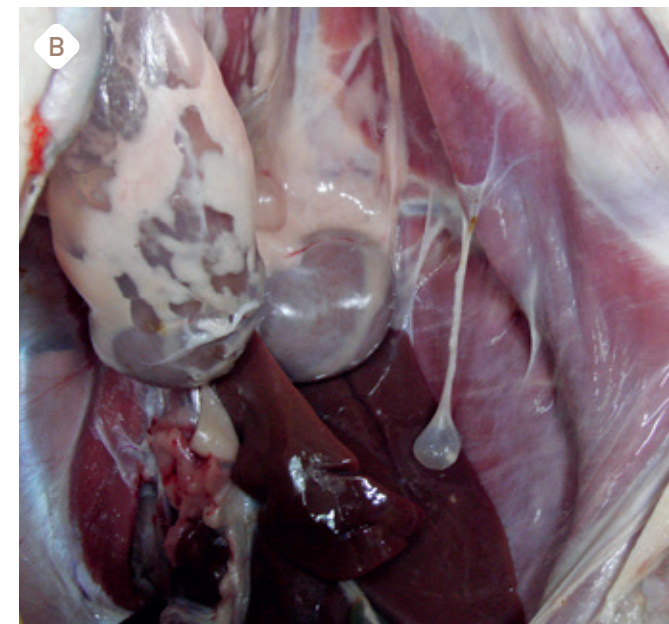
4.111. Progression of the visceral cysticercosis. As the pathways progress, the liver attempts to repair them by accumulating inflammatory cells that give the repaired trajectory a whitish appearance (A-C). The section of the liver shows this repair reaction of the parenchyma (D and E).



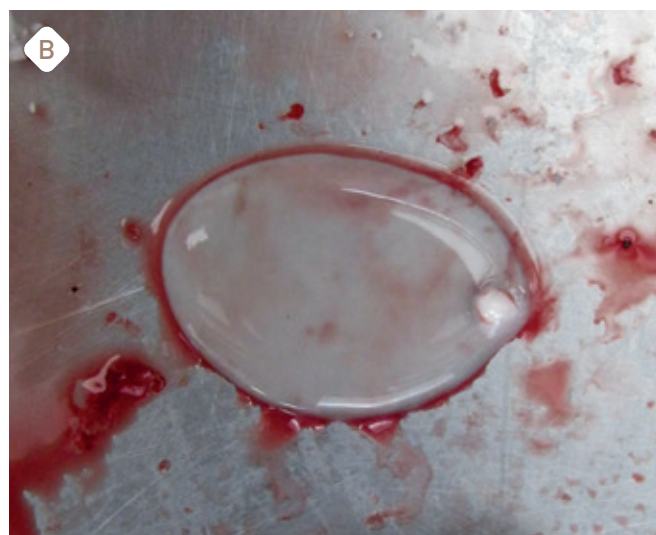
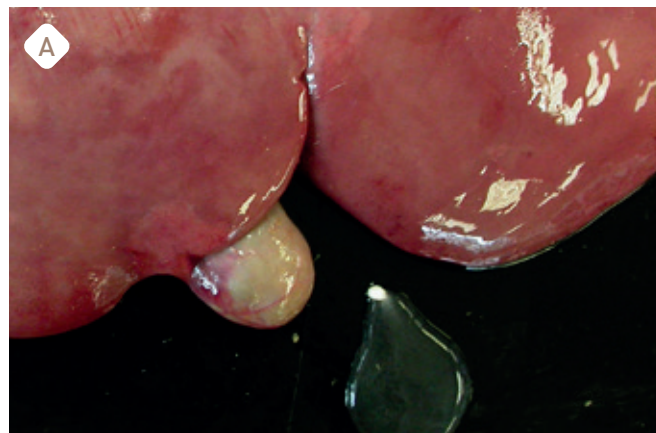
4.112. Visceral cysticercosis. Exit to the abdominal cavity. Once the oncospheres reach the liver capsule, they rise to the surface and form the parasitic vesicle. Not all of them succeed, with some remaining on the liver surface as whitish scars (A and B). However, others succeed, and they can be seen on the liver surface (C and D).



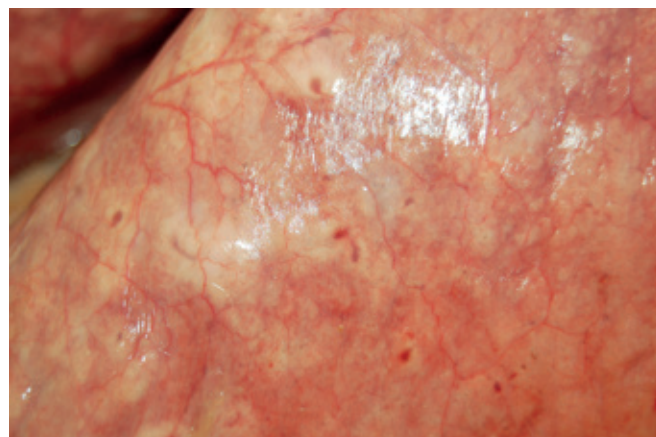
4.113. Visceral cysticercosis. Focal perihepatitis. During the migration of this oncosphere or reaching the surface to transform into a vesicle, it causes a more intense reaction, developing focal perihepatitis (A and B).



4.114. Cysticercosis in the omentum. Some oncospheres leave the liver surface and settle in the omentum, in more or less remote areas from the liver surface (A-E).

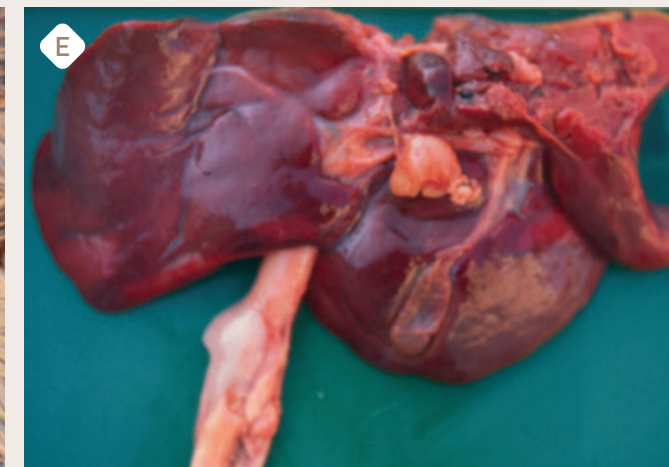


4.115. Vesicles in ovine cysticercosis. The vesicles, lined with peritoneum, can be released from it manually at necropsy. The rupture of the protection and the exteriorisation of the vesicle can be done, observing the protoscolex (A and B).

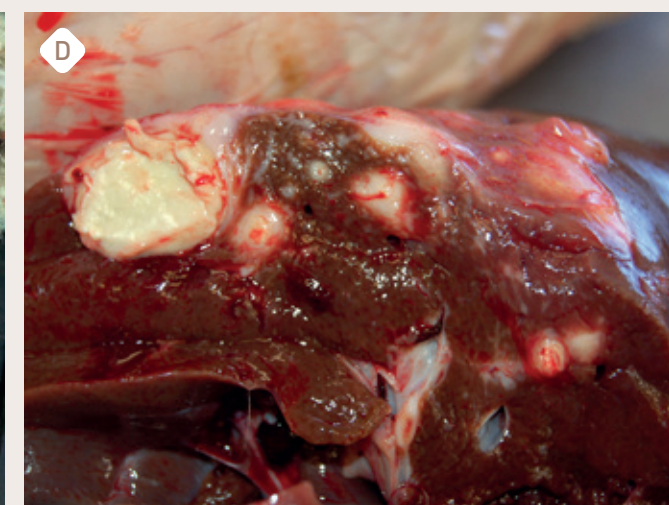
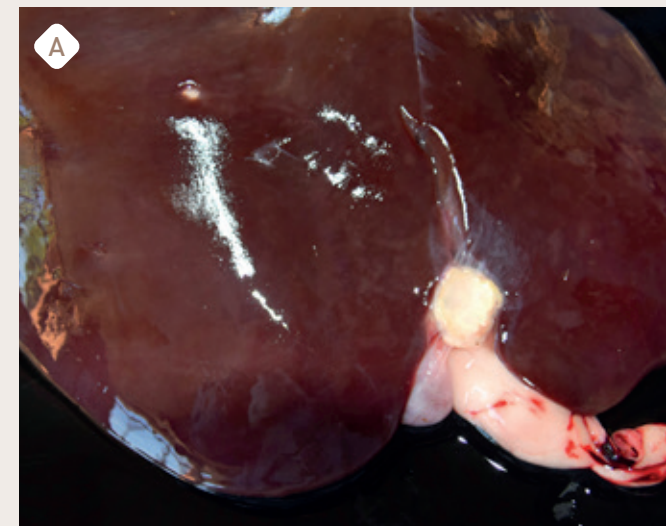


4.116. Other locations. Although the abdominal cavity is the most frequently affected, visceral cysticercosis can be located in other organs, such as the lung.

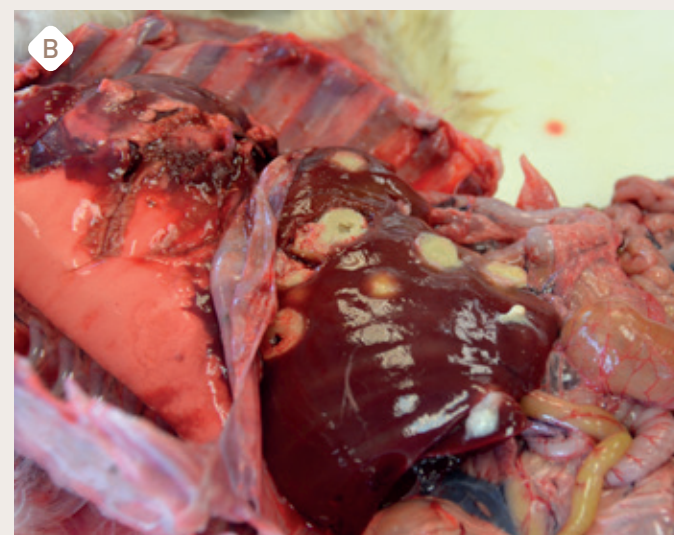
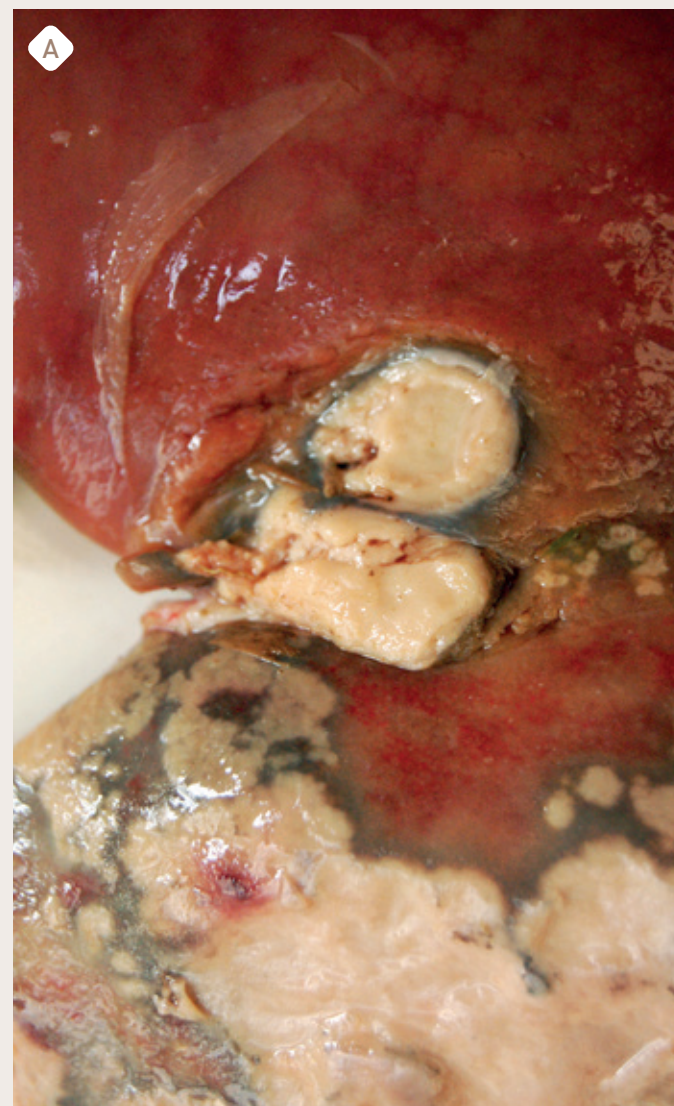
OMPHALITIS-LIVER / ABSCESS SYNDROME



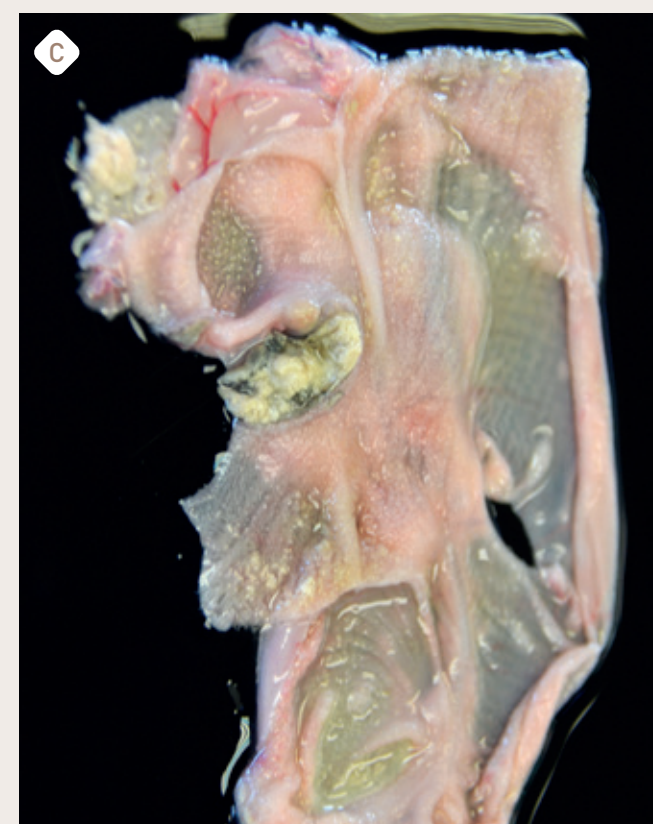
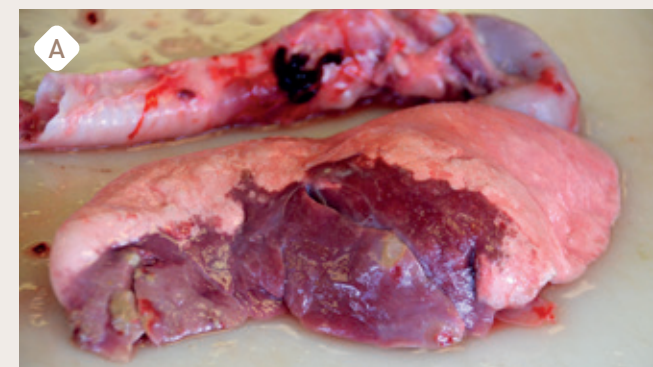
4.117. Omphalitis-liver abscess syndrome. This syndrome affects young lambs. The liver disease begins with the infection of the umbilical cord (omphalitis) (A), which is a painful process (B). The umbilical area is wet due to exudates and licks from the same animal (C and D). Infection and inflammation can progress until reaching the liver (E).



4.118. Liver abscess. One of the consequences of omphalitis is the presence of an abscess in the hepatic notch (A) or in other areas of the liver (B-D).



4.119. Necrobacillosis in the liver. When umbilical infection is caused by *Fusobacterium necrophorum* and it spreads through the liver parenchyma, it produces focal necrosis (necrobacillosis) [A-E].



4.120. Dissemination of necrobacillosis. *Fusobacterium necrophorum* can be spread, and foci can be also found in different organs [A-C].



4.121. Polyarthritis caused by umbilical infection. Sometimes umbilical infections can favour the progression of the microorganisms to the joints, causing polyarthritis [see chapter 8, page 417].