ADULT ANIMALS

ORAL CAVITY: SOFT TISSUES

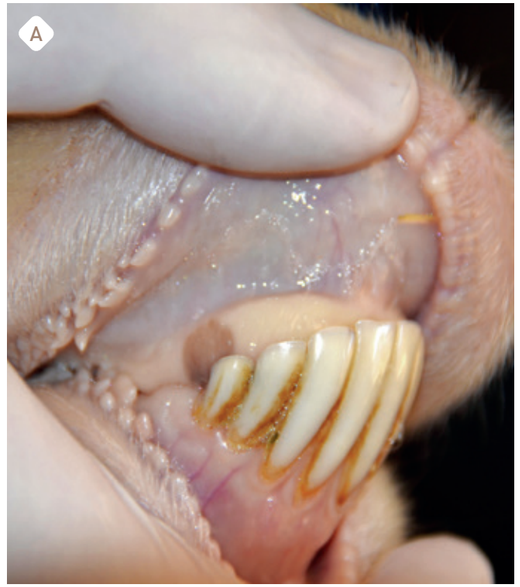
MUCOSAL COLOUR CHANGES

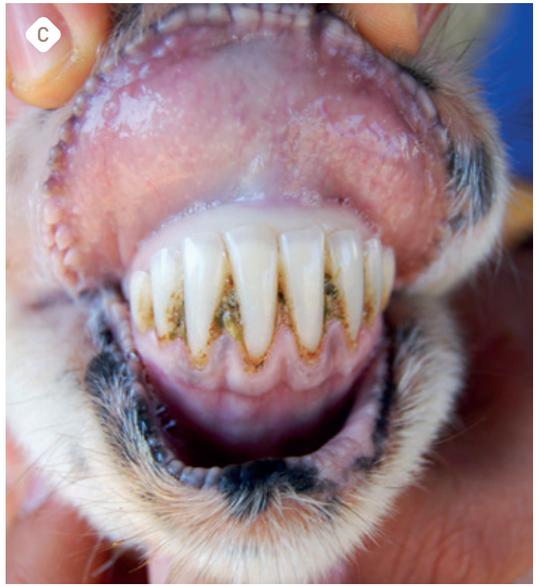


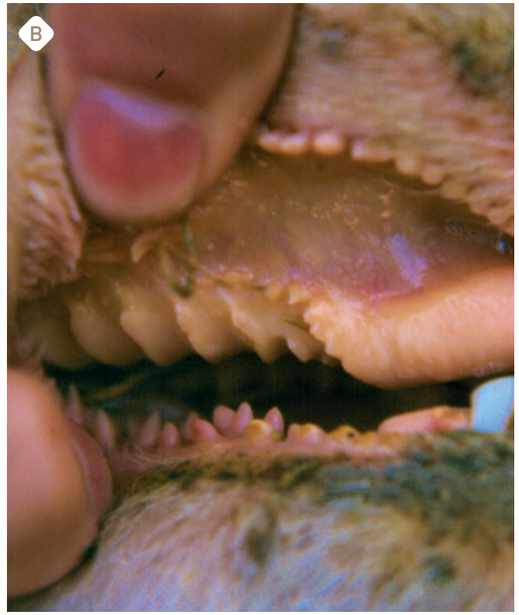




4.122. **Exogenous pigments.** The consumption of poppies in large quantities and for a long time leads to the accumulation of dark red, almost black, pigment on the skin of the snout and in the tactile hairs (A). We can observe similar pigmentations with other foods, such as red beet (B) or orange (C). The colour of the nostrils can be an indicator of abnormal pigmentation in the faeces.





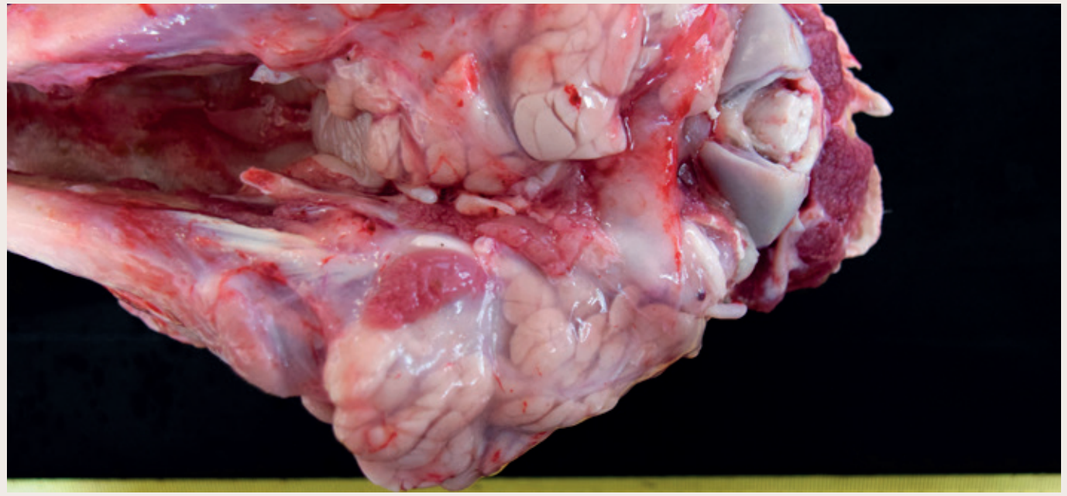


4.123. **Pigmentation of the oral mucosa.** Various disorders can be reflected in the colour of the oral mucosa. Diseases that cause anaemia make the oral mucosa pale (A), jaundice colour the mucosa in yellow (B), and cyanosis processes darken it or turn it bluish (C).

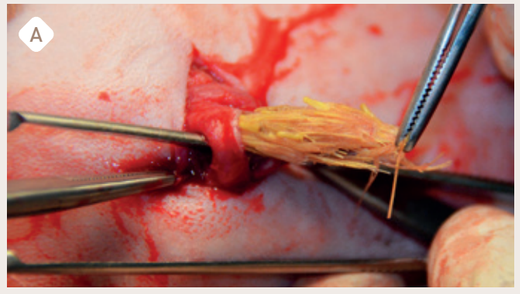


4.124. **Pigmentation of the oral mucosa: haemorrhages.** Some previous haemorrhages can be detected in the oral mucosa as blue spots.

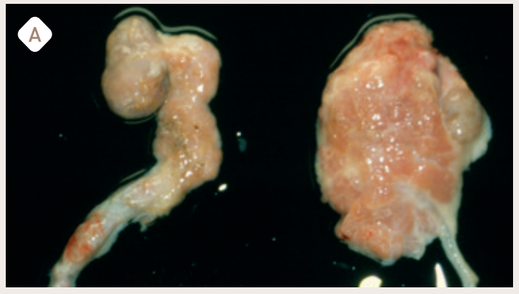
SALIVARY GLANDS



4.125. **Hypertrophy of salivary glands.** Hypertrophy of the major salivary glands of undetermined aetiology, found in a tumoral process in the nasal cavity.



4.126. **Spikelets in ductus parotideus.** Various foreign bodies can enter and progress through Stenon's duct. It is possible to remove the material. The pictures above show the surgery (A) and the material obtained (B).



4.127. **Parotid atrophy.** Parotid atrophy associated with a Horner syndrome and caused by the progression of spikelets through the parotid duct to the salivary gland. This led to its atrophy, affecting the cervical sympathetic ganglion and developing Horner's syndrome (A and B).

SOFT TISSUE TUMOURS



4.128. **Carcinoma on the lower lip.** Although not very frequently, oral tumours can be diagnosed sporadically.



4.129. **Tumours.** Fibroma in the cheek.



4.130. **Tumours** that spread in the mandible, separating the two mandibular bones and invading the interior of the mouth.

PARASITES IN THE MOUTH







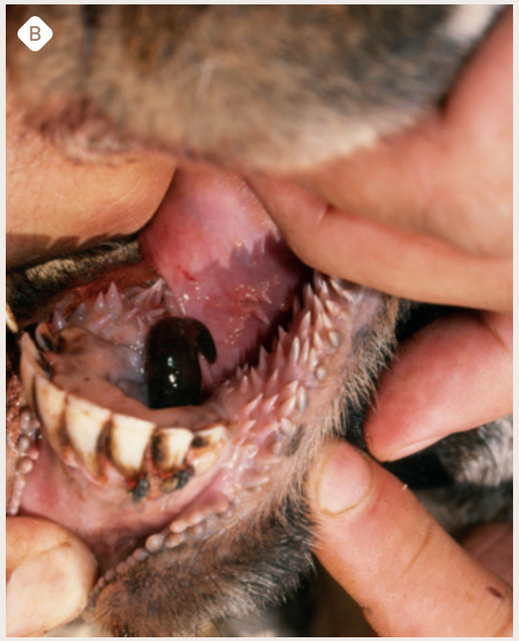


4.131. **Cutaneous myiasis.** Stomatitis (A) and wounds (B and C) in the labial commissure sometimes give rise to secondary myiasis by larvae of *Wohlfahrtia magnifica* (D).



4.132. **Cutaneous myiasis.** *Wohlfahrtia magnifica* flies.





4.133. **Parasitisation by leeches.** *Limnatis nilotica* (A) appears mainly in the mouth and nose (B).

SPIKELETS IN SOFT TISSUES

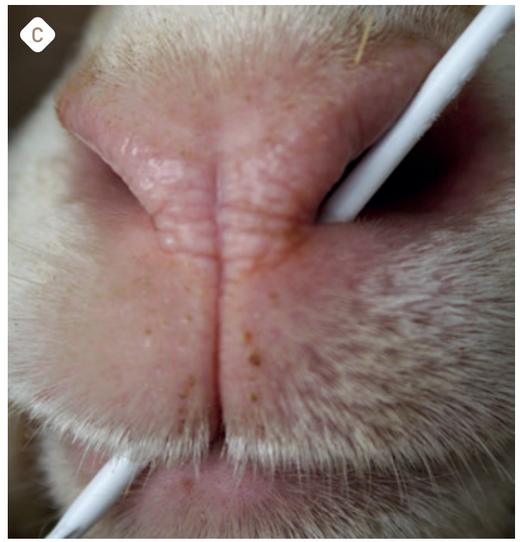




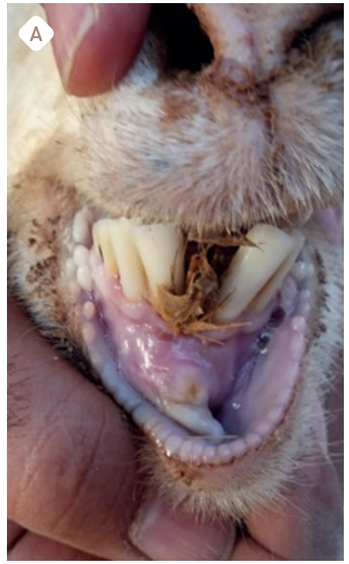
4.134. **Spikelets in soft tissues.** Spikelets and sharp straw in the lips, nostril, larynx, and gums disturb animal comfort (A and B).

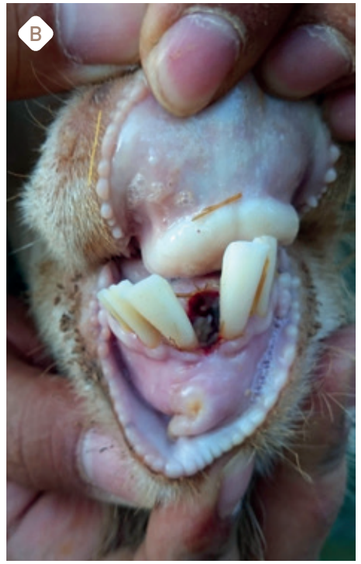


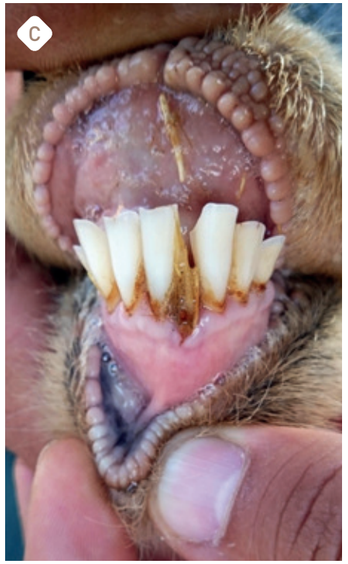




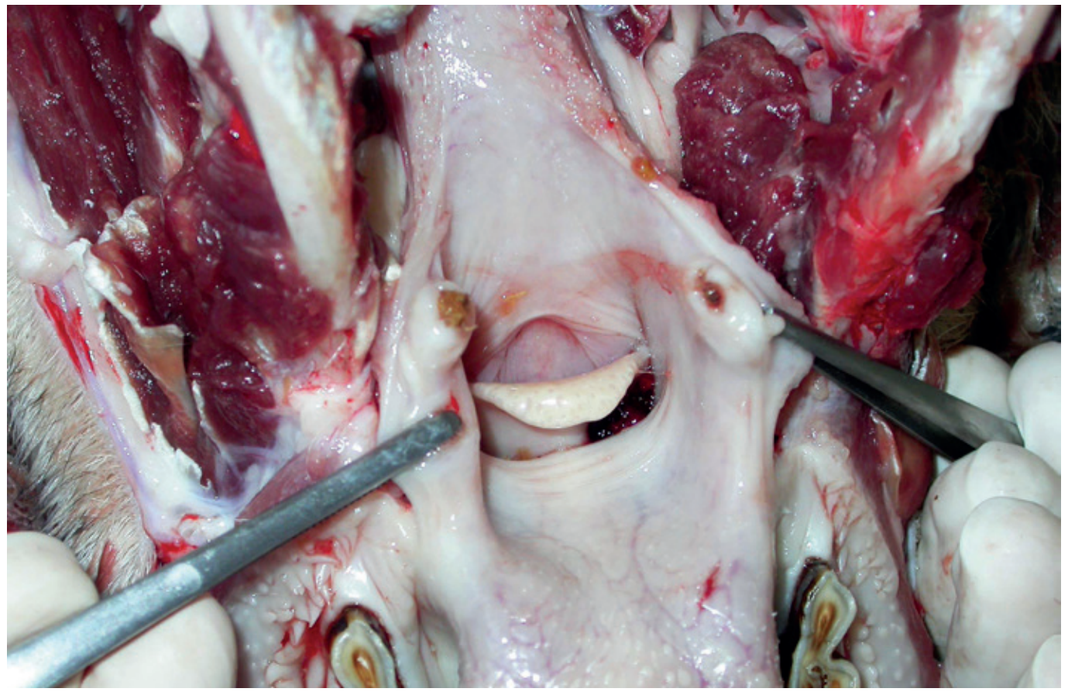
4.135. **Spikelets in soft tissues.** Spikelets embedded in the oral mucosa (A) cross the lip and emerge in the vicinity of the nostrils (B). The lip can be crossed with a probe to visualise the path (C).







4.136. **Spikelets in soft tissues.** Spikelets nailed between the incisors of goats (A-C).

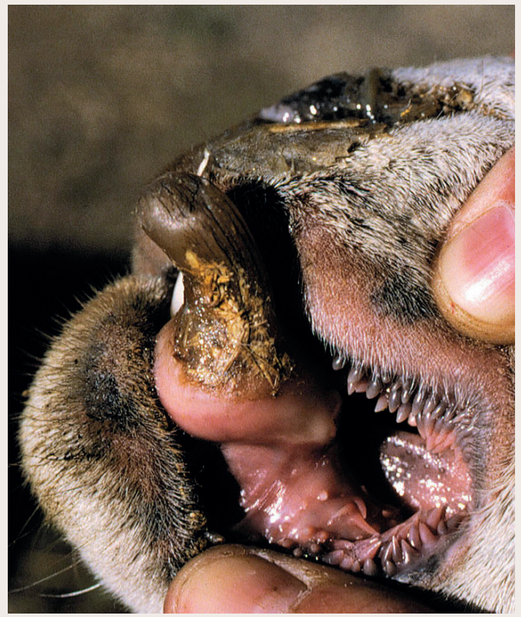


4.137. **Spikelets in soft tissues.** Spikelets stuck in the tonsils.

CUTANEOUS HORN



4.138. **Cutaneous horn.** Large cutaneous horn on the skin of the face that physically prevents normal grazing.



4.139. **Cutaneous horn.** Cutaneous horn on the gingiva, close to the incisors.





4.140. **Cutaneous horn.** Medium-sized cutaneous horn close to the mouth (A and B).

PLANTS THAT CAN CAUSE DAMAGE IN THE ORAL MUCOSA



4.141. **Plants that can cause damage in the oral mucosa.** Some plants can damage the skin of the face and the inner mucous membranes. The most important are classified as: sharp (*Xanthium spinosum*) (A and B), urticants (*Urtica* sp.) (C and D) and irritants (*Phytolaca americana*) (E and F).

CHEMICAL AGENTS



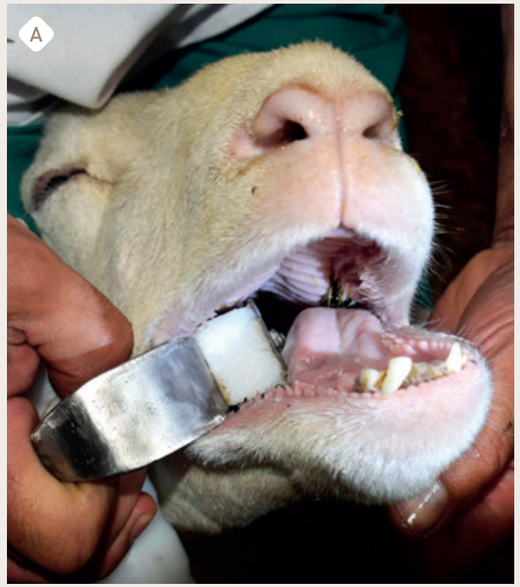
4.142. **Sialorrhea.** Some chemicals with very high or low pH and some plants (see the previous point) can cause irritation and the consequent increase in the production of saliva and hypersalivation. The same clinical signs are observed in the consumption of bitter-tasting diets that stimulate the production of saliva.

PHOTOSENSITISATION (*see chapter 11, page 578*)



4.143. **Photosensitisation.** Different physical, chemical and biological agents can cause inflammation and oedema in the face and lips. Different degrees of facial swelling and oedema caused by photosensitisation can be seen in the images (A and B).

TRAUMATIC ORAL DISORDERS



4.144. **Trauma.** The mouth can suffer trauma during oral examination (A and B) and the application of oral substances with dosing guns. Likewise, it can be injured with thorny plants (see previous page) and other pointed or sharp objects.



4.145. **Trauma.** Teeth fractures, gums injury and swelling of the face (A and B).



4.146. **Trauma.** Lack of tissue on the upper lip caused by a dog bite.

INFLAMMATIONS ASSOCIATED WITH LOCAL OR MULTISYSTEMIC INFECTIOUS DISORDERS

Stomatitis is the inflammation of the soft tissues of the mouth. This can suffer microbiological aggressions, either from infections that only affect the mouth or from systemic diseases with a critical oral component. These disorders can have different degree of affection, giving oedema, inflammation, ulcers or necrosis in part or all of the oral cavity tissues. These disorders will be treated as individualised multisystemic diseases at the end of this chapter.



4.147. **Contagious ecthyma (orf).** Ecthyma can cause different degrees of oral conditions in adults. Cheilitis (A), gingivitis with bacterial contamination (B).



4.148. ***Staphylococcus aureus* abscess.** Different microorganisms can be involved in disorders that affect the oral cavity, like this lamb with an abscess on the upper lip that makes lactation difficult.



4.149. **Glossitis.** Inflammation of the tongue or glossitis can be associated with different infectious diseases (ecthyma, bluetongue, PPR, etc.), as well as allergic, traumatic or chemical causes (A and B).




4.150. **Bluetongue.** Cheilitis and blood stained nasal discharge (A) and cyanotic tongue with oedema and mouth bleed (B).




4.151. **Peste des petits ruminants.** Necrotic lesions appear in the oral cavity and expand over the entire oral mucosa, forming diphtheritic plaques (A and B).




4.152. **Foot and mouth disease.** Since superficial epithelium in sheep and goat is very thin and easily ruptured, it is unusual to see fluid-filled vesicles in the mouths of these species. Erosions may be seen on the gums, hard and soft palate, lips, dental pad and the tongue. Occasionally, they may be found in the nostrils (A and B).

ACTINOBACILLOSIS

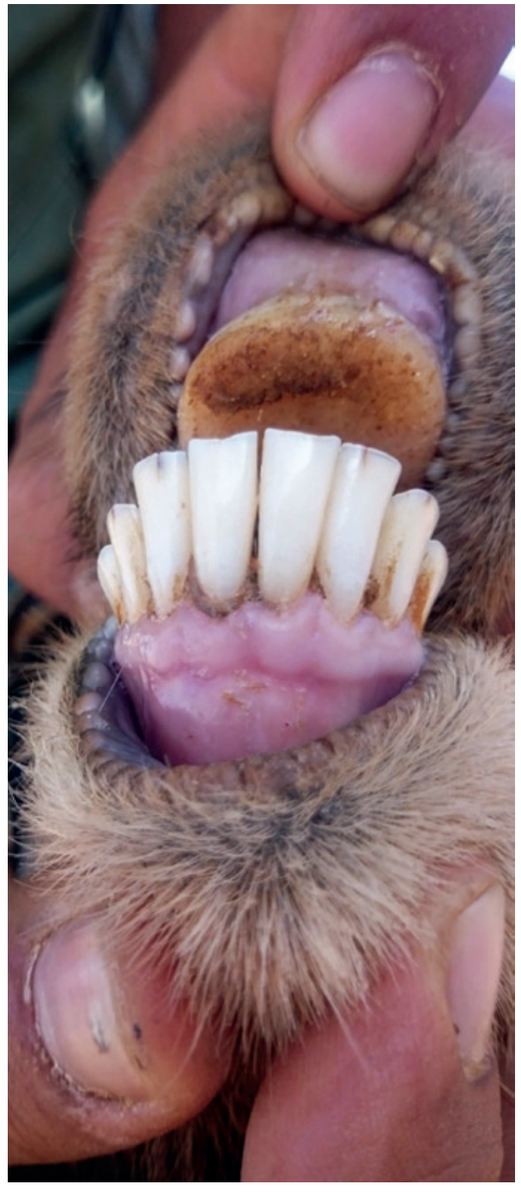




4.153. **Actinobacillosis.** *Actinobacillus lignieresii* is the microorganism that causes this chronic granulomatous infection of the skin and subcutaneous tissues of the head. It is more common in adult sheep and may be associated with wounds, skin abrasions or grass-seed penetration of the skin and oral mucosa. The lesion has thick greenish-yellow pus (A-C).

OTHERS

Sometimes we can find injuries that are difficult to define without a good medical history.



4.154. Dental pad injury due to pasture wood forage.

ORAL CAVITY: HARD TISSUES

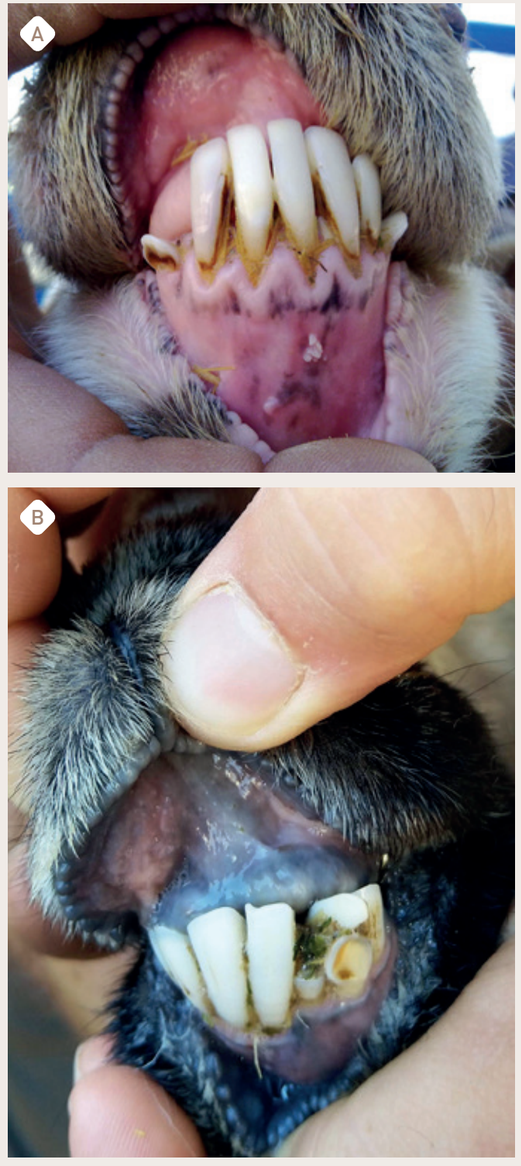
PROGNATHISM AND BRACHYGNATHISM

Although congenital disorders that affect the mouth (*see chapter 1, page 7*) often lead to the elimination of animals as replacement, on occasion, some of them go unnoticed and the animals can reach adulthood. Brachygnathism and prognathism both occur in goats and sheep and adversely affect feeding behaviour under grazing conditions.



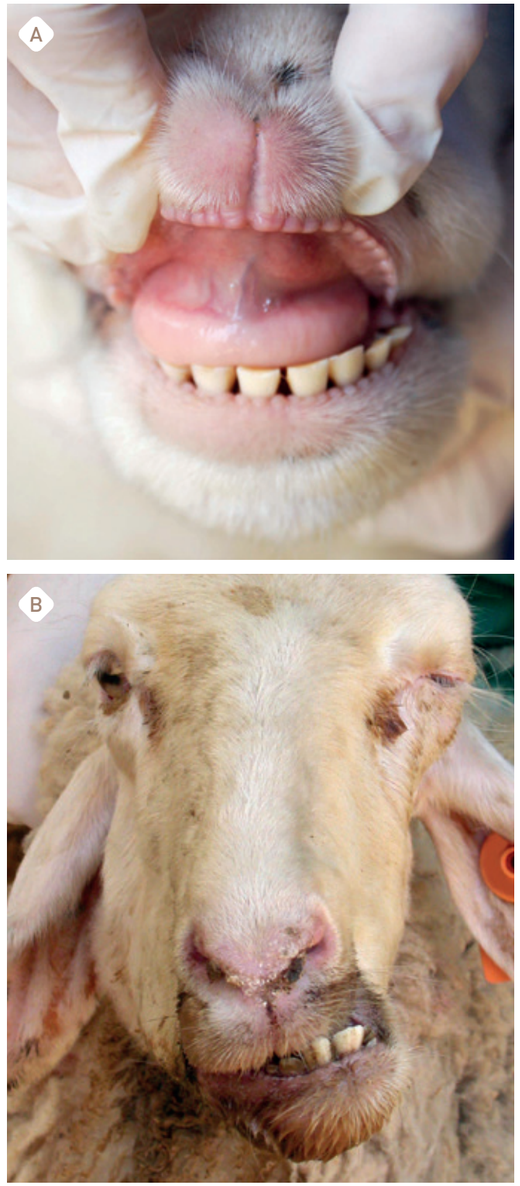
4.155. **Prognathism and brachygnathism.** Prognathism is an extension or protrusion of the mandible or the maxilla. On the contrary, brachygnathism occurs when the mandible or the maxilla are shorter than normal (A-H).

INVERTED AND ECTOPIC TEETH



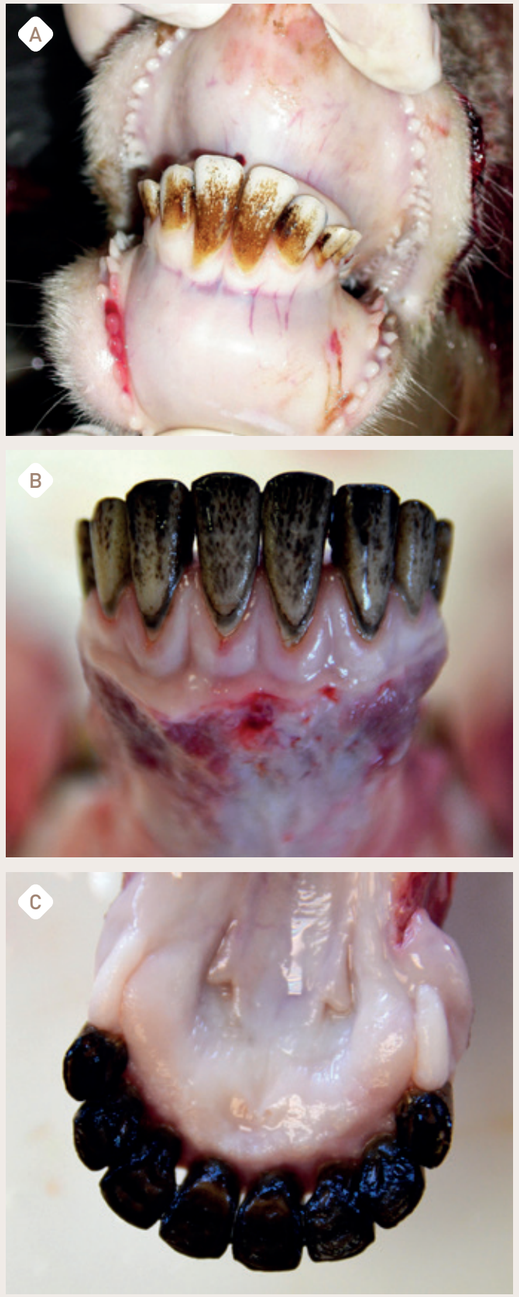
4.156. **Inverted and ectopic teeth.** The presence of ectopic teeth and the inverse growth of a pair of teeth in sheep (A and B).

CROSSBITE



4.157. **Crossbite.** It is a defect in the closure of the dental arches, being crossed in different degrees (A and B). It is associated with poor dental occlusion or disorders affecting the temporomandibular joint.

DENTAL PIGMENTATION





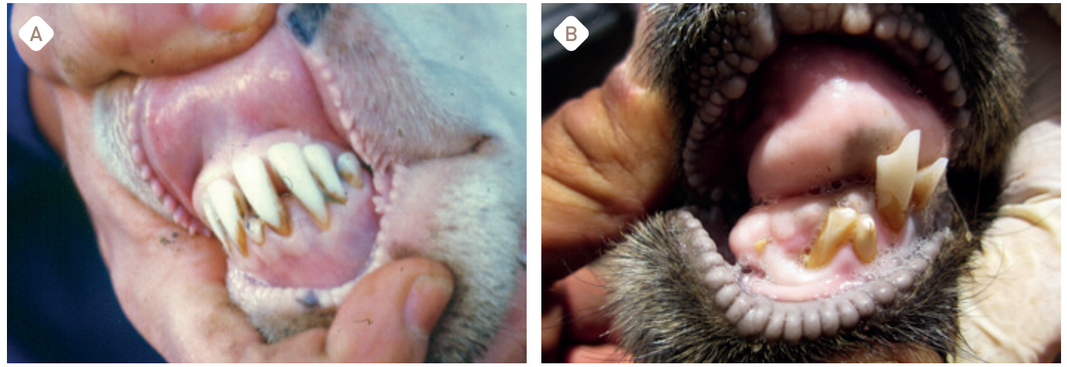
4.158. **Dental pigmentation.** The consumption of some substances such as tetracyclines during tooth formation can cause new teeth with permanent pigmentation. Sometimes the pigments are deposited on the teeth or on the layer of tartar that forms on them. These pigmentations can affect the incisors (A-D) and the molars (E and F).

INCISORS SEPARATION AND LOSS (BROKEN MOUTH)

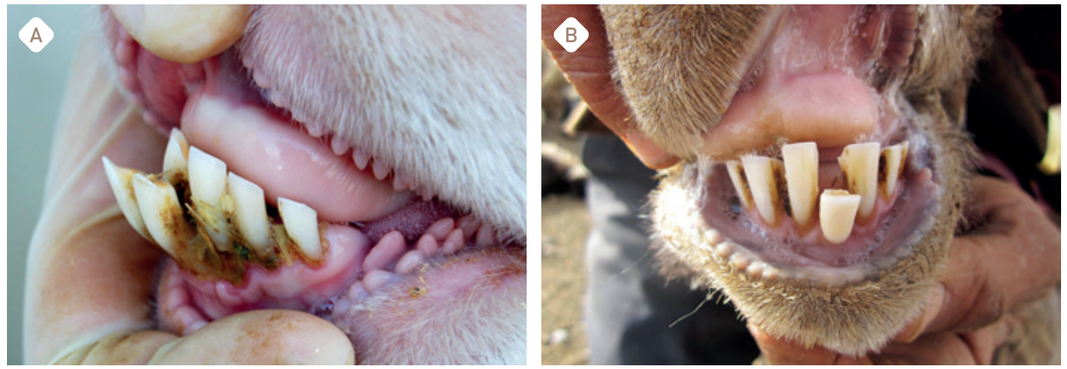
Broken mouth is a colloquial term used widely throughout sheep industries to describe the widespread oral lesions occurring from the excessive wear and loss of incisor teeth in ageing sheep. It is associated with trauma from prehension of harsh feed and thistles, plus the grinding of dirt when sheep need to dig for plant roots, especially in droughts. Periodontal infections also promote tooth loss. Premature loss of incisor teeth or "broken mouth" causes concern because sheep and goats are culled before the end of their economic life. In some cases, some of the teeth have been lost, and in others, all the incisor teeth have fallen out.



4.159. **Incisors separation.** With increasing age, the incisors progressively separate, exposing part of the root (A and B).



4.160. **Incisors fracture.** The fracture of the incisors or part of them of traumatic or deficiency origin can affect food intake (A and B).



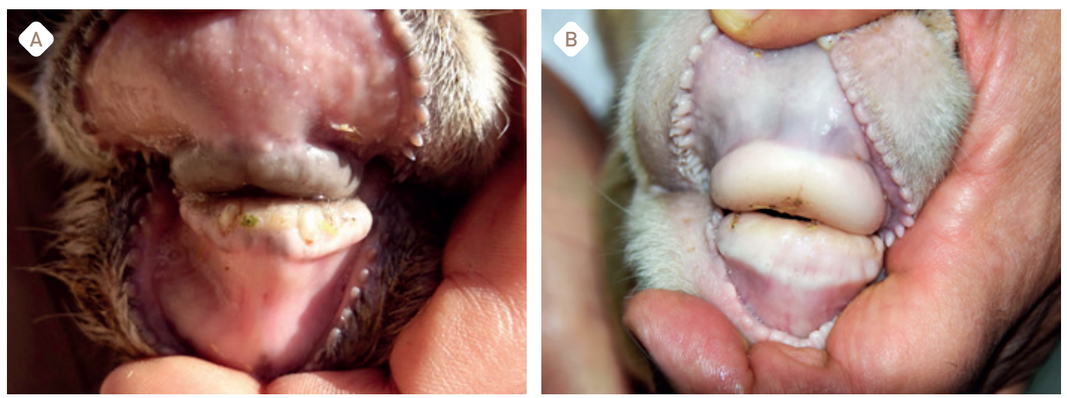
4.161. **Displacement of incisors.** The incisors can become malpositioned after trauma that has not resulted in their loss (A and B).



4.162. **Incisors loss.** With age, the physiological loss of teeth can occur. In young animals it can be due to gingival disease or deficiency disorders that affect the incisor (A and B).



4.163. **Incisors loss.** Old animals' incisors have shallow roots and are worn, short or broken (A-D).



4.164. **Incisors total loss.** After the wear or total loss of the incisors (A and B), the animals can continue to produce if the food is abundant, they have the molars in good condition, and there is not much competition for the food.

INCISORS FOOD IMPACTION



4.165. **Incisors impaction.** It is common to find food remains between the incisors, especially when the animals are not young and their teeth are misplaced.



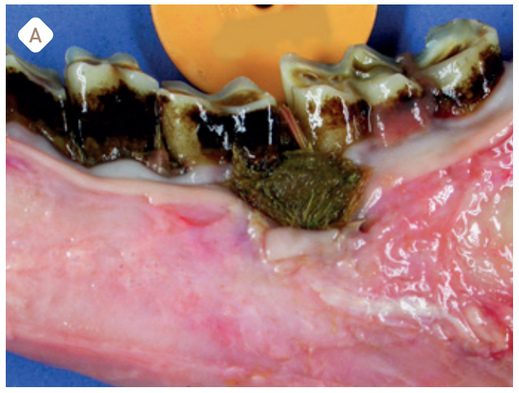


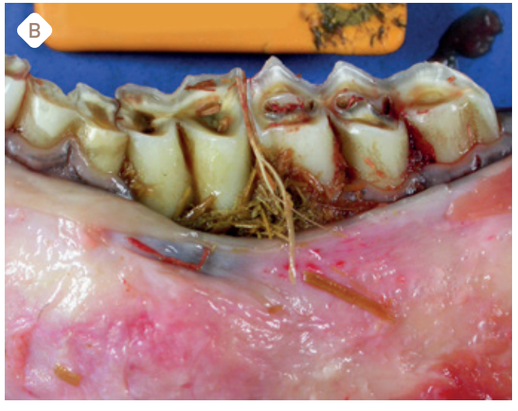
4.166. **Incisors impaction.** It is also common to find wool remains in animals that suffer from itching and bite the wool (A and B) and spikelets on the gums (*see "Spikelets in soft tissues", page 179*).

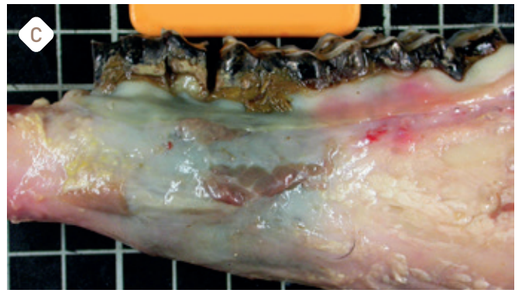
MOLAR FOOD IMPACTION



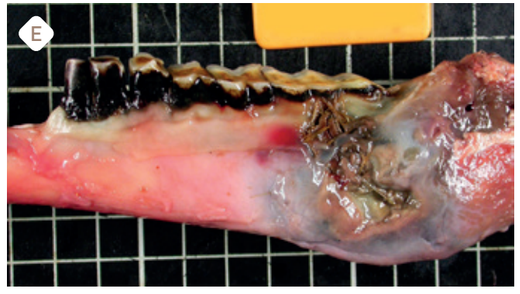
4.167. **Molar impaction.** The impaction of plant material between the tooth and the gum is frequent in adult animals. This material irritates the gingiva, generating gingivitis and retraction.





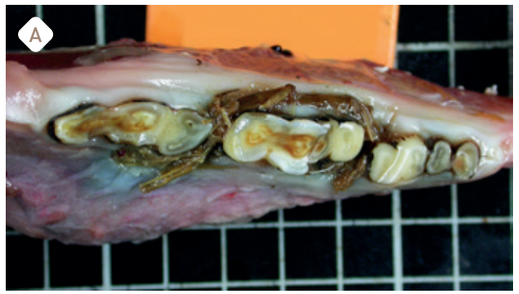


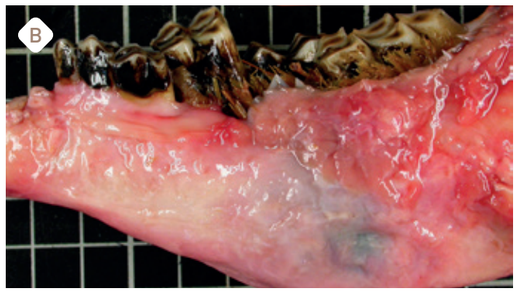


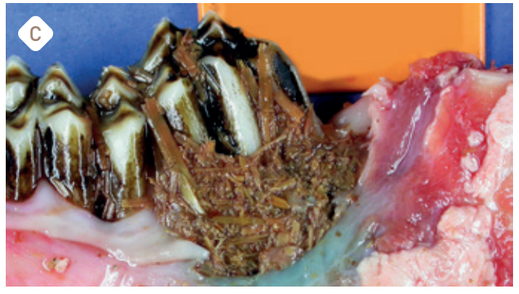




4.168. **Molar impaction.** The impacted material penetrates towards the root, especially in the lower jaw, causing infections around the tooth and periodontitis (A-F).

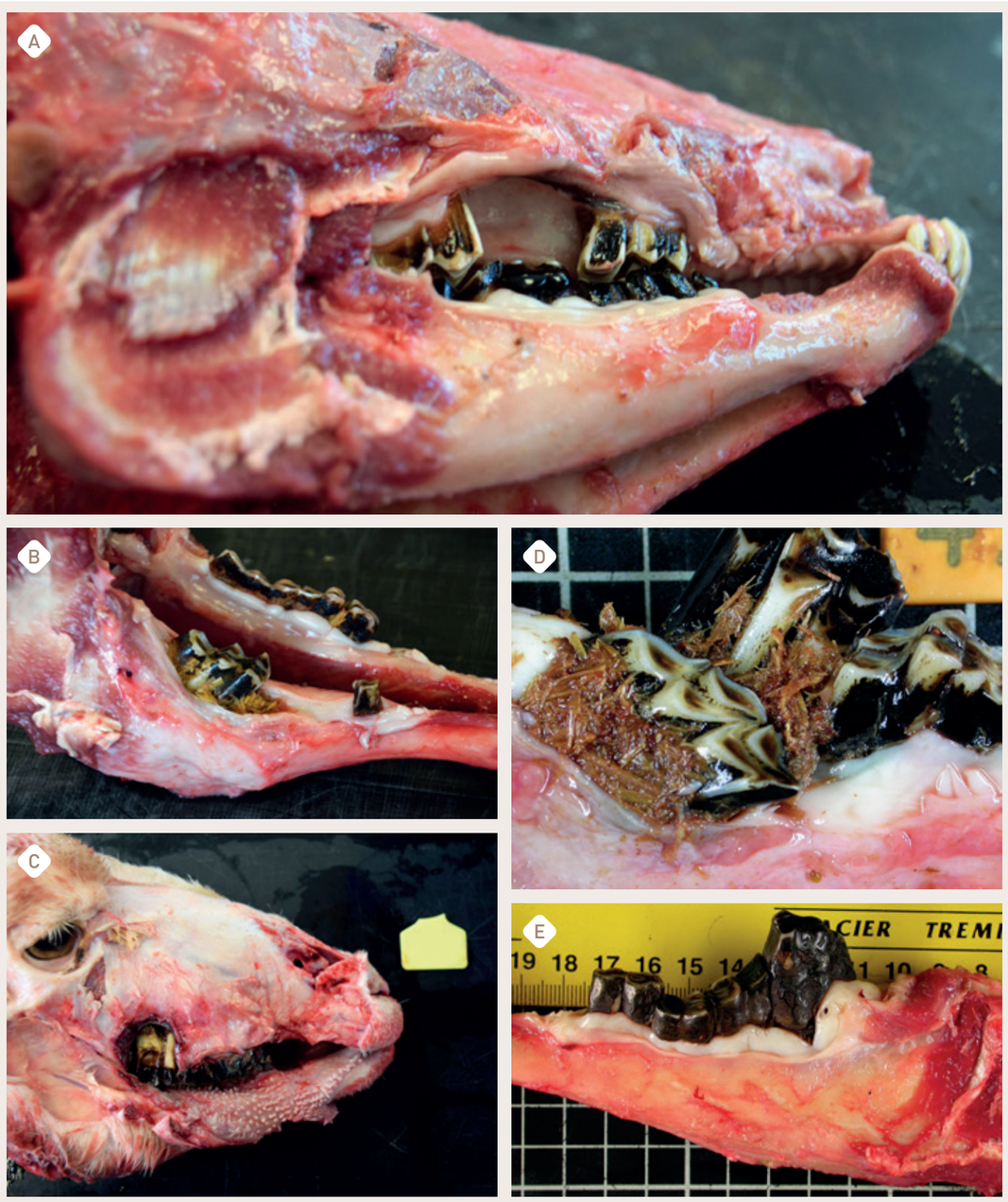






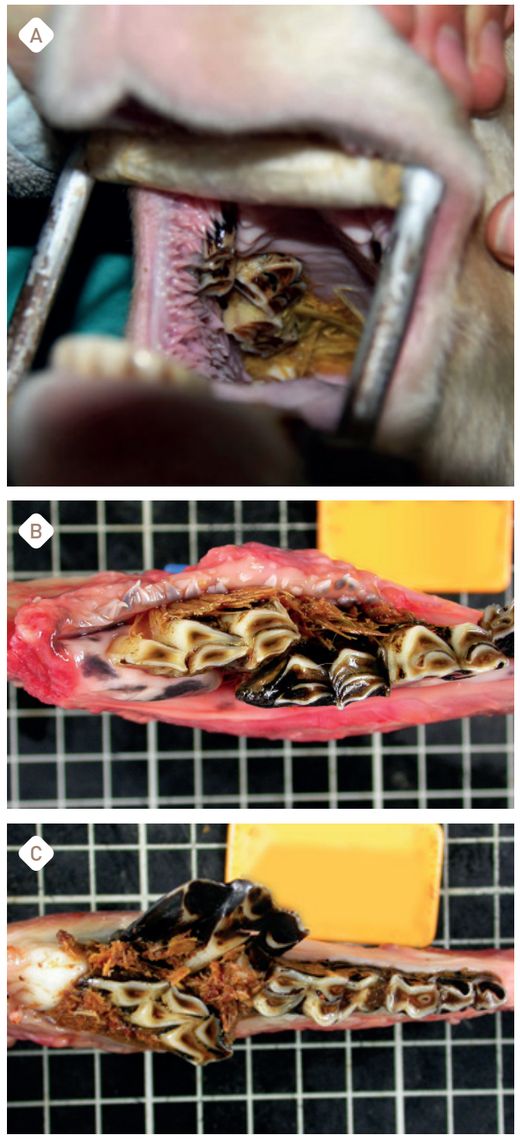


4.169. **Molar impaction.** The consequent infection affects the bone, causing osteitis and facial deformation (A-D).



4.170. **Molar impaction.** Teeth remain loose and generally fall out (A and B). Sometimes the lack of space in the last teeth causes them to remain loose in their space (C and D). The lack of wear on the opposite piece generates abnormal growths that hinder the rumination process (E).

LOSS OF MOLAR ALIGNMENT

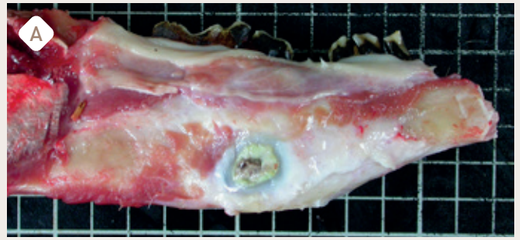


4.171. **Loss of molar alignment.** All the disorders taking place in the anchoring of the teeth cause them to change position and in turn, facilitate the movements of the neighbouring teeth and those of the opposite jaw, finally giving an incorrect alignment that makes feeding difficult (A-C).

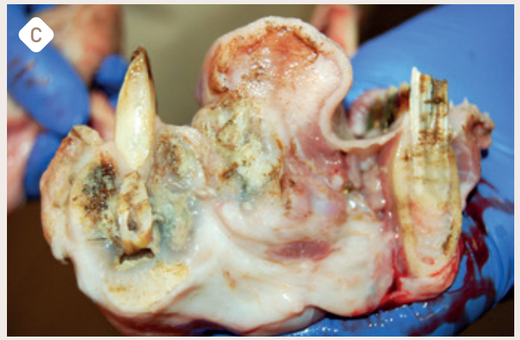
ASYMMETRY AND FISTULAS

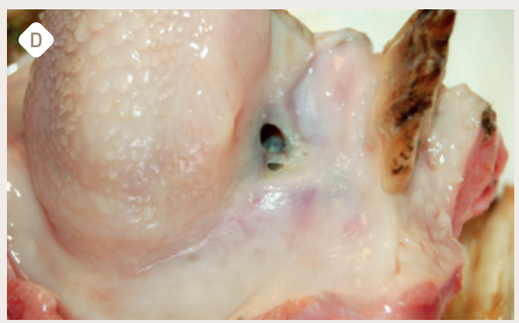


4.172. **Mandibular asymmetry.** The different disorders affecting the molars cause deformation in the implantation area (A). These deformations cause asymmetry in the head (B), easily verifiable on inspection and palpation (C).







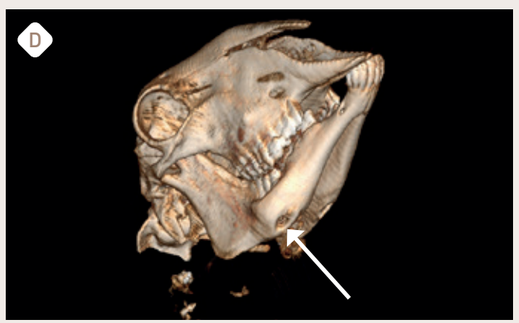


4.173. **Fistula.** Some necrotic disorders that affect the teeth and peripheral area can destroy the bone and fistulise. The fistula can drain into the mouth (A and B), generally draining at the base of the oral cavity (C and D).

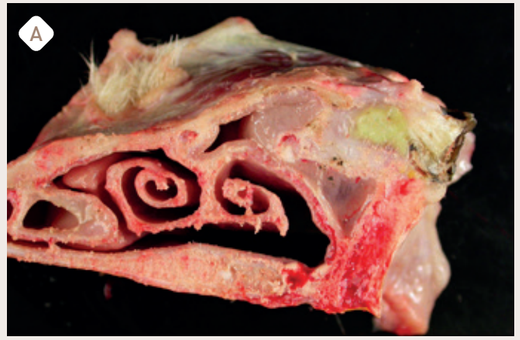




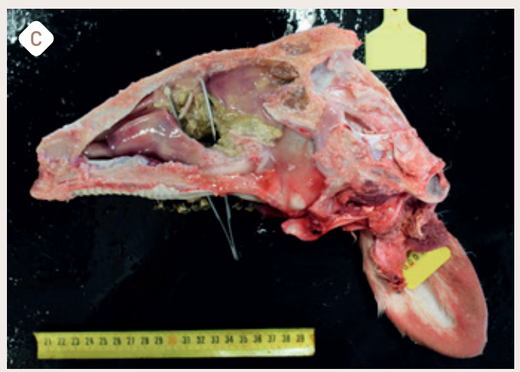




4.174. **Fistula.** The most common fistulas occur from the lower jaw outwards (A-C). A three-dimensional CT scan allows to perfectly visualise the affected bone (D).







4.175. **Maxilla asymmetry.** Less frequent are the disorders affecting the upper jaw or maxilla (A), also altering the symmetry of the face (B). On very few occasions, fistulas can be found into the nasal cavity (C).

ACTINOMYCOSIS





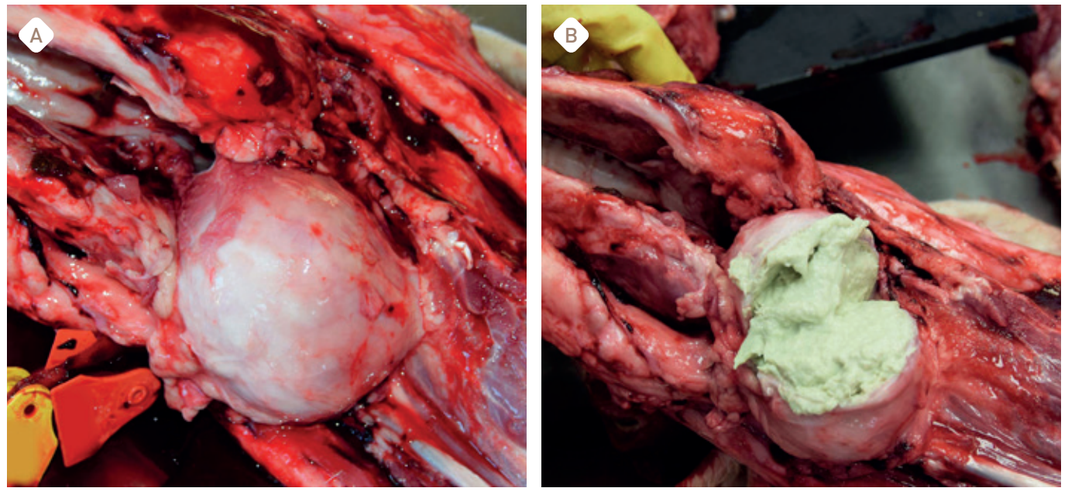


4.176. **Actinomycosis.** It is a sporadic disease that is uncommon in sheep and goats. The principal clinical sign is an enlargement of the maxillary or the mandibular bones that is hard, immovable and painless (A and B). The skin on the lesion is perforated, and a honey-like discharge comes out (C).

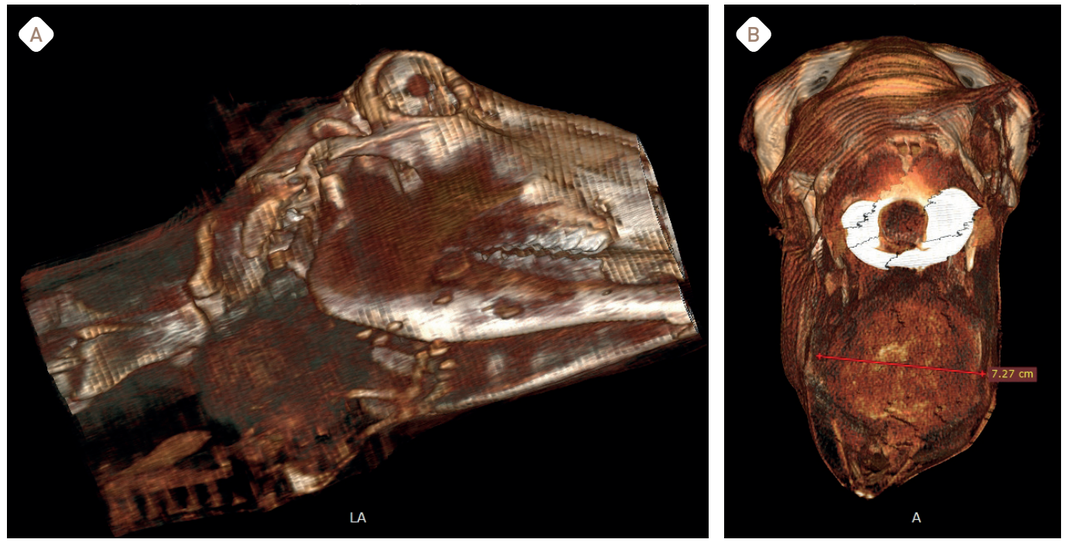
OROPHARYNX AND OESOPHAGUS

In this section, only what influences the disorders of the digestive system will be discussed. The rest will be addressed with the respiratory system.

CASEOUS LYMPHADENITIS (CLA) LESIONS IN THE OROPHARYNX

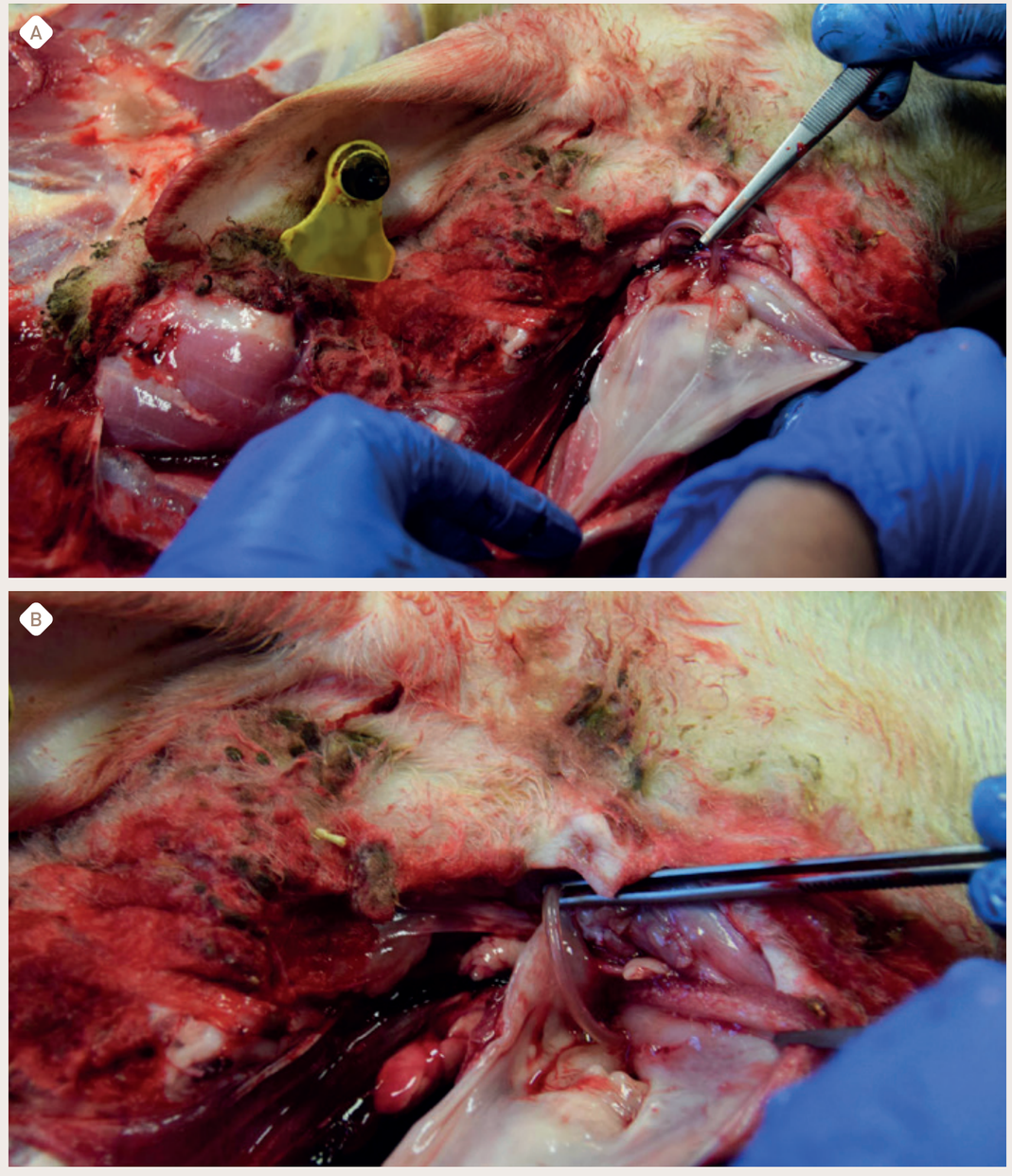


4.177. **CLA pyogranulomas in the oropharynx.** Pyogranulomatous lesions caused by *Corynebacterium pseudotuberculosis* in the oropharynx (A and B). When they are large, they hinder swallowing.



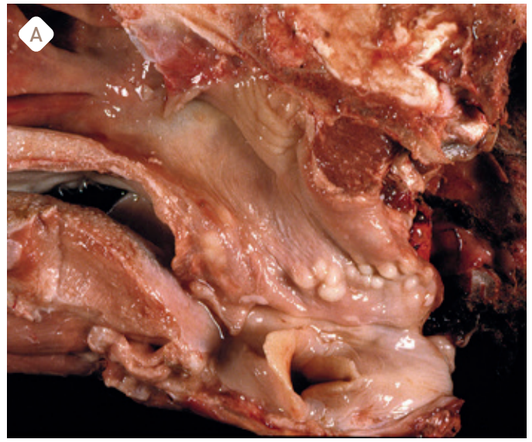
4.178. **CT scan.** Through the 3D reconstruction of the CT scan, it is possible to appreciate the real position of the 7 cm pyogranuloma that is pressing the oesophagus and displacing the glottis ventrally (A and B).

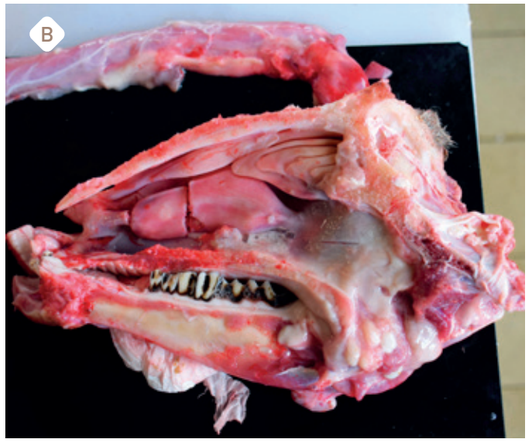
FOREIGN BODIES IN THE OROPHARYNX



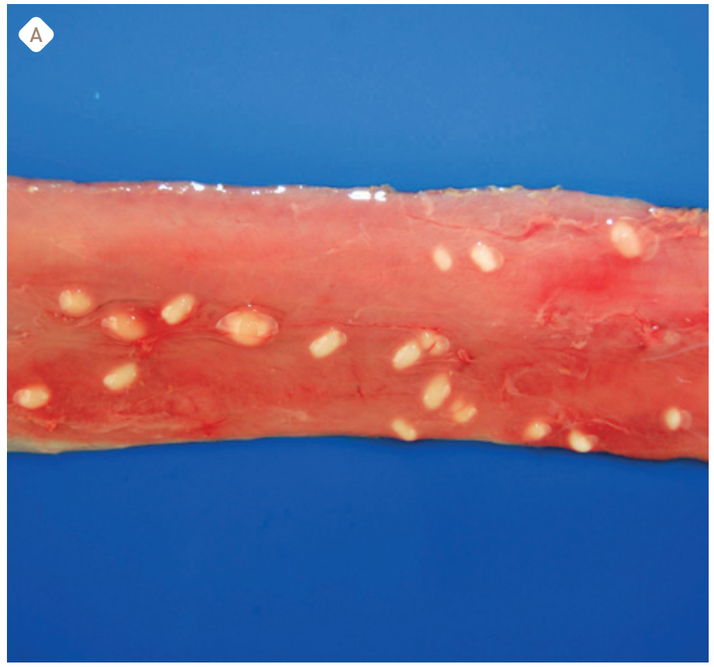
4.179. **Foreign bodies in the oropharynx.** Sometimes it is possible to find foreign bodies that make swallowing difficult, such as the broken nasobronchial tube that appears in the photograph above the glottis and entrance of the oesophagus (A and B).

SARCOCYSTOSIS





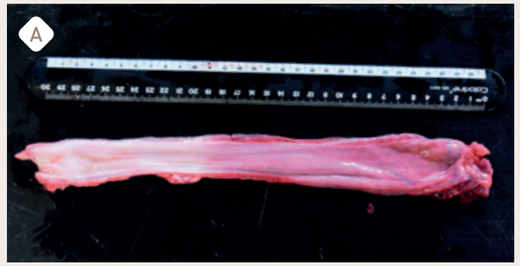
4.180. **Sarcocystosis in the oropharynx.** Different types of *Sarcocystis* are found from the oral cavity to the anterior parts of the digestive system. Presentation in the laryngeal area (A and B) can give similar clinical signs to a laryngeal paralysis, and it may also affect the functionality of the tongue and oesophagus.





4.181. **Sarcocystosis in the oesophagus.** As well as the oropharynx, the oesophagus may be affected by *Sarcocystis* (A and B). Despite being found in large numbers in many cases, they are rarely related to the alteration of oesophagal functionality.

OESOPHAGITIS AND ULCERS



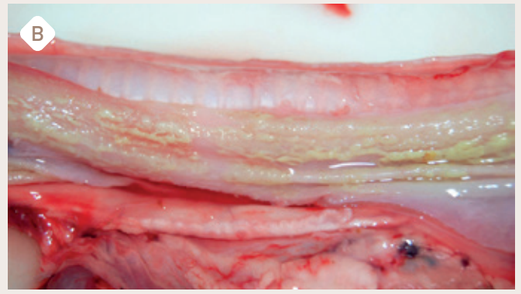


4.182. **Oesophagitis.** Various physical, chemical or microbiological agents, especially viruses, can cause inflammation of the oesophagus (A and B).



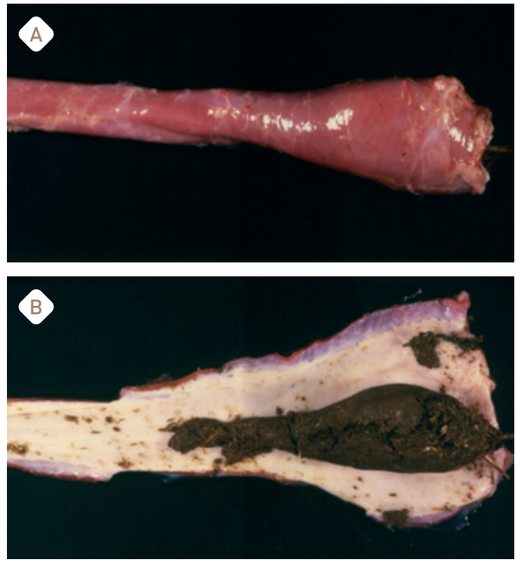
4.183. **Ulcers.** In many cases, oesophagitis evolves, forming ulcers.



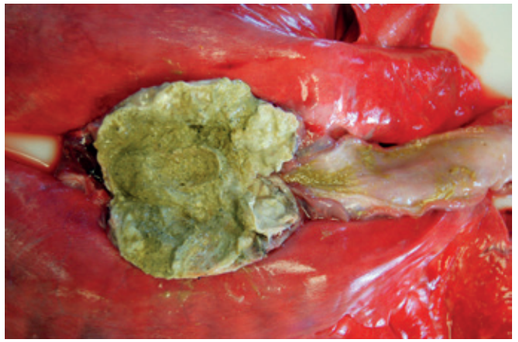


4.184. **Ulcers.** These ulcers are usually covered with fibrin in their resolution (A and B).

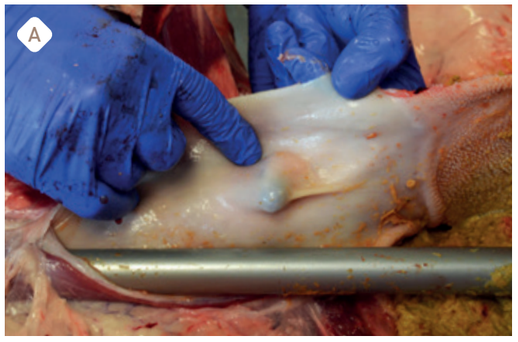
MEGAOESOPHAGUS



4.185. **Megaoesophagus.** The oesophagus can accumulate food, increasing its diameter close to the cardias (A and B) or in the mediastinal area (C), eventually collecting large amounts of food (D).

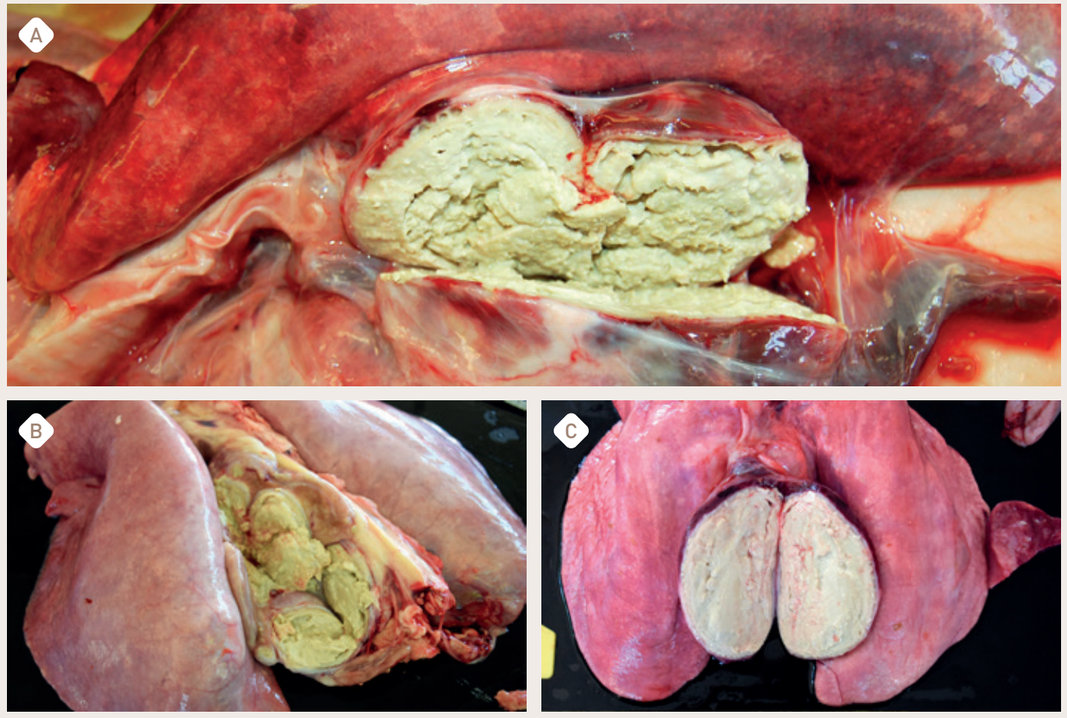


4.186. **Megaoesophagus.** When some obstruction prevents the regurgitation of food to be ruminated, the food found in the oesophagus has a foamy aspect due to the longer time it remains in the rumen subjected to ruminal movements.

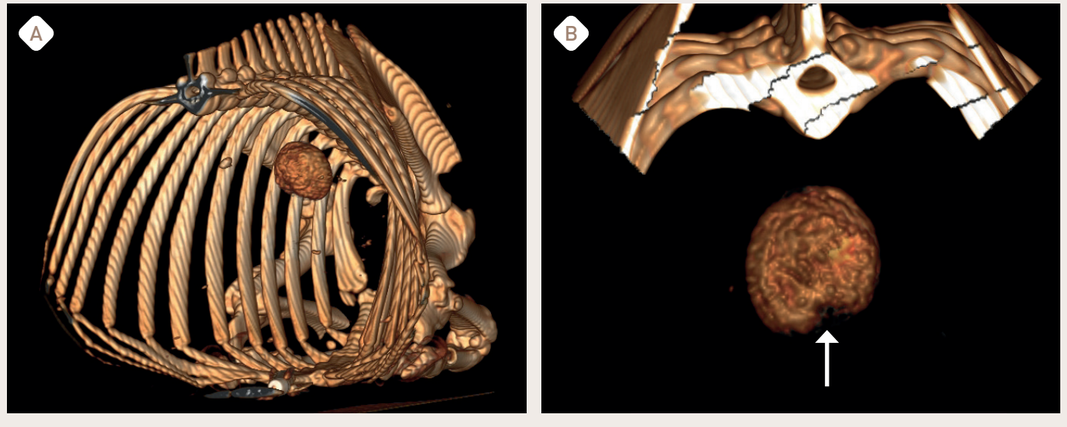


4.187. **Dilation of the oesophagus.** Dilations are generally associated with distension or rupture of some of its muscular layers (A and B).

EXTERNAL PRESSURE OF THE OESOPHAGUS



4.188. **External pressure on the oesophagus.** The functionality of the oesophagus can be compromised by external pressure on the wall, as is the case of large lung abscesses or CLA pyogranulomas, especially those located in mediastinal lymph nodes (A-C).

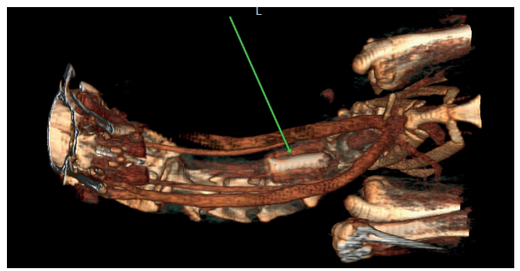


4.189. **External pressure on the oesophagus.** These 3D reconstructions of a CT scan of the chest show the large mediastinal lymph node with the ventral depression through which the compressed oesophagus passed (A and B).

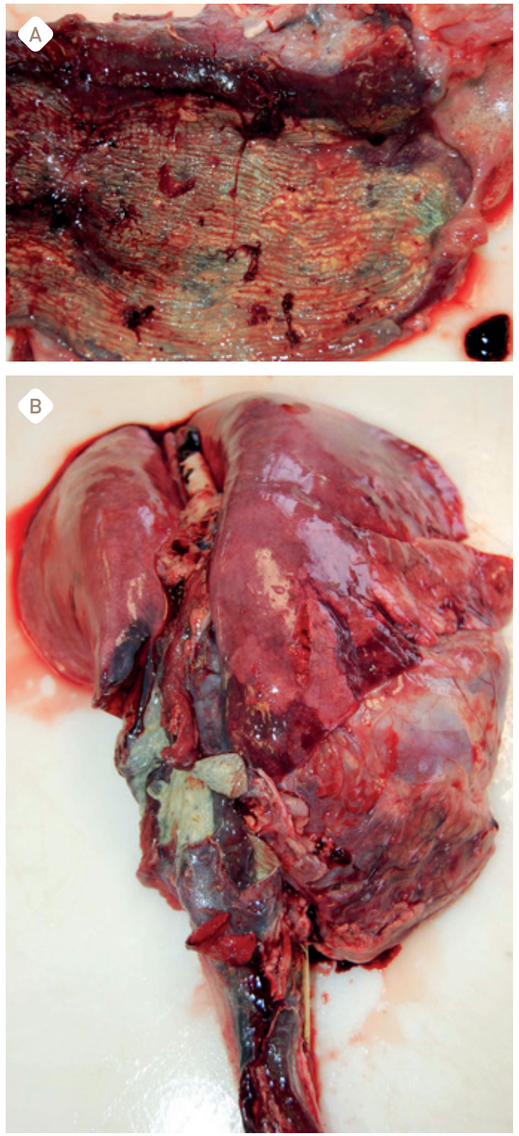
INJURIES DUE TO IMPROPER MANAGEMENT



4.190. **Injuries due to improper management.** The application of ruminal boluses or microchips must be performed properly (A and B).



4.191. **Injuries due to improper management.** The 3D reconstruction of a CT scan shows the neck of a young sheep with scoliosis caused by misapplication of the ruminal microchip. The ceramic bolus containing the microchip can be seen embedded in the wall of the oesophagus (green arrow).



4.192. **Injuries due to improper management.** Poor technique for the introduction of ruminal boluses or microchips can cause injuries to the oesophagus and even perforations, especially in animals that have not reached adequate development or in restless animals without proper containment (A and B).



4.193. **Oesophageal tear.** Husbandry practices to introduce probes or similar can cause serious injuries in the oesophagus wall, with subsequent exit and accumulation of food and even fistulisation to the outside, especially if it is the cervical portion (A-C).

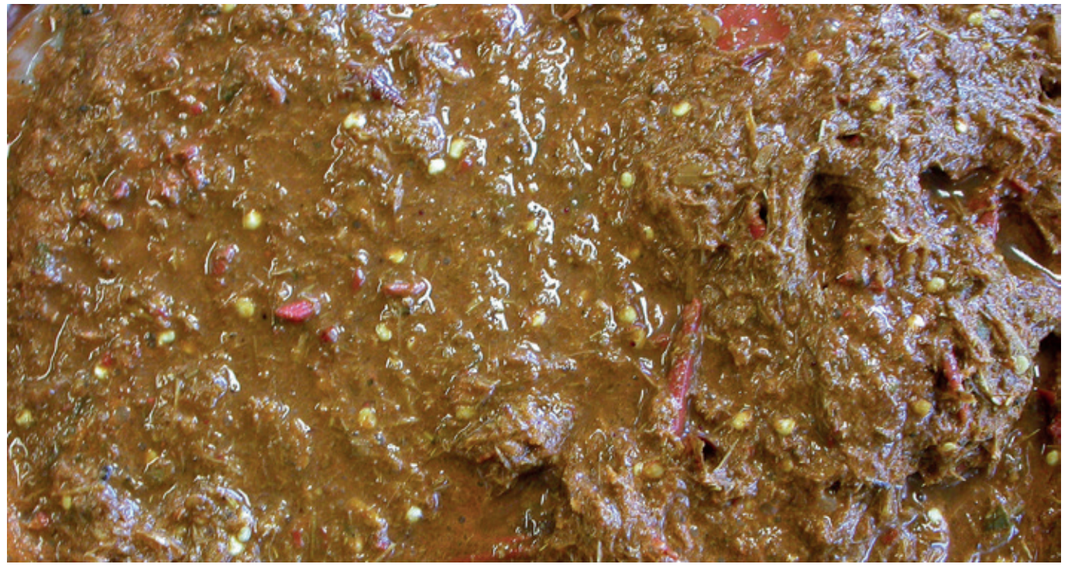
STOMACH

RUMEN OVERLOAD

The feeding behaviour of small ruminants makes them prone to rumen overloads. Overload is usually accompanied by ruminal atony, and on many occasions, dysbiosis is generated with the growth of pathogens such as Clostridia or *E. coli*.



4.194. **Rumen overload.** In cases of overload with ruminal atony, it is common to find abundant and dry content (A and B).



4.195. **Rumen overload.** On other occasions, depending on the characteristics of the food, and if there is no absence of rumen movements, we can find abundant and more liquid content. In the picture, rumen overload due to excessive consumption of peppers.





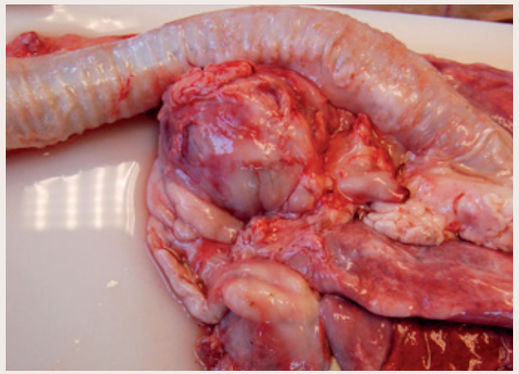
4.196. **Rumen overload due to obstruction.** Occasionally, rumen overload is caused by a content transit disorder caused by foreign bodies (A and B) or by bezoars that occlude the intestine after passing the pylorus (C and D). Generally, overload disorders are accompanied by acute bloating, in addition to ruminal acidosis or alkalosis, when the diet is unbalanced.

BLOAT

Bloat is an overdistension of the rumen, reticulum, omasum or abomasum due to gases of fermentation accumulation. Two types of bloats can occur in small ruminants: frothy bloat (primary ruminal tympany), associated with consumption of tender legumes, and free-gas bloat (secondary ruminal tympany) related to excessive consumption, especially of cereals, or by eructation disorders or lack of motility of the pre-stomachs.



4.197. **Clinical signs.** Bloating can cause sudden death by suffocation with abdominal distension in affected animals.



4.198. **Oesophageal obstruction.** It can be due to external pressure (abscesses), or megaoesophagus.



4.199. **Bloating.** Usually occurs acutely, which can lead to death if left untreated (A-D).



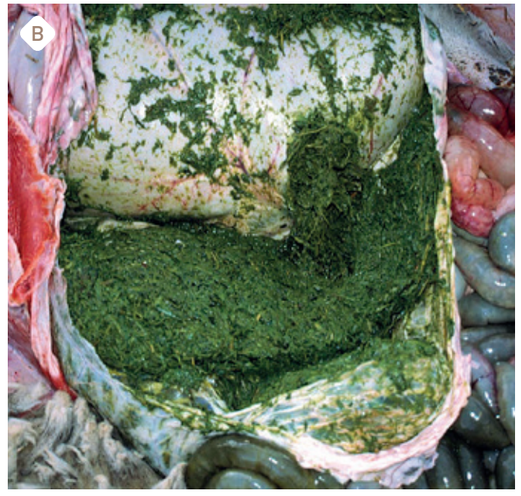




4.200. **Frothy bloat.** Some forages, such as alfalfa or clover, especially if consumed tender and very cold, can cause frothy bloat with the production of a stable foam that traps the normal gases of fermentation in the rumen (A-C).

RUMINAL ALKALOSIS





4.201. **Ruminal alkalosis.** Polyphytic grasses, with an excessive predominance of legumes, such as clover (A), favor alkalosis, especially if there has been no prior habituation to that diet. It can be accompanied by bloating and even rupture of the rumen before or after death (B). It can also be found when non-protein nitrogen is supplemented to the diet and in urea, nitrate and nitrite poisoning.

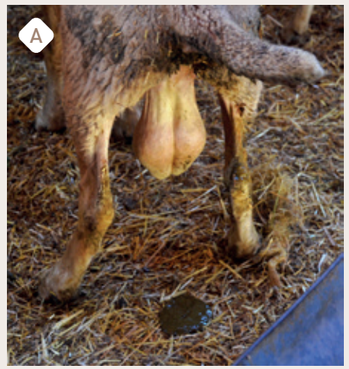
RUMINAL ACIDOSIS

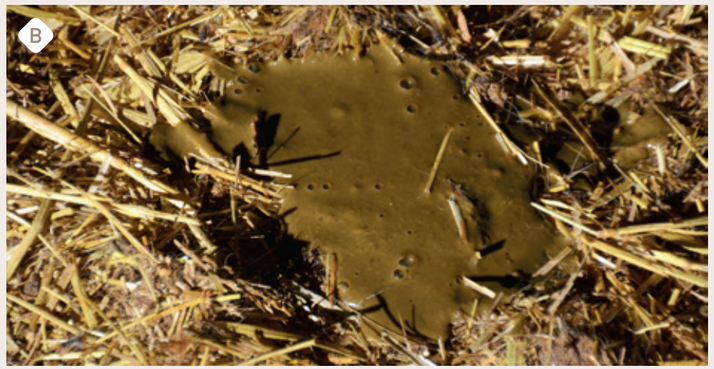


4.202. **Carbohydrate overload.** Ruminal acidosis is generally produced by consuming diets rich in carbohydrates, without balancing the protein and fibre, in large quantities or in a short period of time, which prevents the accustoming of the rumen flora.

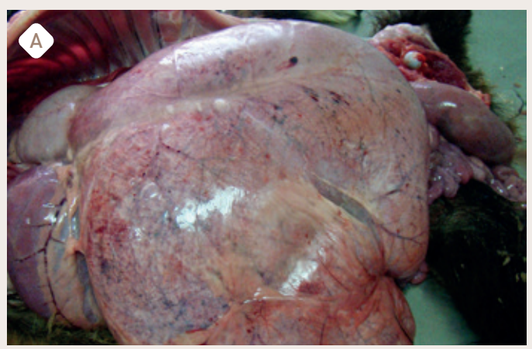


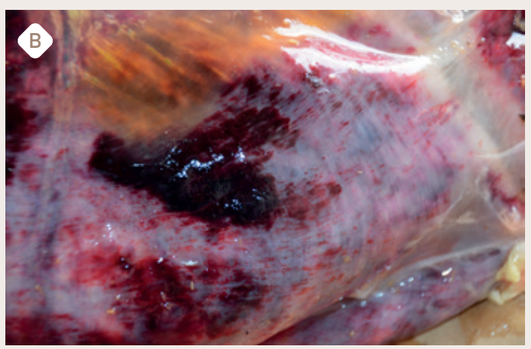
4.203. **Depressive states.** Acute ruminal acidosis can cause bloating and sudden death of the animal, or it can progress to metabolic acidosis, causing depressive states in the animal.





4.204. **Diarrhoea.** In less severe cases, it causes fluid diarrhoea with a foul-smelling odour, eliminating part of the ingested content. When diarrhoea occurs, there is still digestive mobility, which is a sign of possible recovery of the animal (A and B).





4.205. **Acute ruminitis.** At necropsy, animals present with acute ruminitis and haemorrhages that can be seen from the serosa (A and B).



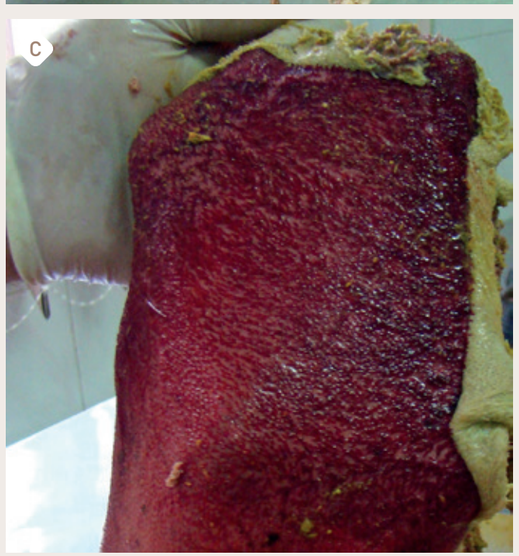




4.206. **Rumen content.** The necropsy of the animal shows ruminal content with abundant carbohydrates, generally cereal with whole grain (A) or ground (B). The pH of the ruminal fluid is clearly acidic (C).







4.207. **Mucosal detachment.** In severe cases, ruminitis is accompanied by a detachment of the mucosa of more or less extensive areas, showing a hyperemic submucosa (A-C). *Picture B courtesy of Dr. A. Akbari.*

ACUTE RUMINITIS

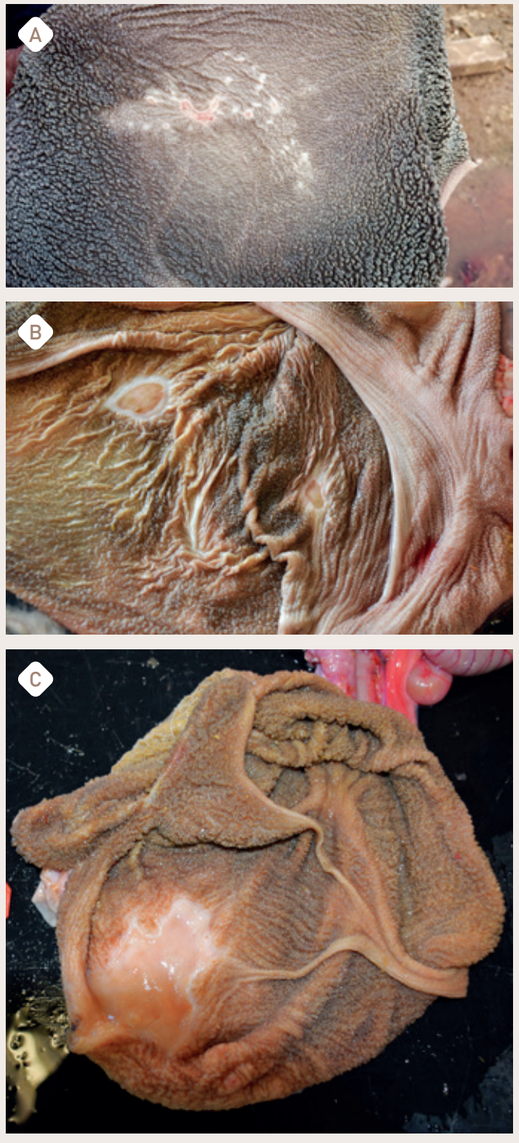
In addition to acidosis and alkalosis, various infectious, parasitic and toxic disorders can cause acute inflammation of the ruminal mucosa.



4.208. **Acute ruminitis.** In acute ruminitis, we can see the reddish mucosa affecting large areas (A-C).

RUMINAL ULCERS

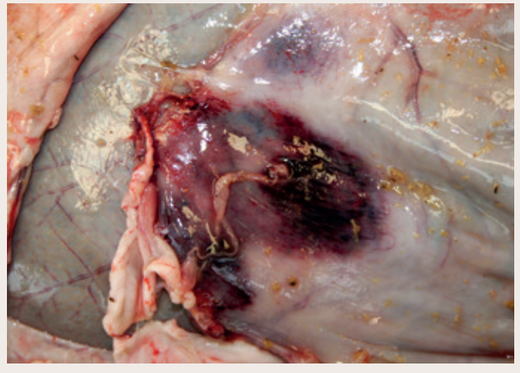
Chemical or biological aggressions on the ruminal mucosa can lead to the presence of ulcers.



4.209. **Ruminal ulcers.** Ruminal ulcers vary in shape and size, from several millimetres (A) to several centimetres (B and C).

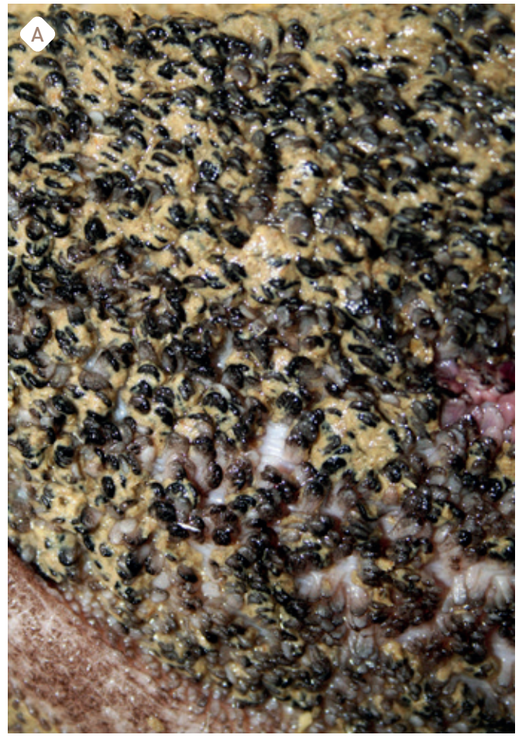


4.210. **Healed ulcers.** The scar retraction of the ulcers produces star shapes (A) that can converge and occupy large areas of the ruminal wall (B).



4.211. **Perforated ulcers.** Occasionally, perforation can affect all layers and cause local peritonitis, with fibrinous reaction or generalised peritonitis.

HYPERKERATOSIS AND PARAKERATOSIS





4.212. **Hyperkeratosis and parakeratosis.** Nutritional imbalances with alterations in pH and fibre deficiencies are the leading causes of excessive keratinisation with hardening and enlargement of the rumen papillae (A and B).

BEZOARS



4.213. **Bezoars.** Concretions of plant debris, hair, wool, foreign fibres and minerals come together to form trichobezoars and phytobezoars. These foreign elements can be located in the rumen (A and B) and the abomasum (C and D). Some bezoars can pass into the small intestine and cause intestinal occlusions due to their size. If the bezoar is not eliminated in faeces, it will cause the death of the animal (*see "Rumen overload", page 208*).

FOREIGN BODIES





4.214. **Foreign bodies in food.** Easy access to foreign bodies during feeding leads to ingestion of these products, especially plastic and feed packaging (A-C).



4.215. **Ingested foreign bodies.** Once ingested, if they are not eliminated in faeces, they accumulate to form a consistent mass that makes digestion difficult (A and B).



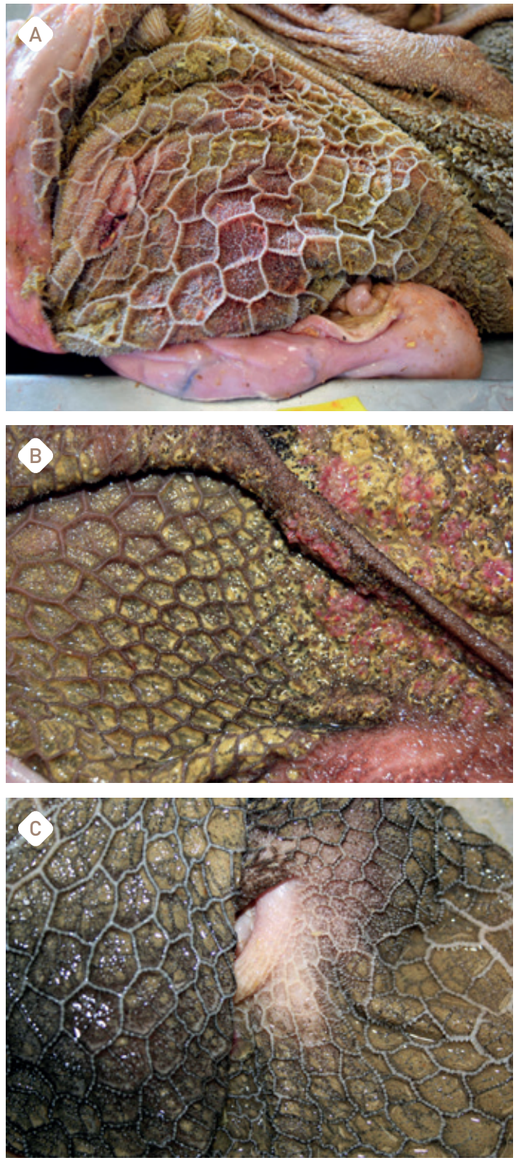
4.216. **Foreign bodies and bezoars.** Sometimes, foreign bodies and bezoars coexist. This can cause occlusions and even rupture of the digestive viscera.

RUMINAL PARASITOSIS

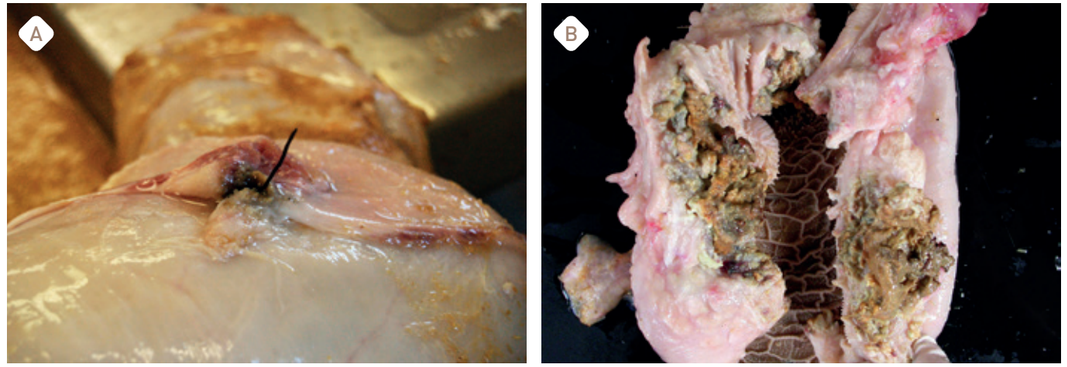


4.217. **Parasitosis caused by *Paramphistomum* spp.** The rumen is the usual location of *Paramphistomum* spp. that can occupy large areas of the mucosa (A-C).

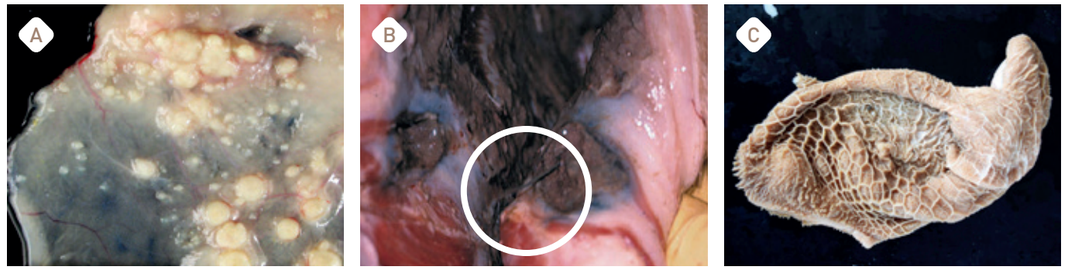
RETICULUM DISORDERS



4.218. **Reticulitis.** As happened in the other stomach of small ruminants, the reticulum can undergo different inflammatory disorders (A and B). Those caused by food imbalance and maintained over time can give rise to reticulum hyperkeratosis, as was seen in the rumen (C).



4.219. **Traumatic reticulitis.** A particular case of reticulitis is produced by puncturing foreign bodies affecting the reticulum wall (A), which can cause abscesses in the reticular wall (B).



4.220. **Traumatic reticuloperitonitis.** Peritonitis is generated if the sharp object goes through the reticulum wall and reaches the peritoneum (A). The stabbing foreign body can progress and damage other organs (B). It can be resolved spontaneously when the process is benign, leaving a scar on the wall (C).



4.221. **Reticulo-omasal obstruction.** The passage of content from the reticulum to the omasum can be obstructed by foreign bodies. The picture shows the deformation of the object obstructing the hole.



4.222. **Anterior vagal syndrome.** The damage of some branches of the vagus nerve causes a particular case of reticulo-omasal obstruction. The vagal branch that innervates most of the omasal reticulum foramen can be affected by external pressures. That is the case of calcified *Cysticercus* over the vagal branch that innervates the reticulo-omasal opening.

OMASUM



4.223. **Omasitis, ulcers and bleeding.** In addition to the vagal disorders described previously, the omasum can suffer injuries like the other stomachs. The picture shows circular swellings, fibrin-coated ulcers, and a haemorrhage.

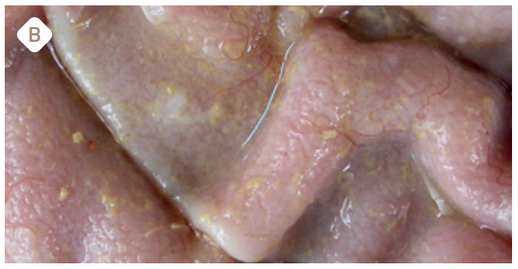


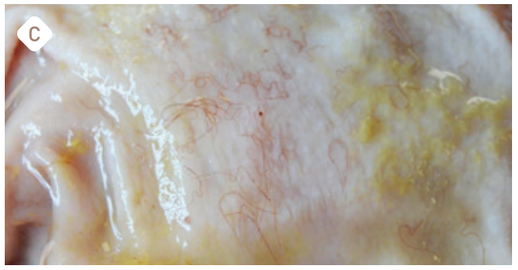
4.224. **Abscesses.** An abscess affecting the liver and omasum wall.

PARASITIC ABOMASITIS

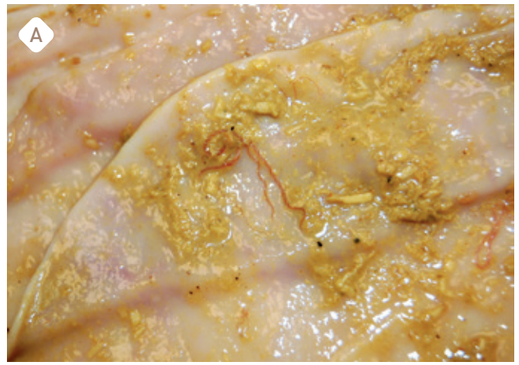
The digestive system of small ruminants can be parasitised by different species, being *Teladorsagia* (formerly, *Ostertagia*) and *Haemonchus* spp. the most prevalent in the abomasum.
The migration of *Teladorsagia* larvae causes abomasitis with the presence of parasitic nodules where the larva will remain as larva IV until leaving the nodule to complete its development into adults. Under unfavourable circumstances, the larvae can remain in hypobiosis for long periods of time.

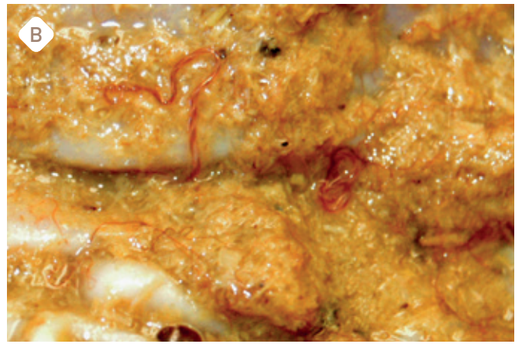






4.225. ***Teladorsagia* spp.** Mucosa with a large number of parasitic nodules on the abomasal wall, in a hypobiosis state, during winter (A). Nodules and adults with their characteristic small size and reddish colour (hematophagous) (B) and adults with the abomasal wall without nodules (C).



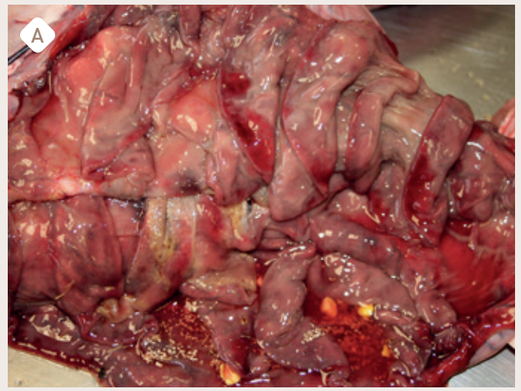


4.226. ***Haemonchus contortus.*** *Haemonchus* is much larger than *Teladorsagia* and has the typical red and white bands (barber pole) (A). Sometimes we can find both species in the same animal (B).



4.227. **Abomasitis.** High infestations cause abomasitis with traces of blood staining the contents. Haemonchosis can cause severe anaemia and death.

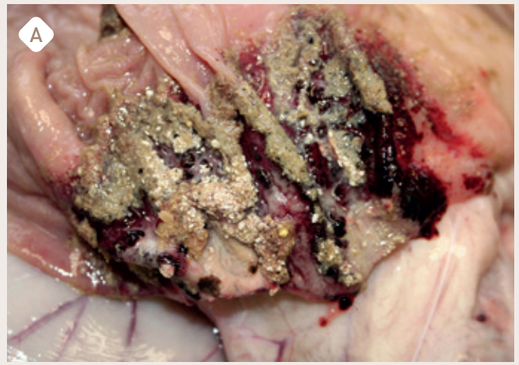
INFECTIOUS ABOMASITIS







4.228. **Infectious abomasitis.** The inflammation of the abomasum can vary from catarrhal (A) to haemorrhagic, with small petechiae (B) or extensive haemorrhages (C).





4.229. **Clostridial abomasitis.** Many microorganisms can cause abomasitis, but those of the genus *Clostridium* are usually the most frequent. *Clostridium septicum* causes acute haemorrhagic abomasitis (A), while *Clostridium sordellii* causes abomasitis with emphysema of the abomasal mucosa (B).



4.230. **Rupture of the abomasum.** Some severe abomasitis can lead to rupture of the abomasum and subsequent peritonitis and death.

ABOMASAL ULCERS

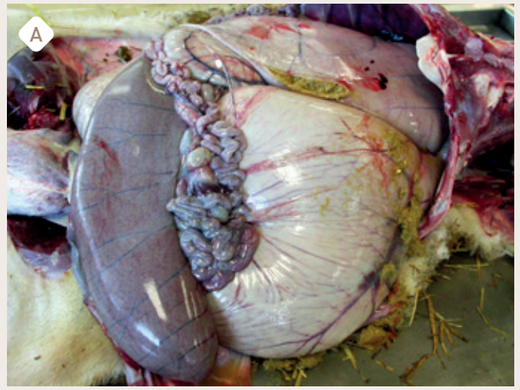


4.231. **Abomasal ulcers.** These ulcers can have a very varied aetiology, mainly related to the pH of its content and infectious agents such as *Clostridium*.



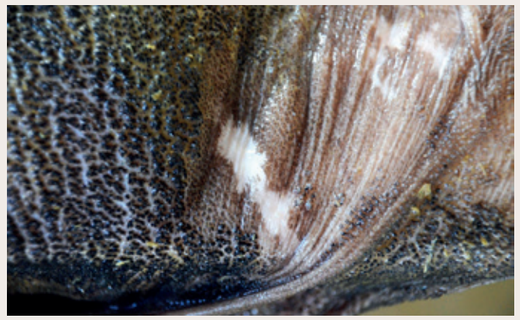
4.232. **Pyloric ulcer.** When abomasal ulcers are near the pylorus, these can interfere with its function, causing the posterior vagal syndrome and food retention.

VAGAL INDIGESTION OR HOFLUND SYNDROME





4.233. **Posterior vagal syndrome.** Disorders of the vagus nerve branch that innervates the pylorus can affect its functionality and cause retention and increase in digestive content (A). In small ruminants, these conditions can be caused by the pressure of calcified cysticerci in the omentum of the pyloric proximities (B).



4.234. **Reflux ulcers.** In this disorder, it is common to find stomach ulcers due to the reflux of hydrochloric acid from the abomasum.







4.235. **Pyloric ulcers (see abomasal ulcers).** Vagal indigestion can be caused by the presence of ulcers in the vicinity of the pylorus (A), generating retention of the content (B and C).

INTESTINE

ENTERITIS

Enteritis is an inflammation of the intestine. In addition to being the primary lesion in diseases as crucial as enterotoxaemia, Johne's disease or gastrointestinal parasitosis, which are discussed below, we can find this lesion associated with multiple aetiologies such as acidosis, alkalosis, different types of toxins, etc.

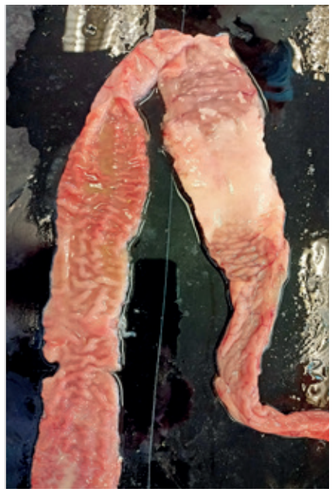




4.236. **Acidosis.** Catarrhal enteritis with a very liquid content is associated with a case of acidosis due to excessive consumption of cereals (A). The caecum is shown to be filled with pasty yellow contents, mainly composed of cereals (B).



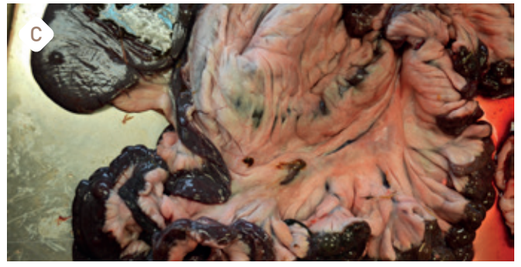
4.237. **Alkalosis.** Catarrhal enteritis with greenish liquid content and related to a case of alkalosis due to excessive consumption of very tender plants.



4.238. **Abrasive agents.** Compounds such as chlorine overdosed for water purification can cause irritation of the intestine and even loss of mucosa in more or less extensive areas.









4.239. **Catarrhal-haemorrhagic enteritis.** Various toxi-infections, including classic enterotoxaemia and other toxic, viral and bacterial infections, can cause different degrees of enteritis from catarrhal (A and B) to haemorrhagic (C). In these cases, it is common to find reactive Peyer's patches (D).

HAEMORRHAGES AND ULCERS







4.240. **Haemorrhages and ulcers.** Some infectious disorders, especially clostridiosis, can cause visible bleeding through the serosa (A) and when opening the intestine (B). Occasionally, they can ulcerate and cross the serosa (C), generating focal or generalised peritonitis.

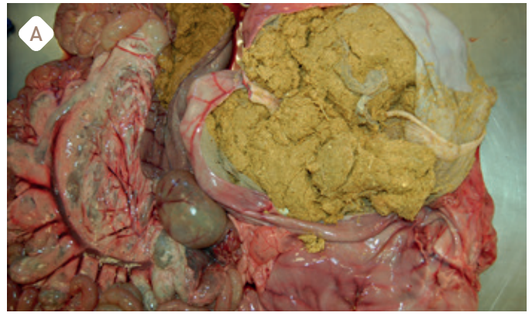
TYPE D ENTEROTOXAEMIA OR PULPY KIDNEY

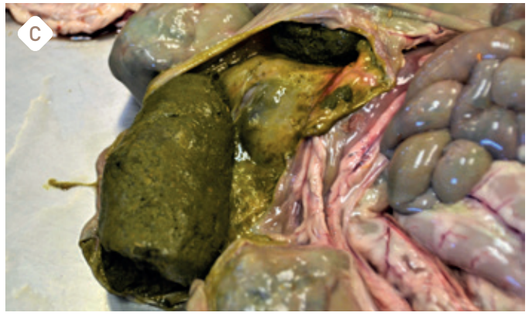
Acute and peracute enterotoxaemia in sheep and goats is caused by *Clostridium perfringens* type D, which produces epsilon (ε) toxin responsible for the damage in different organs.





4.240. **Clinical signs.** This is an acute disorder that causes paralytic ileus and neurological clinical signs with ataxia, myoclonus, pedalling (A), and opisthotonos. Death occurs usually within a few hours and is generally accompanied by bloating (B).





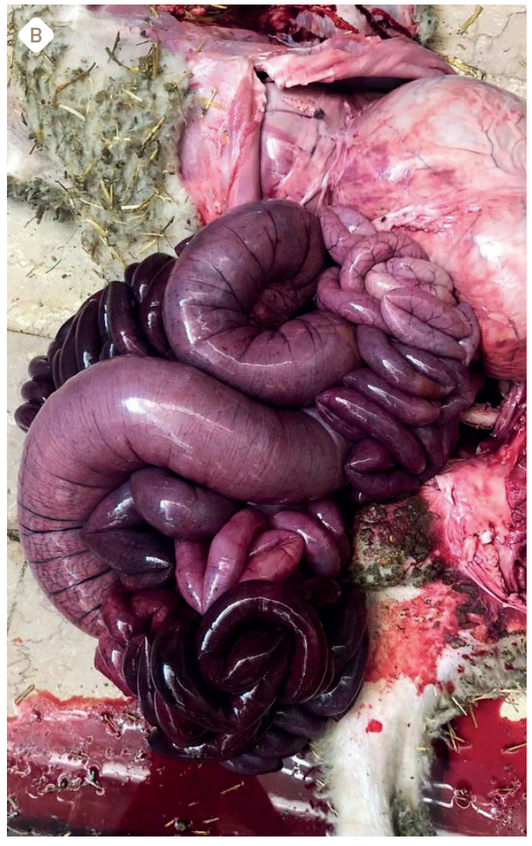


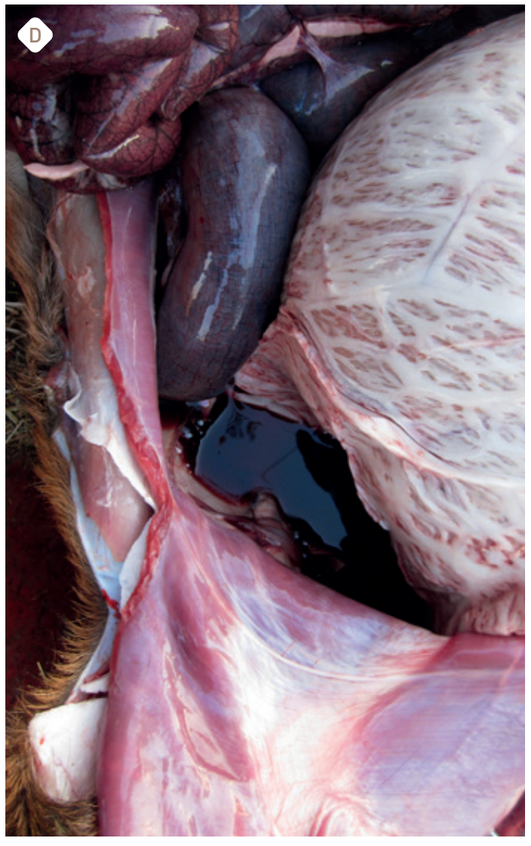


4.242. **Paralytic ileus.** The rumen is usually found with much content, generally dry (A). The small intestine usually presents fluid content or gas-filled loops (B), and the caecum has a more compact content than usual (C). The liver presents a full gallbladder due to a lack of emptying (D).

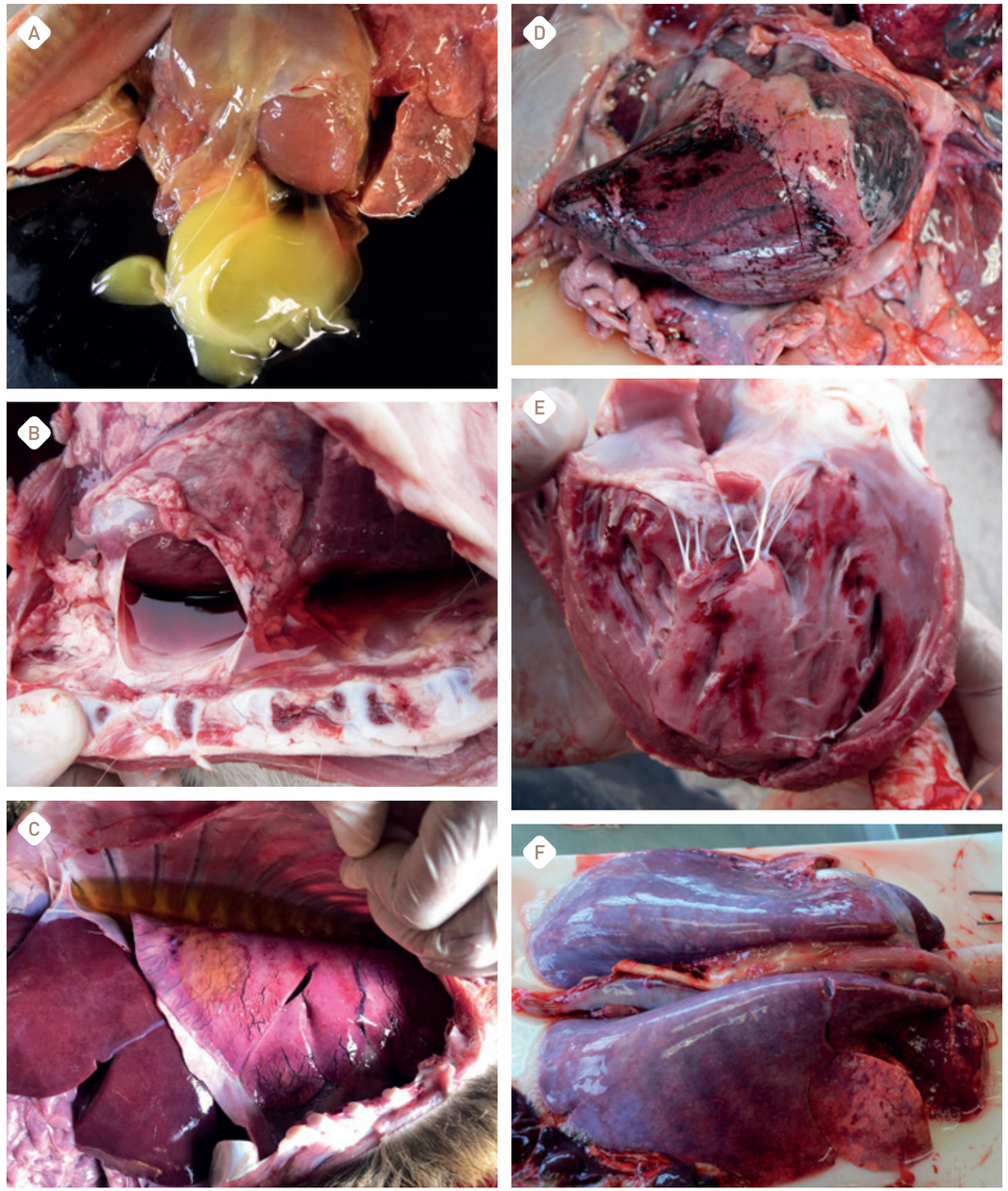




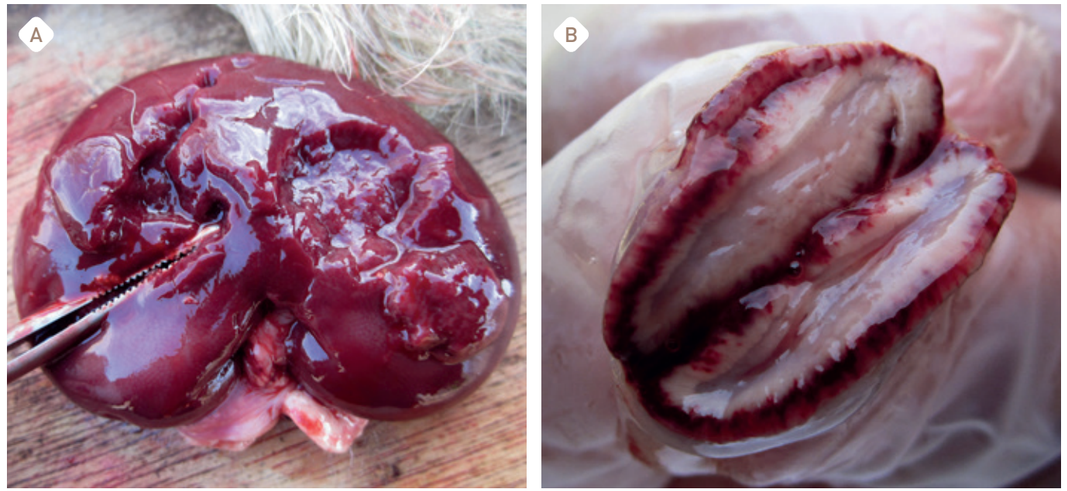




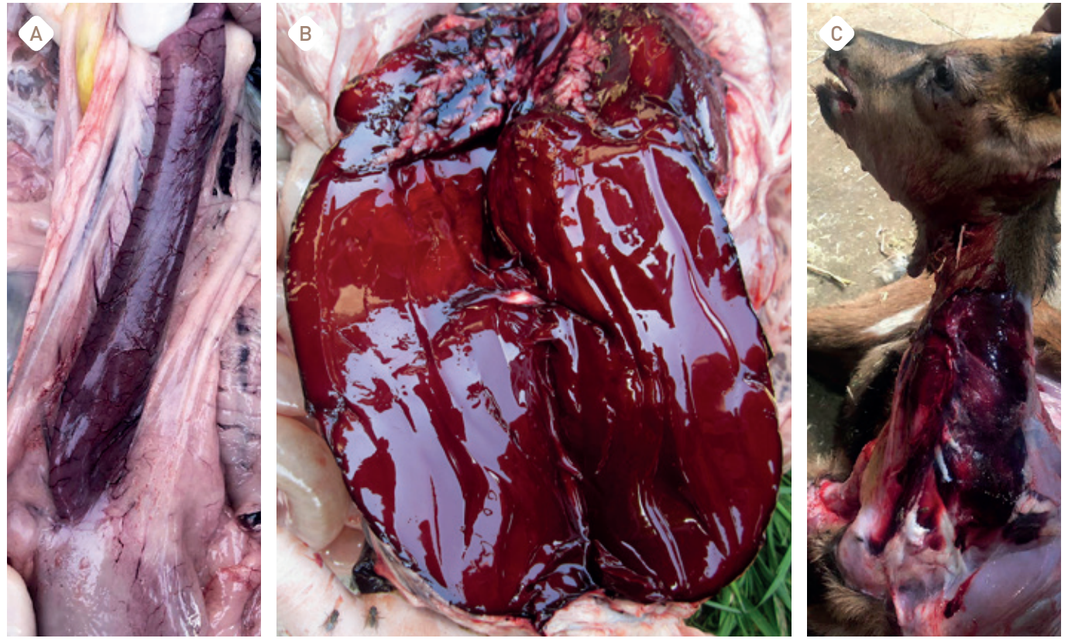
4.243. **Haemorrhagic enteritis.** Haemorrhagic enteritis is one of the predominant postmortem findings in adult animals, predominantly in the jejunum (A and B). Rapid autolysis of the carcass occurs (C) with abundant bloody fluid in the abdominal cavity (D).



4.244. **Pericardial effusion and pulmonary congestion and oedema.** There is clear straw-coloured or bloody pericardial (A and B) and thoracic fluid (C) at necropsy. Many petechiae are present in the epicardium and endocardium (D and E), and there is pulmonary congestion and oedema (F).



4.245. **Pulpy kidney.** The pulpy kidney finding is reliable only in animals that have been dead for a few hours because it is non-specific and merely correlates to a more rapid rate of autolysis (A). The autolytic parenchyma can be removed with pressurised water, leaving only the stroma. Congestion and oedema of peripheral and mesenteric lymph nodes are also found in some cases (B).



4.246. **Abdominal haemorrhages.** Haemorrhages and congestion in the duodenum and abomasum are common signs of type D enterotoxaemia (A). Gelatinous creamy haemorrhage in the mesenteric space (B) and neck bleeding is occasionally seen in goats (C).

TYPE A ENTEROTOXAEMIA

Icteric enterotoxaemia or type A enterotoxaemia is caused by *Clostridium perfringens* type A, especially alpha (a) toxin, although other toxins are also involved. It predominantly affects young animals.

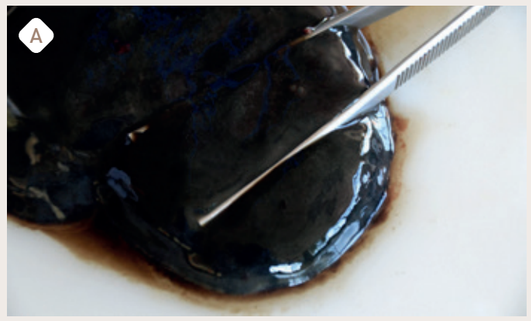


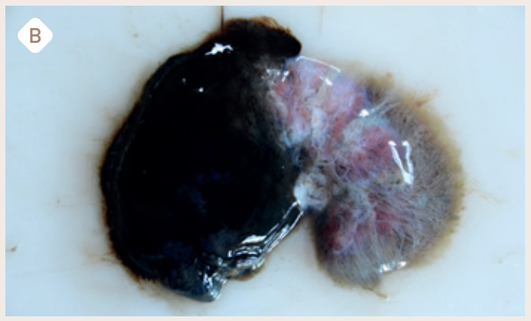


4.247. **Clinical signs.** In this case, the affected animals do not show neurological signs, and the only notable sign is the presence of pale or jaundiced mucous membranes (A and B).



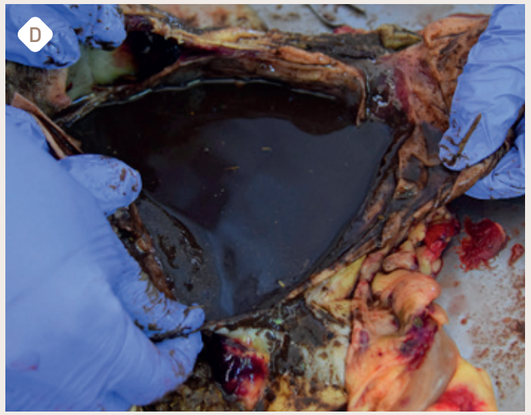
4.248. **Carcass.** At necropsy, a jaundice carcass is observed, especially in the fatty deposits.

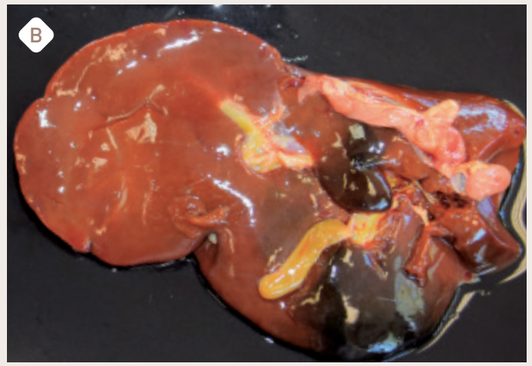




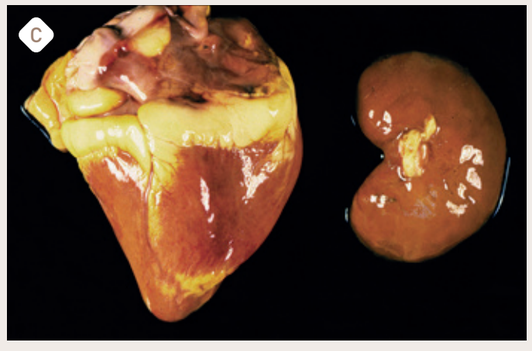
4.249. **Haemoglobinuric nephrosis.** Occasionally, the kidneys are shown black and autolytic (A), and the parenchyma can be detached when a water jet is applied (B).

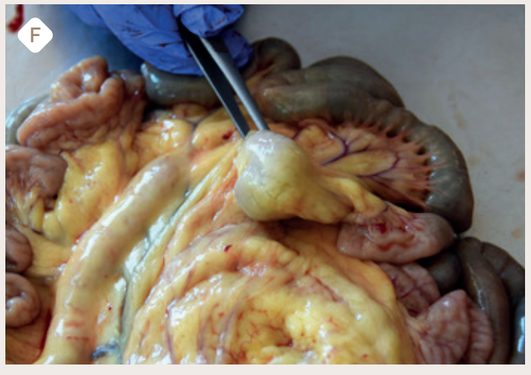








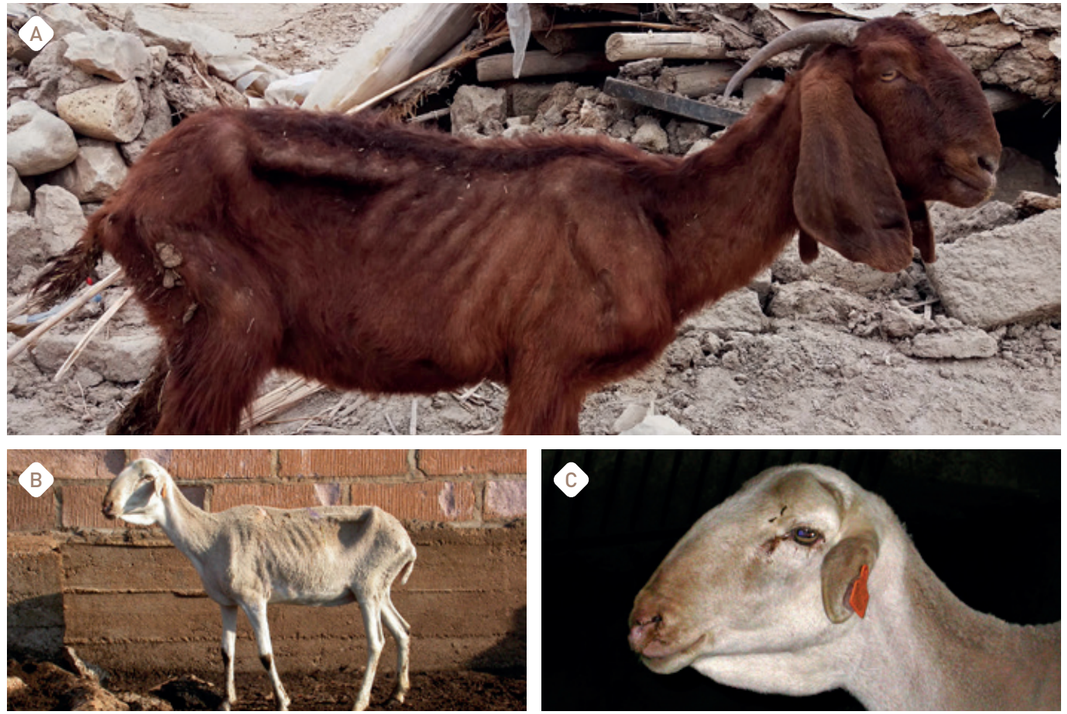




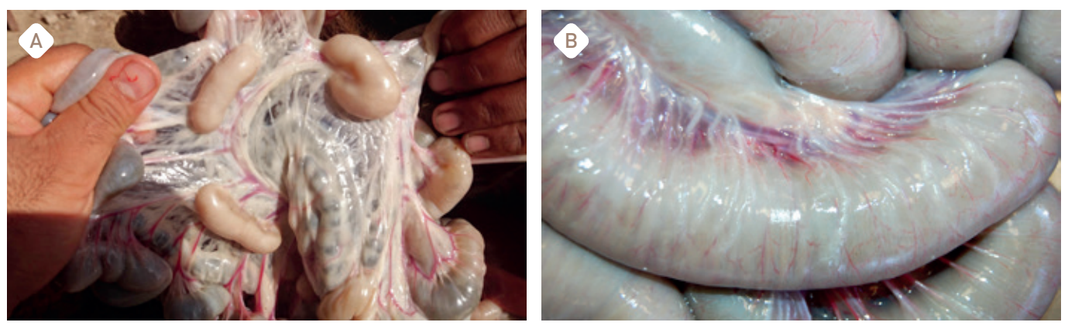
4.250. **Postmortem findings.** Icteric pigments stain the viscera and their contents (A-C). In addition, abomasitis with blackish liquid content and catarrhal enteritis are seen (D). The fat of the omentum is yellow (E), and the swollen lymph nodes (F) stand out.

JOHNE'S DISEASE OR PARATUBERCULOSIS

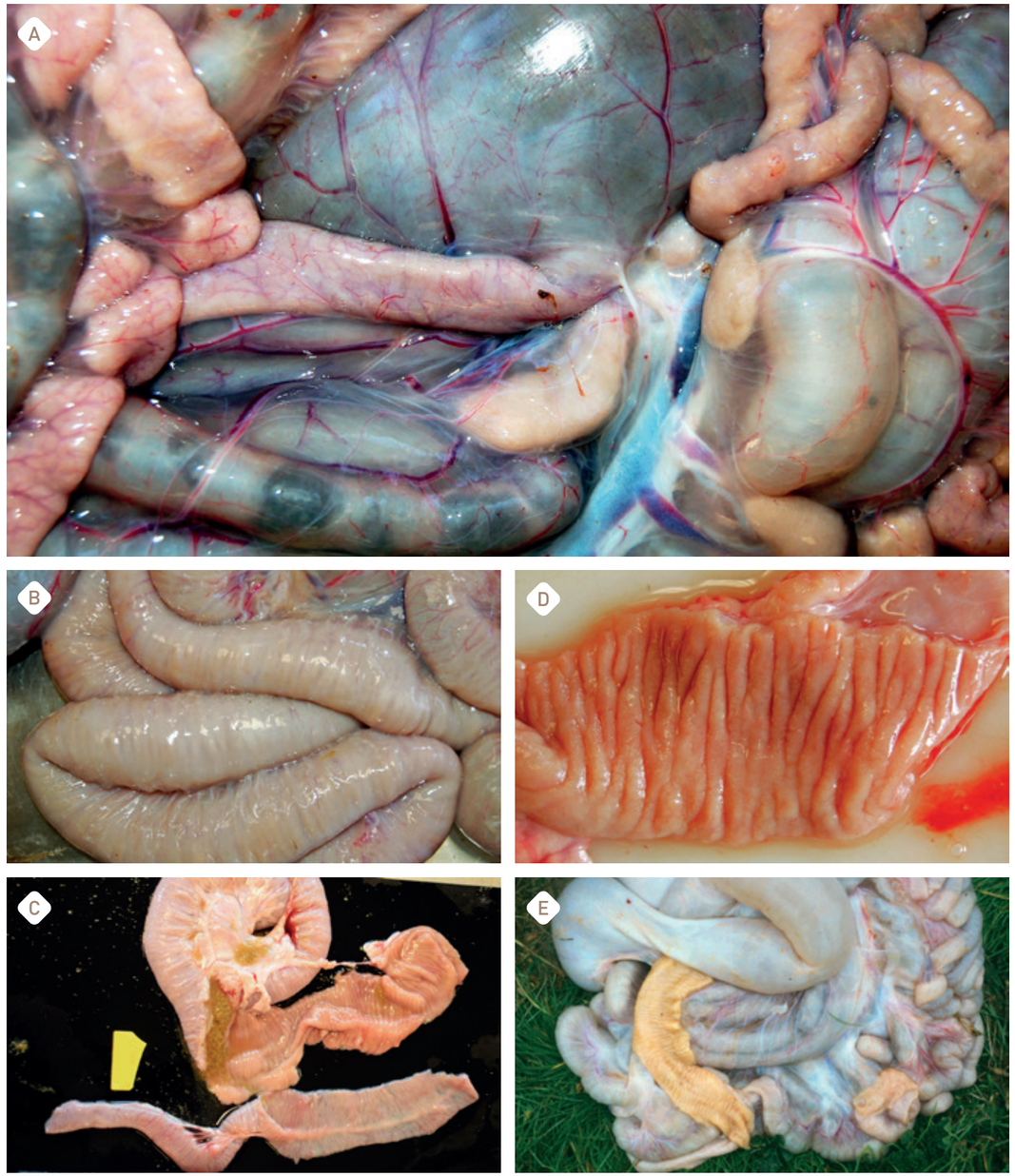
The causative agent of paratuberculosis in sheep and goats is *Mycobacterium avium* subsp. *paratuberculosis*, which causes a wasting syndrome mainly observed in animals of 2 to 4 years of age.



4.251. **Clinical signs.** Johne's disease causes principally an intermittent loose, pasty diarrhoea that leads to a wasting syndrome in sheep and goats (A and B) and signs of dysproteinemia with the formation of oedema in the intermaxillary space (B and C). Infected animals may be partially anorexic and cachectic for 6 to 12 months before they die.



4.252. **Lymphangitis and lymphangiectasia in the intestinal serosa.** One of the most characteristic lesions of Johne's disease is the almost total absence of mesenteric fat and the presence of marked lymphadenitis (A). The lymphatic vessels with lymphangitis and lymphangiectasia in the serosa are visible due to thrombosis and inflammation (B).



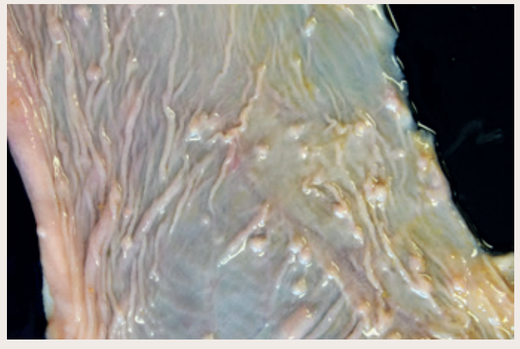
4.253. **Granulomatous enteritis.** Gross postmortem findings include thickened and corrugated intestinal mucosa, especially in the ileocecal portion (A), forming transverse folds visible from the outside (B). The opening of the affected intestine shows a thickened greyish-white mucosa (C and D), except when the so-called "yellow strains" cause the infection, which presents the mucosa coloured in yellow (E).

INTESTINAL PARASITISM





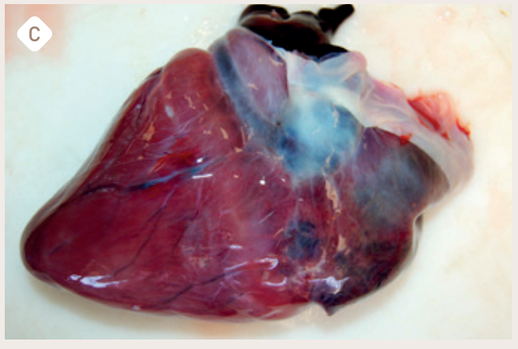
4.254. **Clinical signs.** Animals affected by intense parasitism have a poor general appearance, with wasting and cachexia (A). In severe cases, submandibular oedema may be present (B).



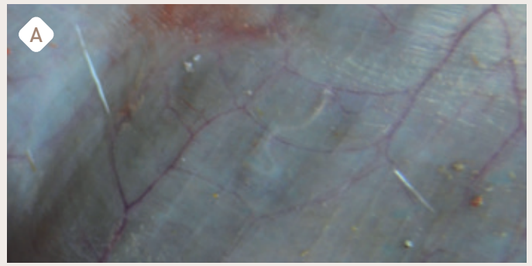
4.255. **Granulomas.** Some species, such as *Oesophagostomum* sp., can create granulomas in the mucosa visible in the serosa.

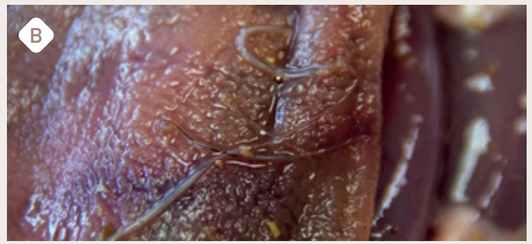


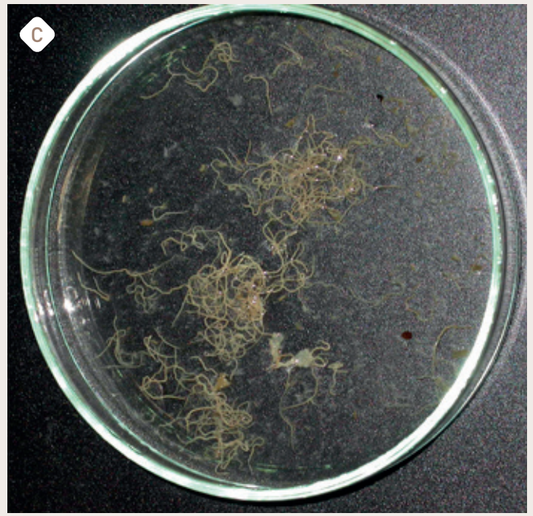




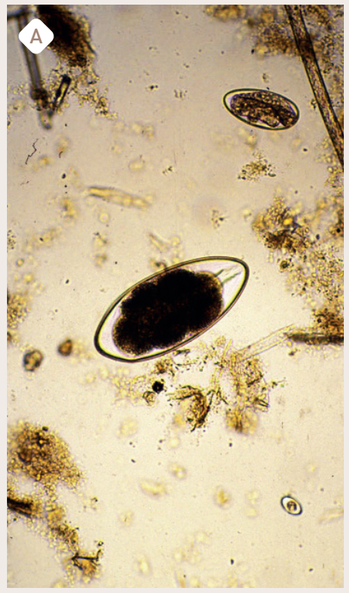
4.256. **Serous atrophy.** The animal presents serous atrophy in the different organs (A-C).



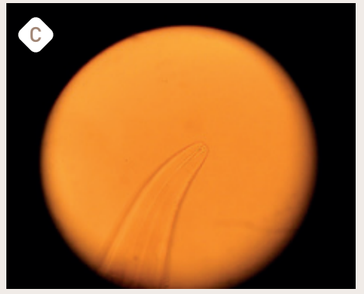


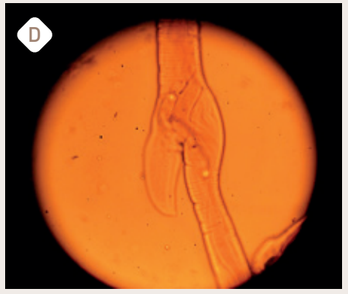


4.257. **Gastrointestinal nematodes.** Many species of nematodes can affect the intestine of small ruminants. The most commonly found are *Nematodirus*, *Trichostrongylus*, *Cooperia*, *Bunostomum*, *Chavertia*, *Oesophagostomum*, *Strongyloides*, *Trichuris*, etc. Some of them can be detected through the serosa (A) or when opening the intestine (B) and can be collected for later identification (C).

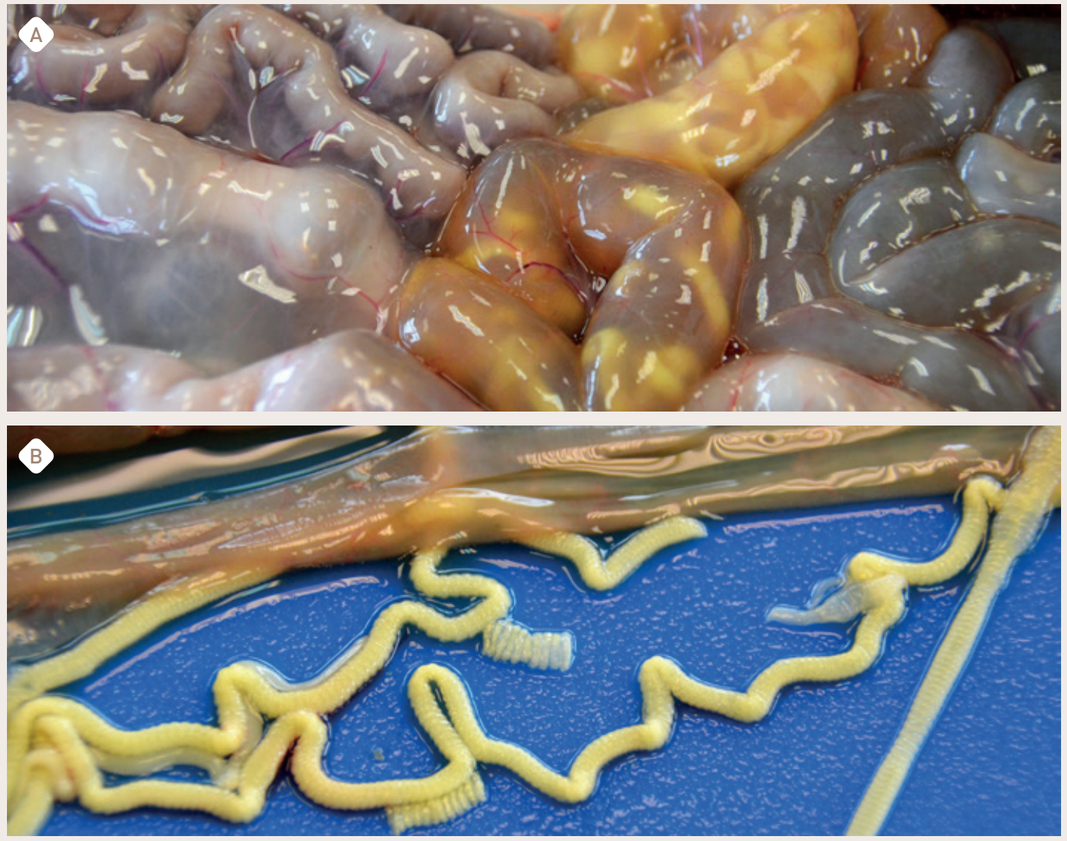




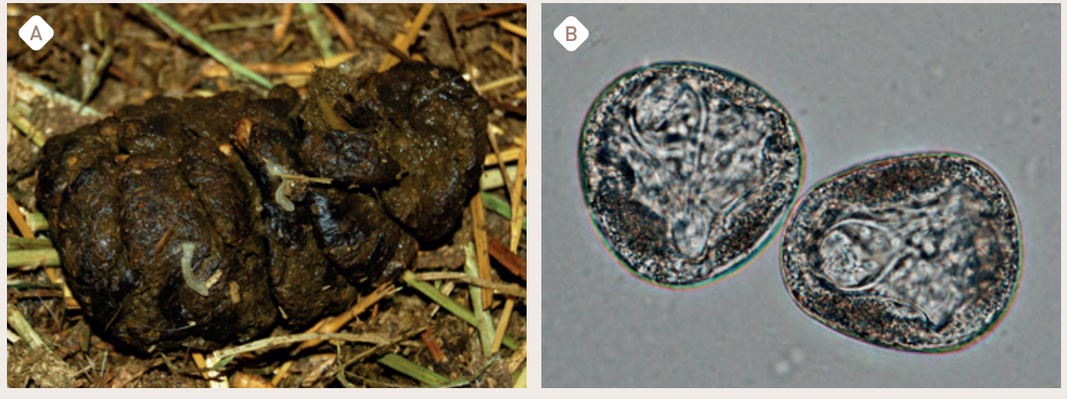




4.258. **Coprology and coproculture.** A faecal analysis let us know the state of parasitisation of the animals and quickly recognise some species such as *Nematodirus* (more prominent and with black blastomeres) and *Strongyloides* (with larvae inside) (A), *Trichuris* (B) and classify the parasites by recognising its shape and measuring its length and width. Observation of the anterior (C) and posterior (D) pole allows their classification in the coproculture.

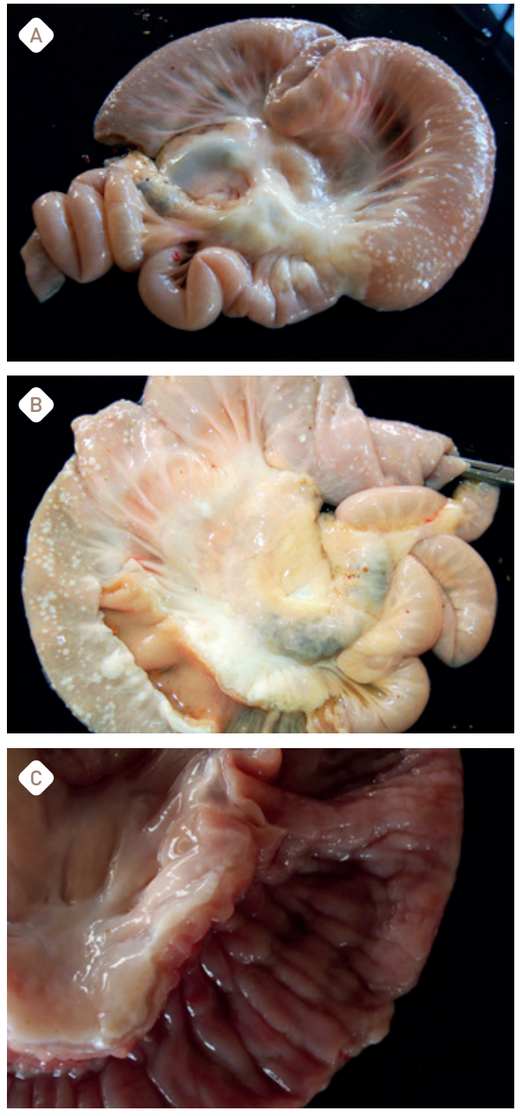


4.259. **Gastrointestinal cestodes.** Adult cestodes can be seen, during necropsy, through the intestinal wall (A). Their length can reach several meters, the most important being *Moniezia* (B), *Thysaniezia*, *Avitelina* and *Stilesia*.



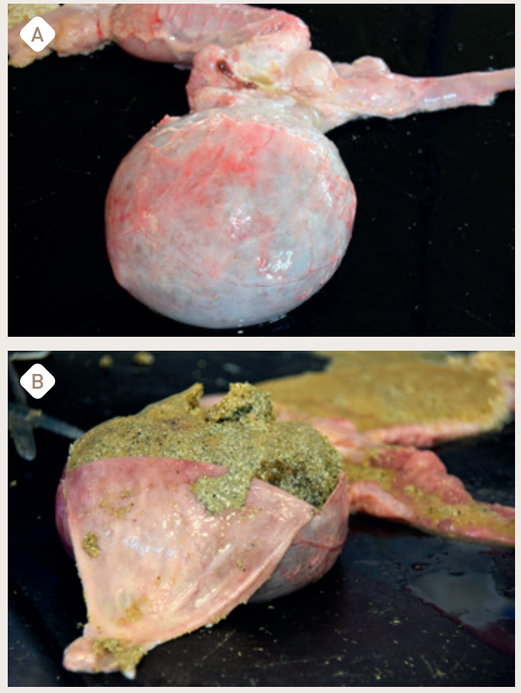
4.260. **Proglottids and eggs.** Tapeworm proglottids can be seen in faeces (A) and eggs on stool analysis (B).

INTESTINAL ADENOCARCINOMA

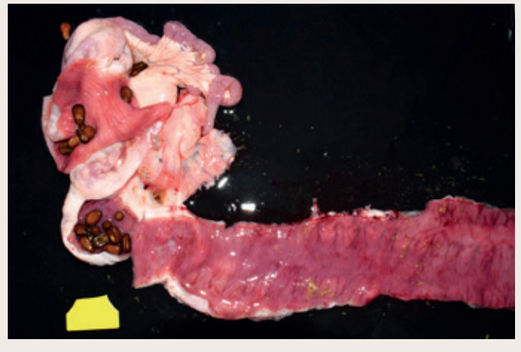


4.261. **Intestinal adenocarcinoma.** It is one of the most frequent tumours of the digestive tract in sheep, with more significant thickening of the intestinal wall than that produced by granulomatous enteritis, with which it could be confused. It causes changes in the intestinal diameter and can almost completely occlude the lumen at the starting point of the lesion (A-C).

OTHER INTESTINAL AFFECTIONS



4.262. **Intestinal diverticulum.** Sporadic diverticula can be found in areas of enteritis, such as this one containing a sandy material (A and B).



4.263. **Intestinal obstructions.** Also, occasionally, scar retractions of the mesentery can be found that generate folds and intestinal twists that hinder and partially obstruct the passage of food. In the images, an intestine with very closed folds and partial torsion made it difficult for food to pass through (corn grains).

FAECES

Faeces reflect what is happening during the digestive transit. Their observation is as interesting as any symptom or injury of the process to be investigated.



4.264. **No pathological aspects.** The normal appearance of the faeces of small ruminants is in small pellets or larger and pasty (dung) and of greenish to black colour when they are fed with grass (A and B). The colours may change depending on the diet, with yellowish tones due to the consumption of cereals (C), orange for consumption of carrots (D) or reddish tones due to the diet with remains of red beet (E).



4.265. **Mucous faeces.** The presence of mucus in the stool is usually related to slow transits or temporary stops in transit. It is also related to some bacterial infections or parasites (A and B).



4.266. **Diarrhoea.** Nutritional diarrhoea in adult animals is usually caused by the high consumption of grains or grazing in lush pastures, especially in spring (A and B). Infections and parasitosis also cause diarrhoea (C). The hindlimbs get dirty with watery stools and dry pellets in chronic cases of diarrhoea (D and E).



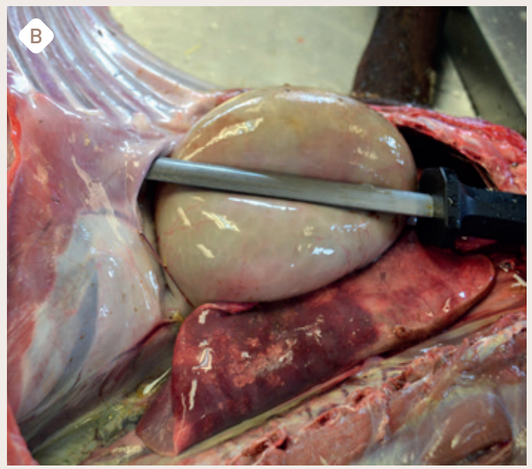
4.267. **Bloody faeces.** The haemorrhagic conditions of the digestive system, abomasitis, enteritis and ulcers usually cause diarrhoeic bloody stools (A), although sometimes it is not easily detected with the naked eye. When the blood comes from the initial portions of the gastrointestinal tract, the stools appear black (B), while if they come from lower sections, they appear more reddish (C).

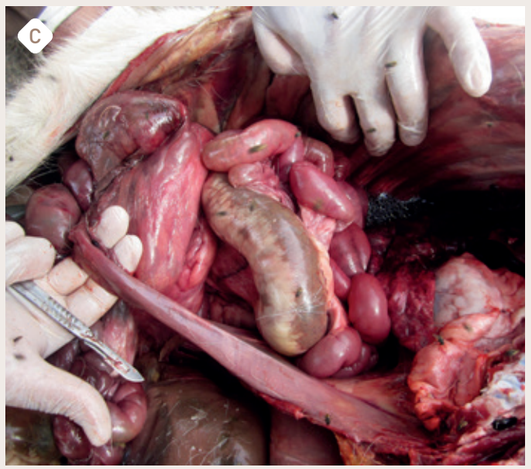


MISCELLANEOUS DISORDERS OF THE DIGESTIVE SYSTEM

DIAPHRAGMATIC HERNIA





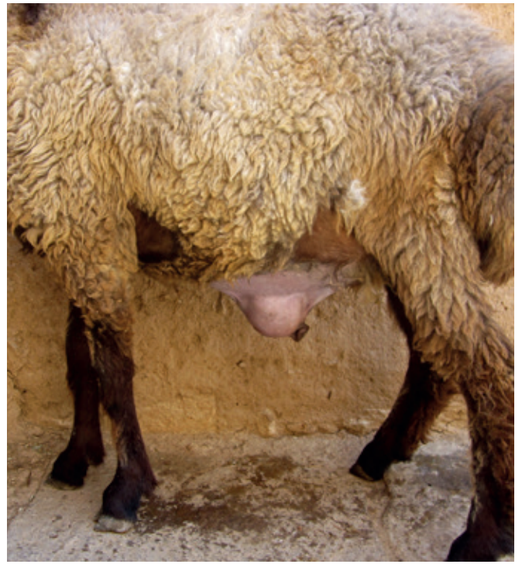


4.268. **Diaphragmatic hernia.** It is the incomplete closure of the diaphragm, leaving communication between the thoracic and abdominal cavities. It usually is the abdominal viscera that penetrate the thorax. Its origin may be congenital or acquired, usually caused by a trauma (A-C).

ABDOMINAL WALL HERNIA



4.269. **Abdominal wall hernia.** In the sheep and goat grazing system, the possibility of traumatic hernias in the abdominal wall is prevalent, especially in flocks that have horned animals.



4.270. **Umbilical hernia.** A particular case is umbilical hernias, generally congenital and that increase in size over time if they are not solved at the beginning.







4.271. **Diagnosis.** In order to confirm the diagnosis of an abdominal hernia, pressure must be applied to see if the mass easily returns to the initial position and disappears (A and B). In addition, we have to look for a hernial ring (C).





4.272. **Detachment of the *rectus abdominis* muscle at the pubis.** It is a condition that happens when there is excess volume and weight in the abdominal cavity. It occurs in animals that consume very large rations and in very prolific breeds. It is associated with trauma or very sudden movements. The abdomen is asymmetrical and is drooping laterally (A and B).

ABDOMINAL HAEMORRHAGES













4.273. **Abdominal haemorrhages.** They are usually of traumatic origin due to rupture of blood vessels (A-C) or abdominal organs (D). They can be caused by perforating ulcers (E) and are favoured and aggravated by the ingestion of anticoagulant (F).

VISCERAL CYSTICERCOSIS

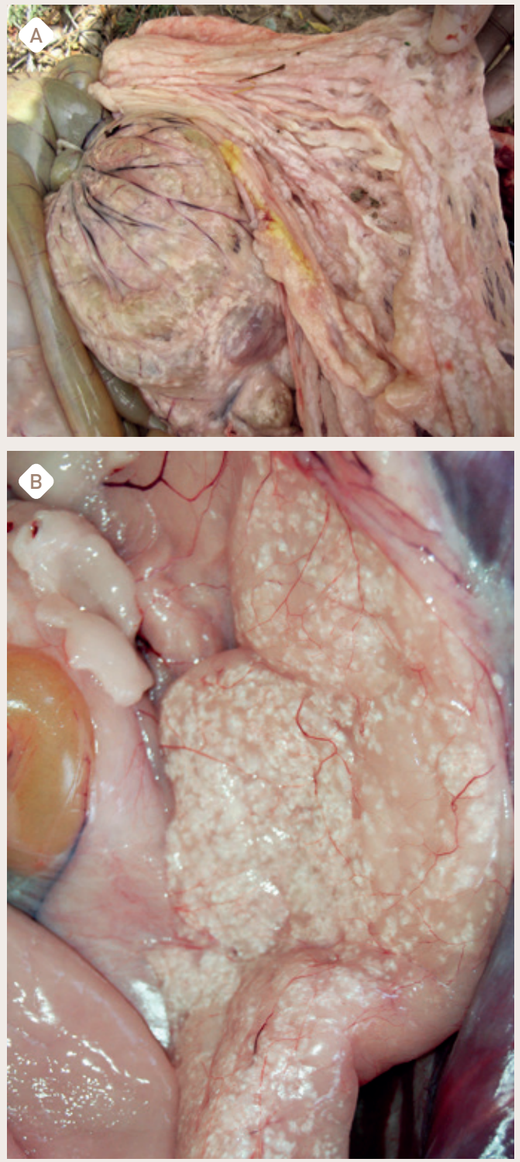


4.274. **Cysticerci vesicles.** As in young animals, adults, especially young adults, can be parasitised by cysticerci, which can be seen in visceras or on the omentum.



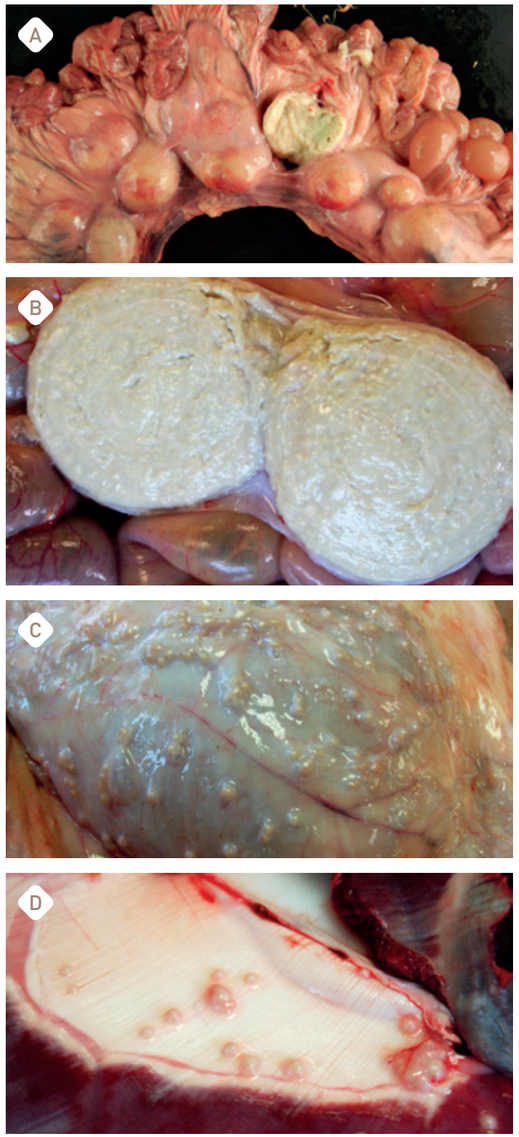
4.275. **Calcified cysticerci.** Some of the cysticerci are calcified in the process of destruction and control of the organism, this being a common finding.

FAT NECROSIS



4.276. **Fat necrosis.** The rapid mobilisation of fat deposits in situations of sudden energy deficit causes large areas of fat necrosis that give the abdominal fat a mottled white appearance (A and B). It is a common finding in pregnancy toxaemia and after strong and fast energy deficits.

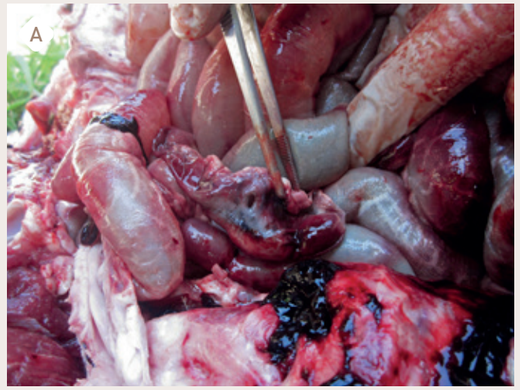
CASEOUS LYMPHADENITIS (CLA) IN THE ABDOMINAL CAVITY



4.277. **CLA in the mesentery.** CLA, in its visceral form, can affect the mesenteric lymph nodes (A and B). The fistulisation of an affected lymph node can cause it to spread, like small pyogranulomas, through the peritoneum and diaphragm (C and D).

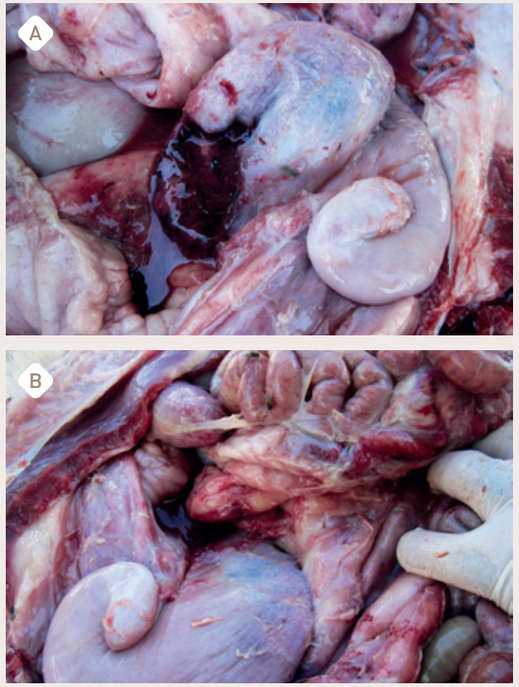
PERITONITIS

Inflammation of the peritoneum can occur as a primary or secondary condition in sheep and goats. The leading causes of peritonitis in small ruminants include digestive perforation or passage of microorganisms through the digestive mucosa, perforation of the reproductive tract after dystocia and infectious agents from the genitourinary tract (this will be widely discussed in its chapter) or iatrogenically through punctures or abdominal surgery. They can affect locally or diffusely.



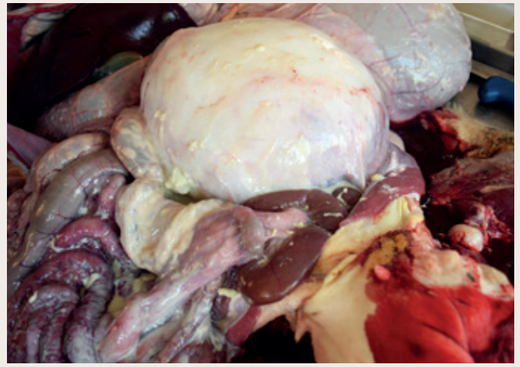


4.278. **Peritonitis due to intestines rupture.** Intense parasitosis and digestive infections can lead to intestinal perforations and peritonitis (A and B).

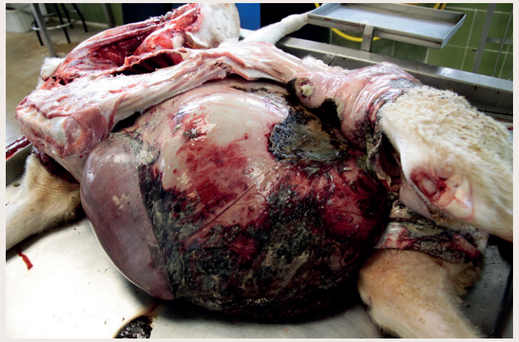




4.279. **Peritonitis of genitourinary origin.** Peritonitis due to reproductive tract perforation after trauma. The uterine wall in goats is fragile and thin, which can be ruptured if dystocia correction is not done gently. The animals usually die from peritonitis a few days after dystocia (A-C).

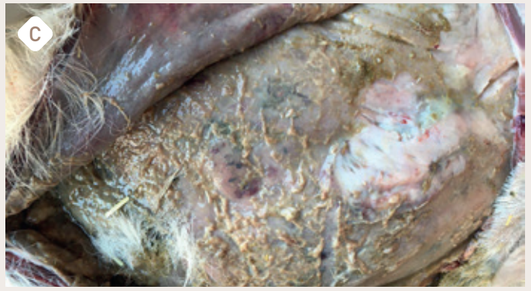


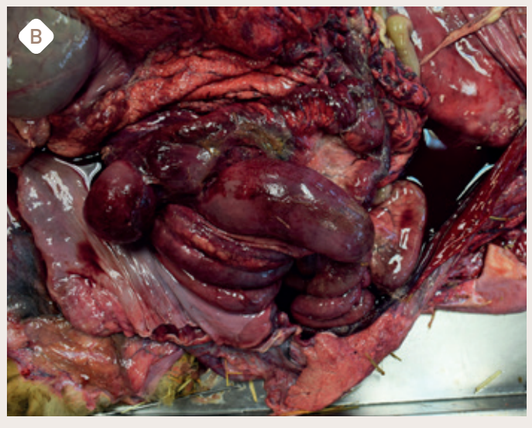
4.280. **Abscesses and peritonitis.** Abscesses in the abdominal cavity can fistulate and cause diffuse peritonitis.

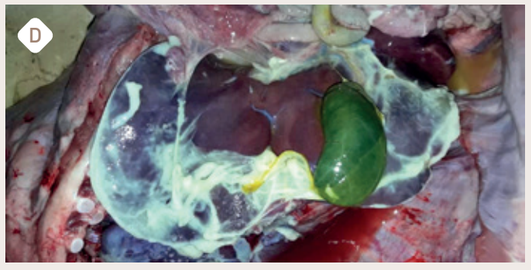


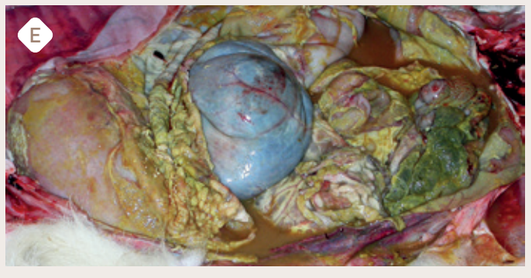
4.281. **Peritonitis of traumatic origin.** Trauma to the abdominal cavity can be the origin of fibrin bleeds and possible bacterial infection.











4.282. **Diffuse peritonitis.** The most severe lesions in acute diffuse peritonitis are usually in the ventral abdomen (A-C), and in less acute cases, the exudate is purulent and forms a thick, cheesy covering over most of the viscera (D and E).

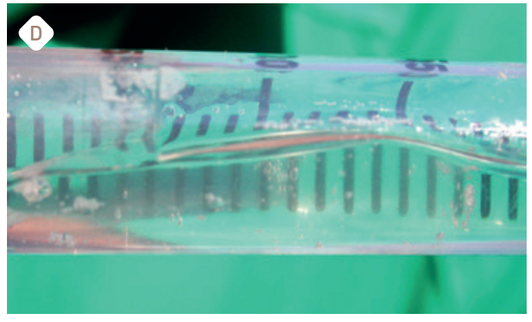
ASCITES













4.283. **Ascites.** Ascites is the accumulation of fluid, rich in proteins, in the abdominal cavity. It is mainly associated with liver disorders (parasitosis, cirrhosis, etc.) and peritonitis. Affected animals have a distended abdomen in the ventral area on both sides (A and B). In mild conditions, the accumulation of liquids is small, and the ascites fluid obtained is clear and coagulates after collecting it (C and D). In severe cases, especially in peritonitis, a large amount of fluid can be accumulated and may be contaminated by bacteria (E and F).

RECTAL PROLAPSE









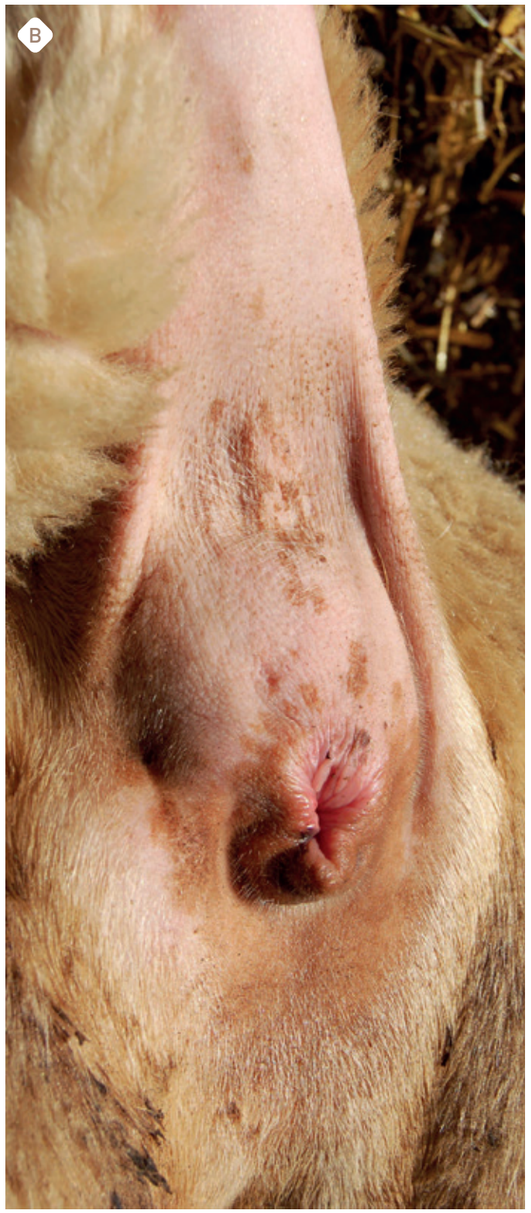
4.284. **Rectal prolapse.** Prolapse of the rectum is an occasional occurrence caused by intestinal inflammatory disorders, increased pressure in the abdominal viscera, and neglected tail docking. Mild cases are usually temporary and can be resolved on their own (A and B). Acute cases can occur and cause animal death (C). The prolapsed area of the rectum can be lacerated, and intestinal loops protrude through it (D).



4.285. **Postmortem rectal prolapse.** Due to abdominal pressures during the putrefaction of dead animals with meteorism, the rectum may prolapse after the death of the animal.

RECTUM VAGINAL FISTULA





4.286. **Congenital rectum vaginal fistula.** The rectum-vaginal fistula is a communication between the vagina and the rectum (A). It can be caused during obstetric manoeuvres, more frequent in cattle, and it can also be congenital, associated with anal atresia (B).

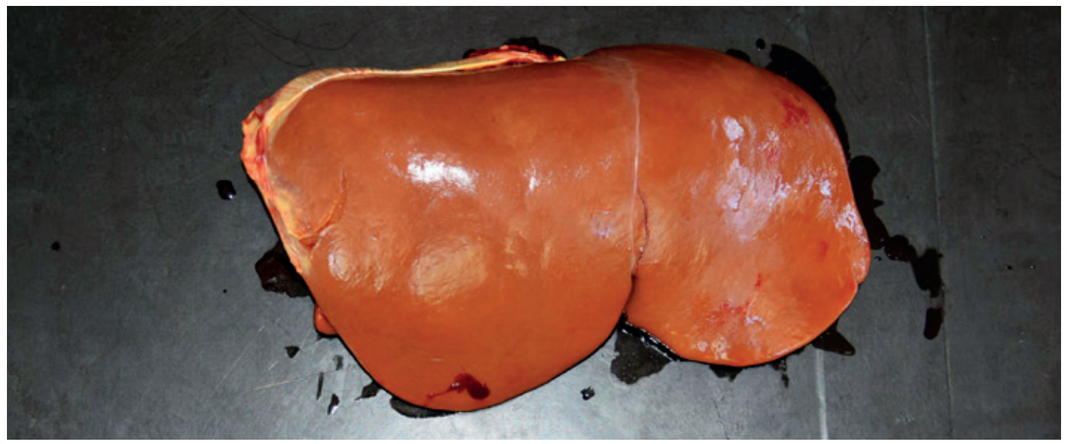
LIVER DISORDERS

HEPATIC MELANOSIS



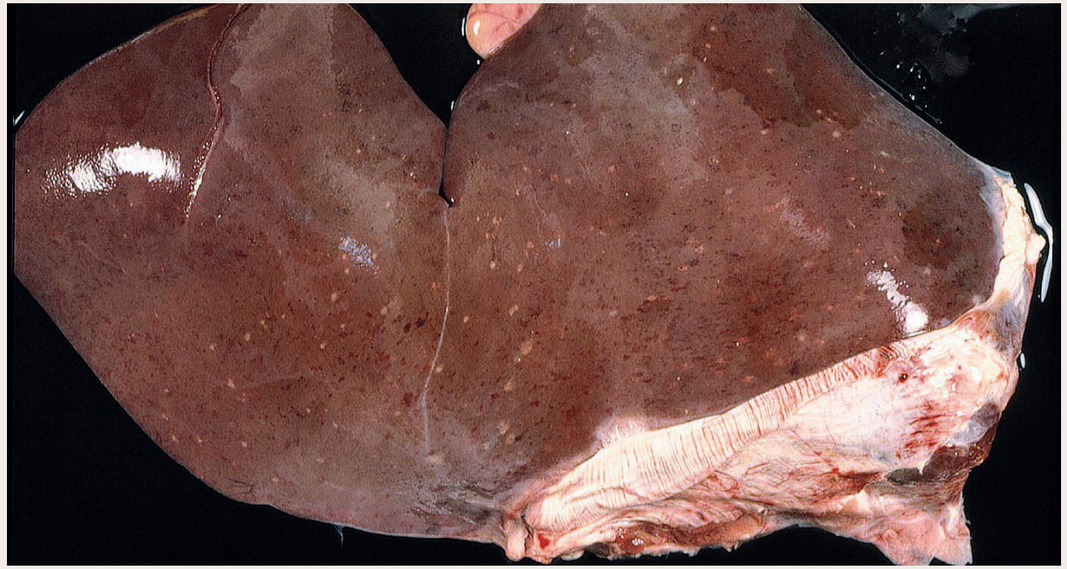
4.287. **Black liver or melanosis.** Dark brown to black pigmentation of the liver often occurs in sheep and goats, usually not associated with illness. This condition is due to the deposition of the pigment lipofuscin at various stages of oxidation.

HEPATIC JAUNDICE

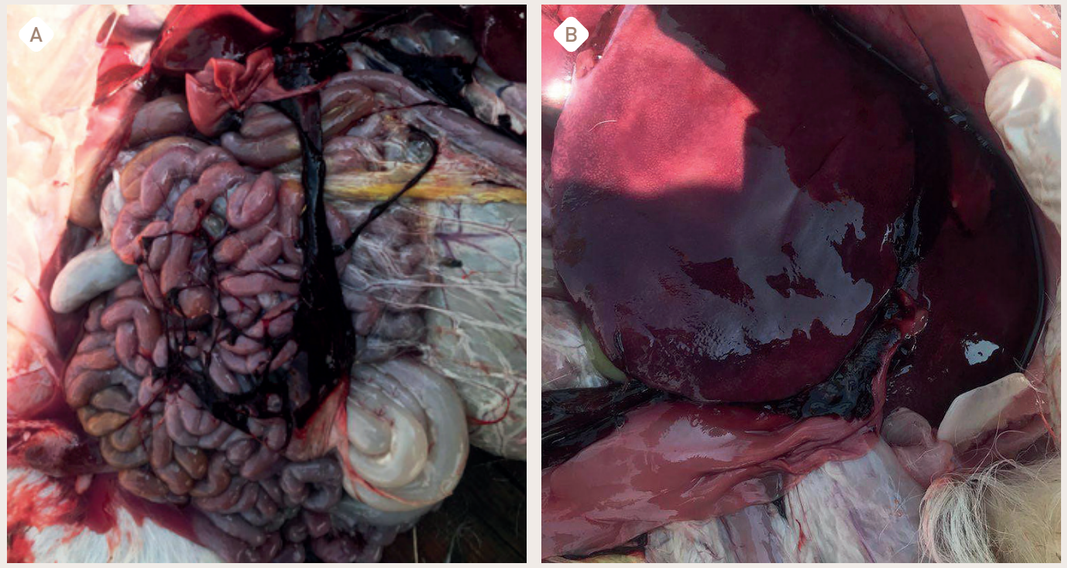


4.288. **Hepatic jaundice.** The accumulation of pigments derived from the massive rupture of erythrocytes can cause jaundice, giving a yellow/orange colour to different organs. The causes can be varied, from very large and rapid water consumption in thirsty animals, blood parasites or toxi-infections such as *Clostridium perfringens* type A (alpha-toxin).

HEPATIC HAEMORRHAGES

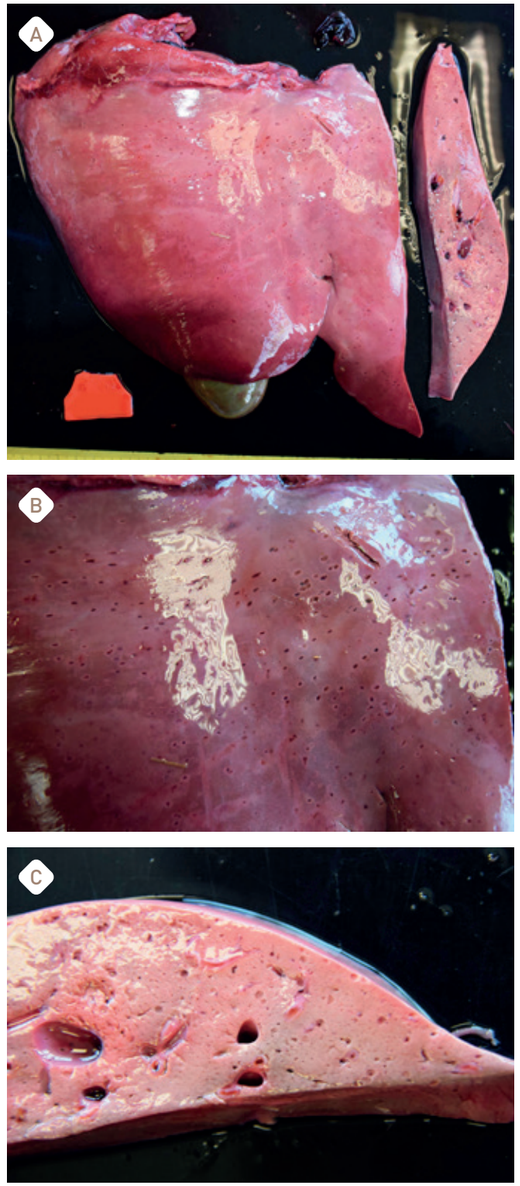


4.289. **Petechiae.** Various septicaemic processes can cause the appearance of petechiae. In the image, septicaemia due to *Streptococcus* spp.



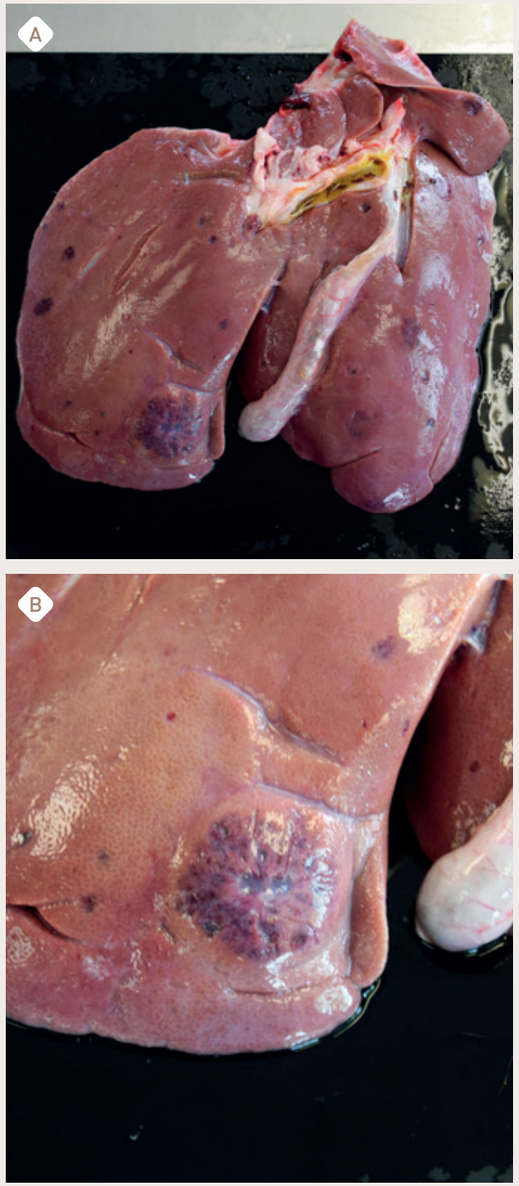
4.290. **Traumatic haemorrhages.** Trauma to the abdominal cavity can cause a ruptured liver with heavy bleeding (A and B).

HEPATIC TELANGIECTASIA



4.291. **Hepatic telangiectasia.** The aspect of the surface (A and B) and the section (C) of a liver with dilated blood vessels. Telangiectasia is a rare process and could be associated with degenerative disorders.

HEPATIC ANEURYSMS



4.292. **Hepatic aneurysms.** Aneurysms of different sizes, visible on the liver surface. Its aetiology could not be diagnosed (A and B).

HEPATOCELLULAR CARCINOMA



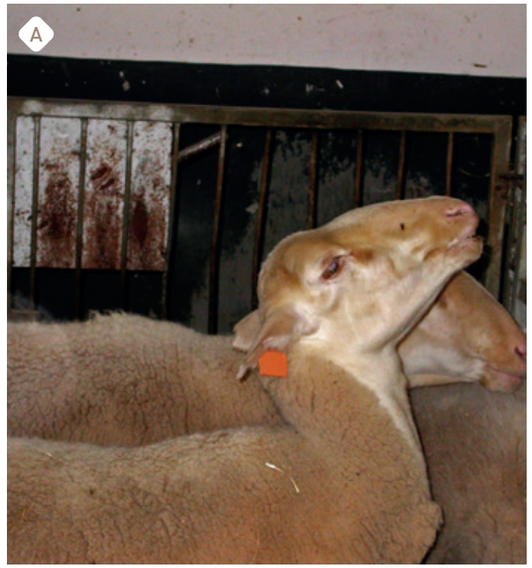
4.293. **Hepatocellular carcinoma.** Liver with nodules making prominence on the surface (A). The carcinoma penetrates the parenchyma (B), and the section shows the total loss of the liver's normal structure, with large white patches of tumoural tissue (C).

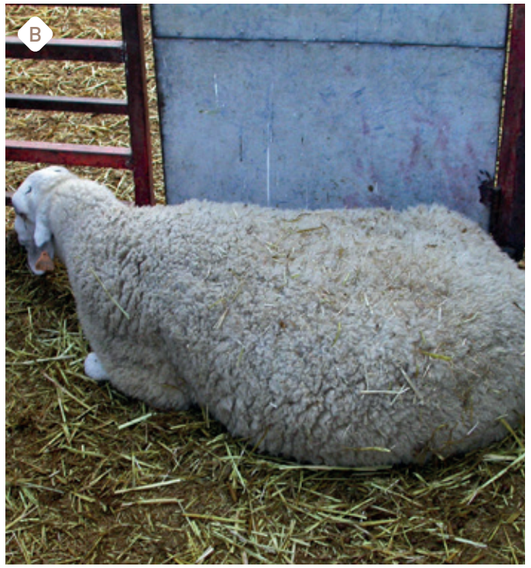
LEUKOSIS



4.294. **Leukosis in the liver.** Numerous tumour foci, with the appearance of poorly defined white spots, can be seen both on the surface of the liver and in the liver parenchyma in the section (A and B).

FATTY NECROSIS AND PREGNANCY TOXAEMIA





4.295. **Pregnancy toxaemia.** It is a metabolic disease with high levels of ketone bodies in the blood that cause nervous symptoms of excitement (A) and subsequent depression with the recumbency of the animal (B).



4.296. **Oedema of ventral areas.** Fat animals with multiple gestations may suffer venous return problems and oedema in the lower parts of the forelimbs and hindlimbs. This oedema starts in the hindlimbs, and in acute cases, the forelimbs are also affected.



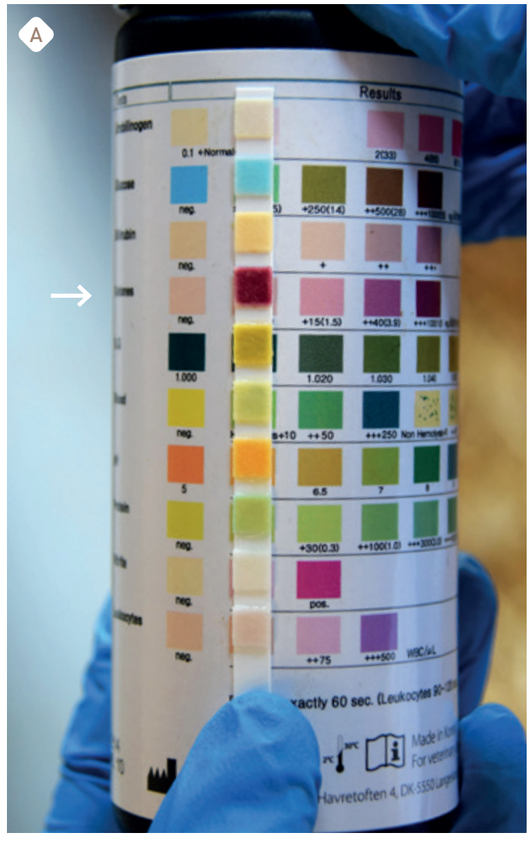
4.297. **Pregnancy toxaemia.** At necropsy, a carcass with abundant mesenteric fat deposit dotted with small foci of fat necrosis is shown.





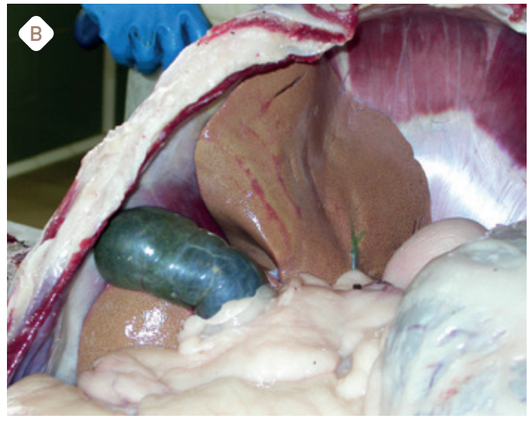


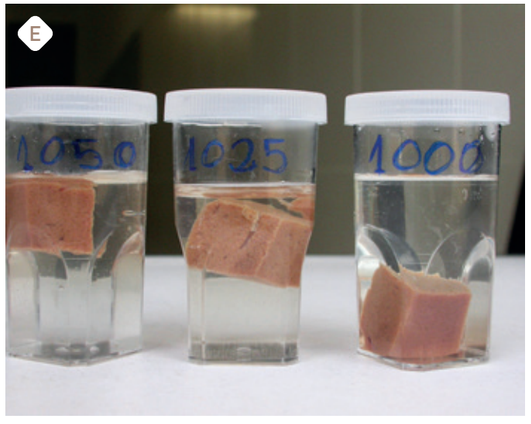
4.298. **Multiple gestations.** Pregnancy toxaemia is usually associated with multiple gestations (A and B), leaving a reduced space for food ingestion. If the sheep finally gives birth, the animal remains in poor general and mental condition, making it difficult to recover (C).







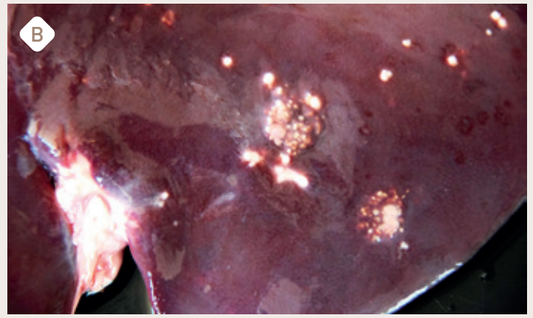




4.299. **Hepatic fatty degeneration.** During the disease, fatty deposits are released abruptly, triggering an increase in ketone bodies, easily detected in urine (A) and rapid fat storage in hepatocytes (fatty degeneration) (B-D). The floating test at different densities allows us to confirm the process quickly (E).

FOCAL HEPATITIS

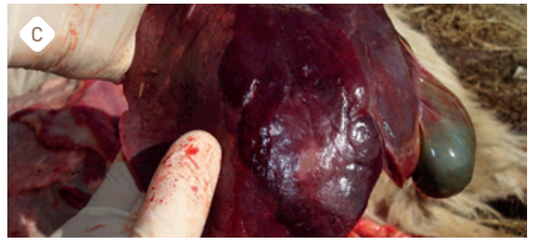


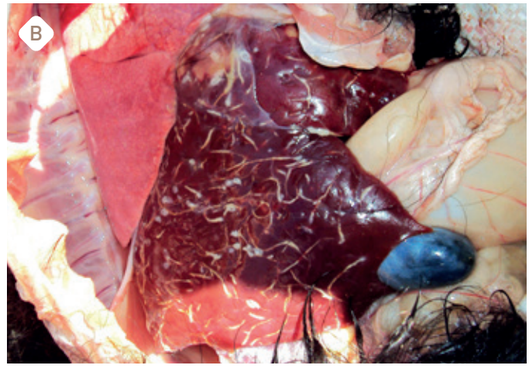


4.300. **Focal hepatitis.** Liver with multiple disseminated foci of hepatitis. This lesion is often associated with septicaemic bacterial dissemination from hepatic abscesses caused by omphalitis with a central focus on the hepatic notch (A and B).

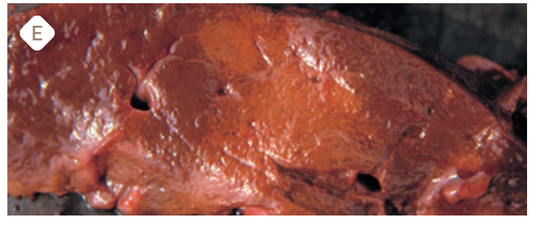
NECROTIC HEPATITIS











4.301. **Infectious necrotic hepatitis (Black disease).** It is acute toxaemia of sheep and goats caused by *Clostridium novyi* type B. Toxins are produced in damaged liver tissue under local anaerobic conditions created by parasites (A and B). The liver is swollen and exhibits areas of hyperaemia and haemorrhages with areas of necrosis that go deep into the parenchyma (C-E).

CIRRHOSIS





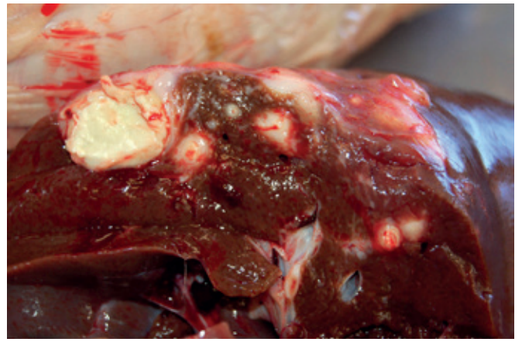
4.302. **Cirrhosis.** Livers with cirrhosis have a rough surface, firm consistency, and a withdrawn appearance (A and B). Cirrhosis is usually associated with toxins, nutritional disorders, etc.



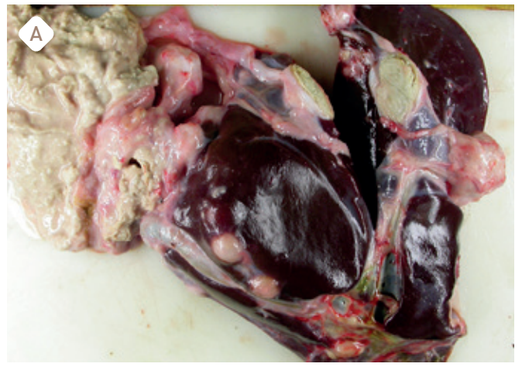


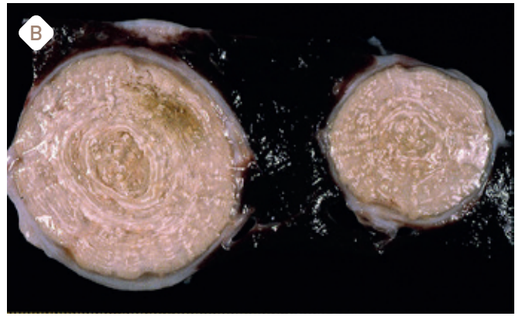
4.303. **Pithomycotoxicosis.** Sporidesmin, a *Pithomyces chartarum* toxin, causes cirrhosis with atrophy of some liver lobes and hypertrophy of others. The appearance of these livers is rounder and much thicker (A and B).

HEPATIC ABSCESSES AND CASEOUS LYMPHADENITIS



4.304. **Hepatic abscess.** The liver receives many micro-organisms in septicaemic processes due to its abundant vascularisation. It is common for some to settle in the liver and create abscesses.

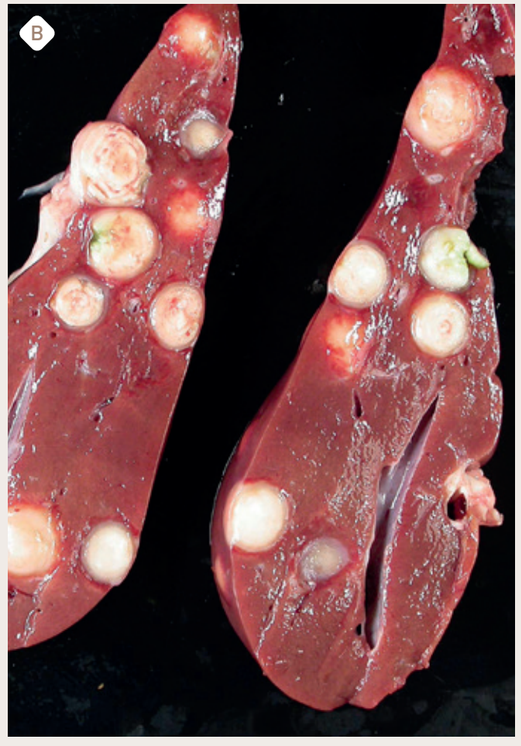




4.305. **Hepatic caseous lymphadenitis.** A particular case is CLA or pseudotuberculosis. In this case, the pyogranulomas tend to have a typical "onion layer" appearance on section (A and B).

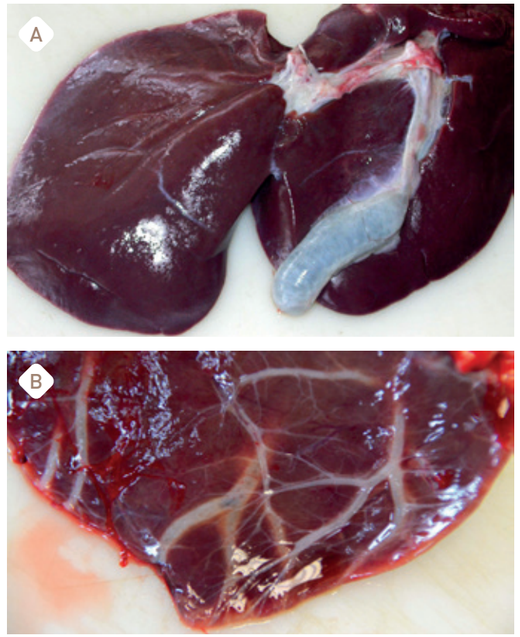
TUBERCULOSIS



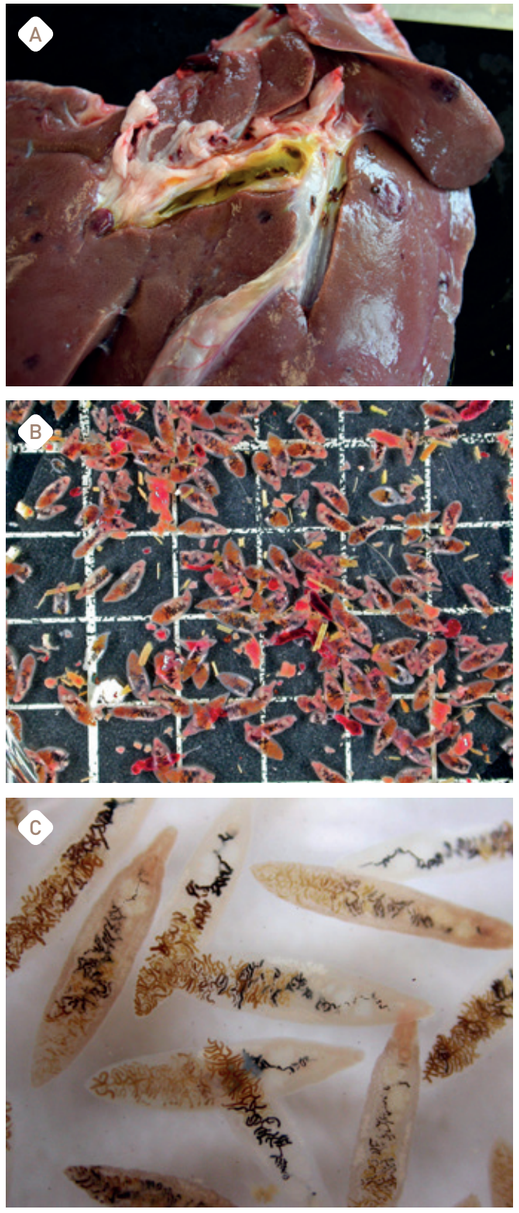


4.306. **Granulomas.** Tuberculosis can affect sheep but is much more prevalent in goats. The images show an affected liver of goat with many granulomas (A), which can be seen in detail on section (B).

DICROCOELIOSIS

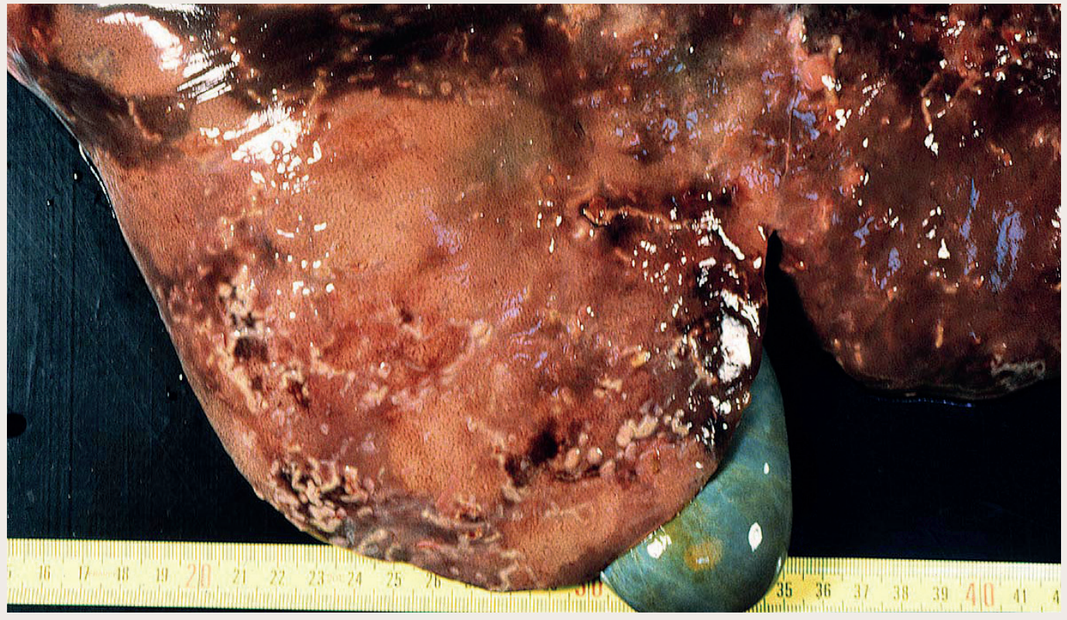


4.307. ***Dicrocoelium dendriticum.*** It is a liver trematode, which infects grazing animals when they swallow ants containing metacercariae. Flukes can cause parenchyma fibrosis and proliferation and thickening of smaller bile ducts (A-D).

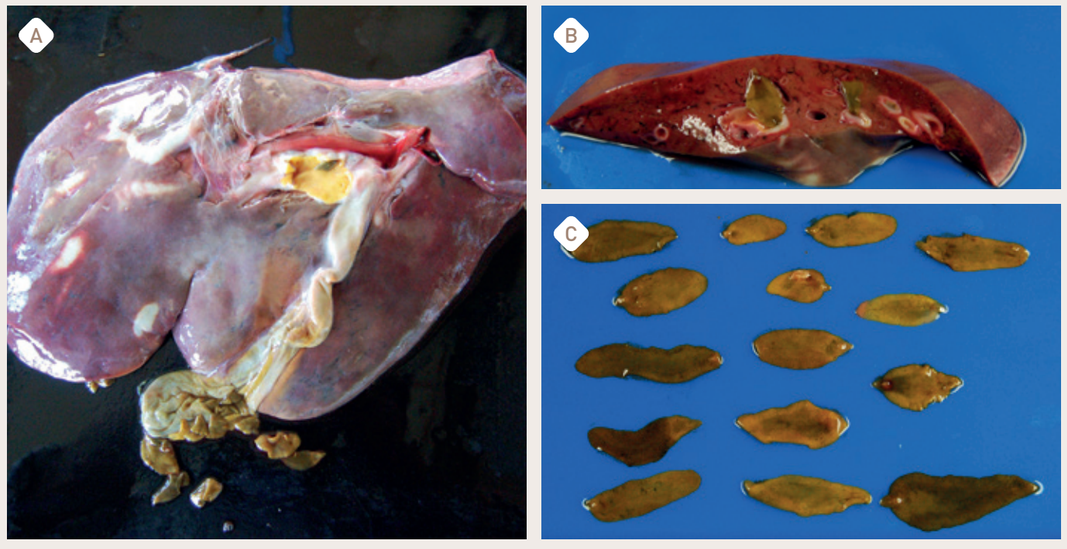


4.308. ***Dicrocoelium dendriticum.*** Adult parasites are visible in bile ducts and gall bladder and can be collected for identification (A-C).

FASCIOLOSIS



4.309. **Acute fasciolosis.** It is a parasitic disease of the liver caused by the migration and penetration into the liver, through Glisson's capsule, of the immature forms of *Fasciola hepatica* or *gigantica*.



4.310. **Cholangitis and cholangiectasia.** The flukes produce cholangitis and cholangiectasia, and they are often demonstrated by slicing the gall bladder liver thinly (A-C).

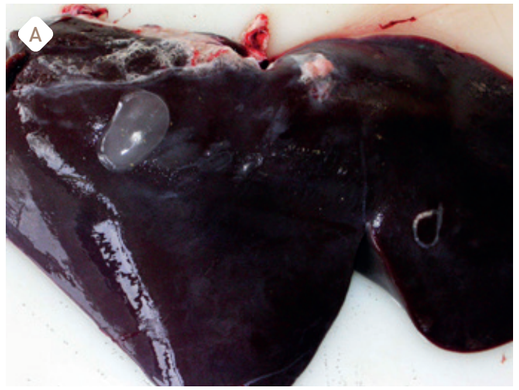




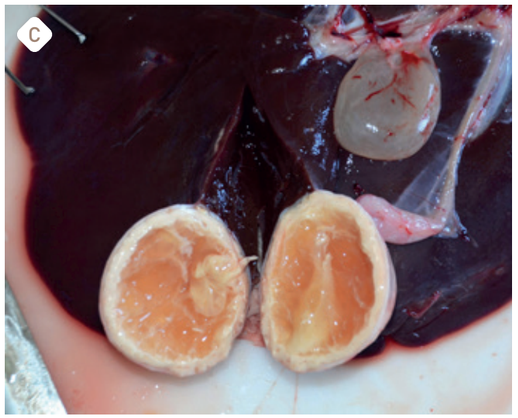


4.311. **Chronic fasciolosis.** Clinical signs are losing weight, bottle jaw (submandibular oedema) and pallor of the mucous membranes (A-C).

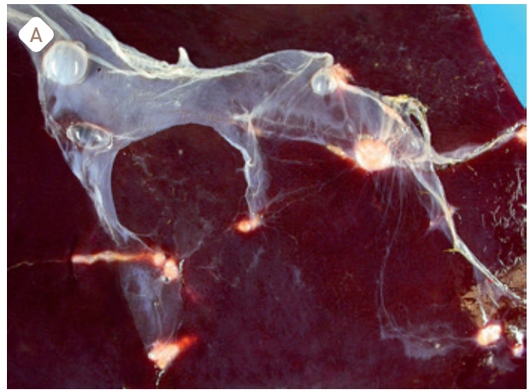
CYSTICERCOSIS

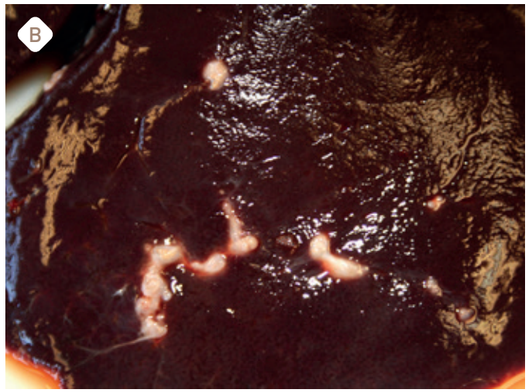




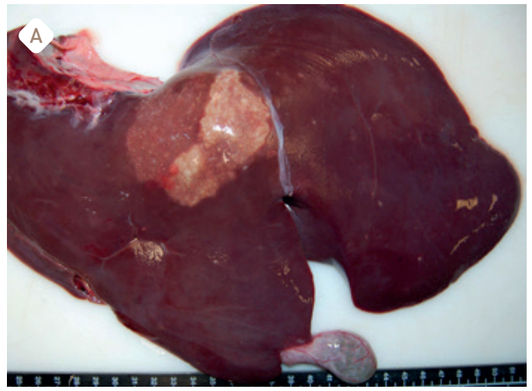


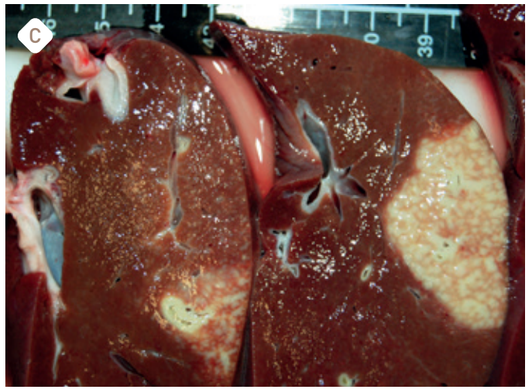
4.312. **Cysticercosis.** Once through the liver, cysticerci can be preserved on Glisson's capsule (A). Sometimes the organism tries to encapsulate these parasites (B and C).

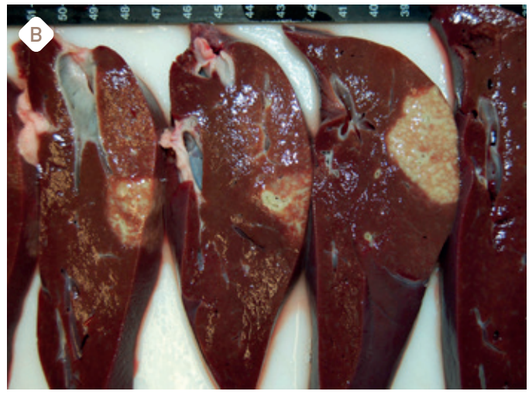


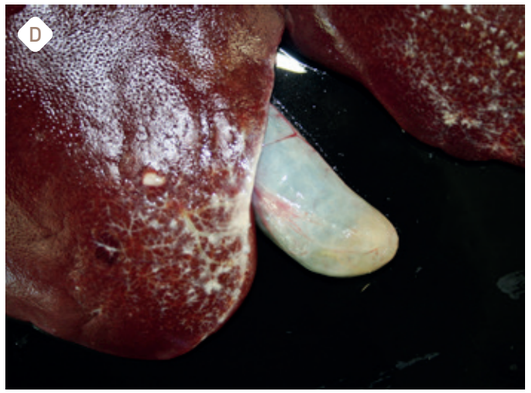


4.313. **Cysticercosis.** Usually, in the same liver, we can find cysticerci on the surface, other calcified, and inflammatory reactions in the resolution phase, following the paths through which the parasite moved (A and B).







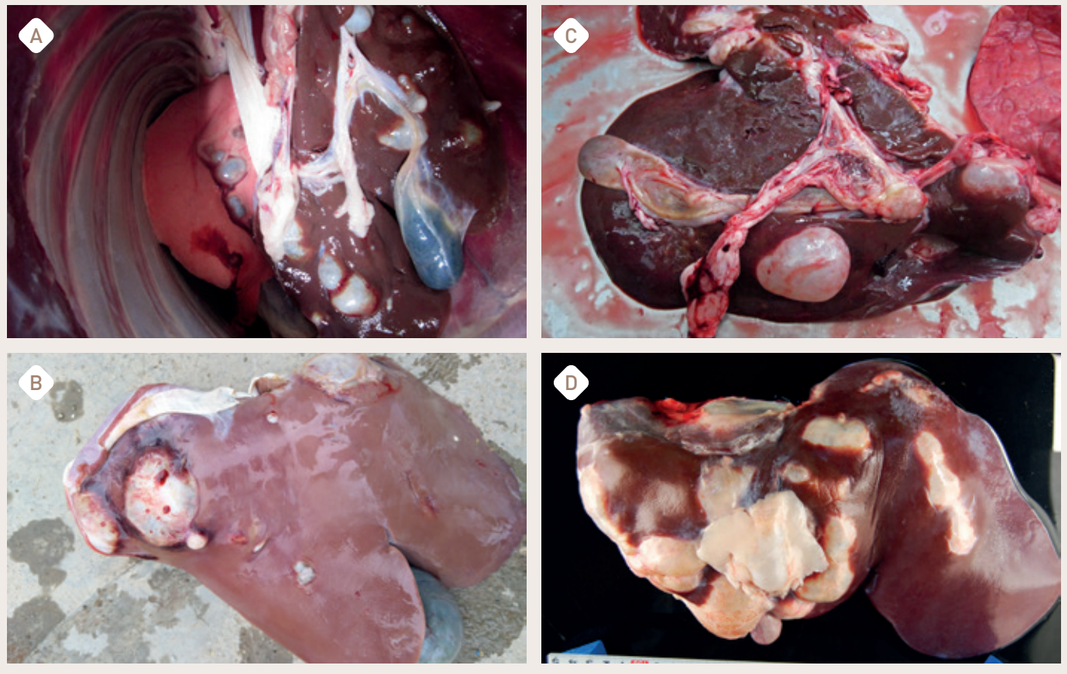


4.314. **Other injuries.** In some cases, focal perihepatitis reactions can be seen around a gall bladder, extending and deepening into the liver parenchyma and sometimes appearing unrelated to the gall bladder (A-D). These infiltrates are composed mainly of eosinophils.

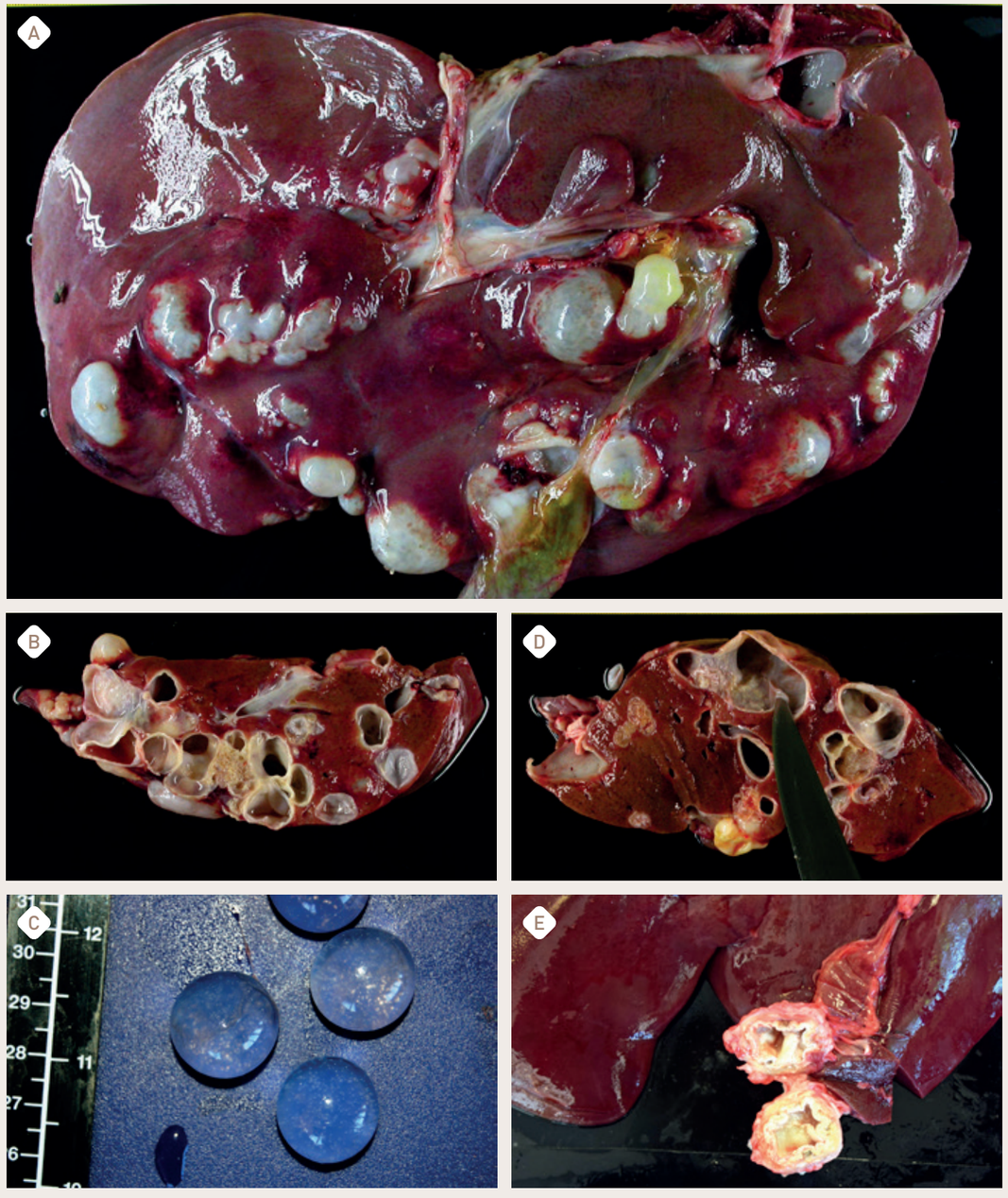
HYDATIDOSIS



4.315. **Transmission.** Dogs are infected by eating the cysts in uncooked meat and offal of infected animals.

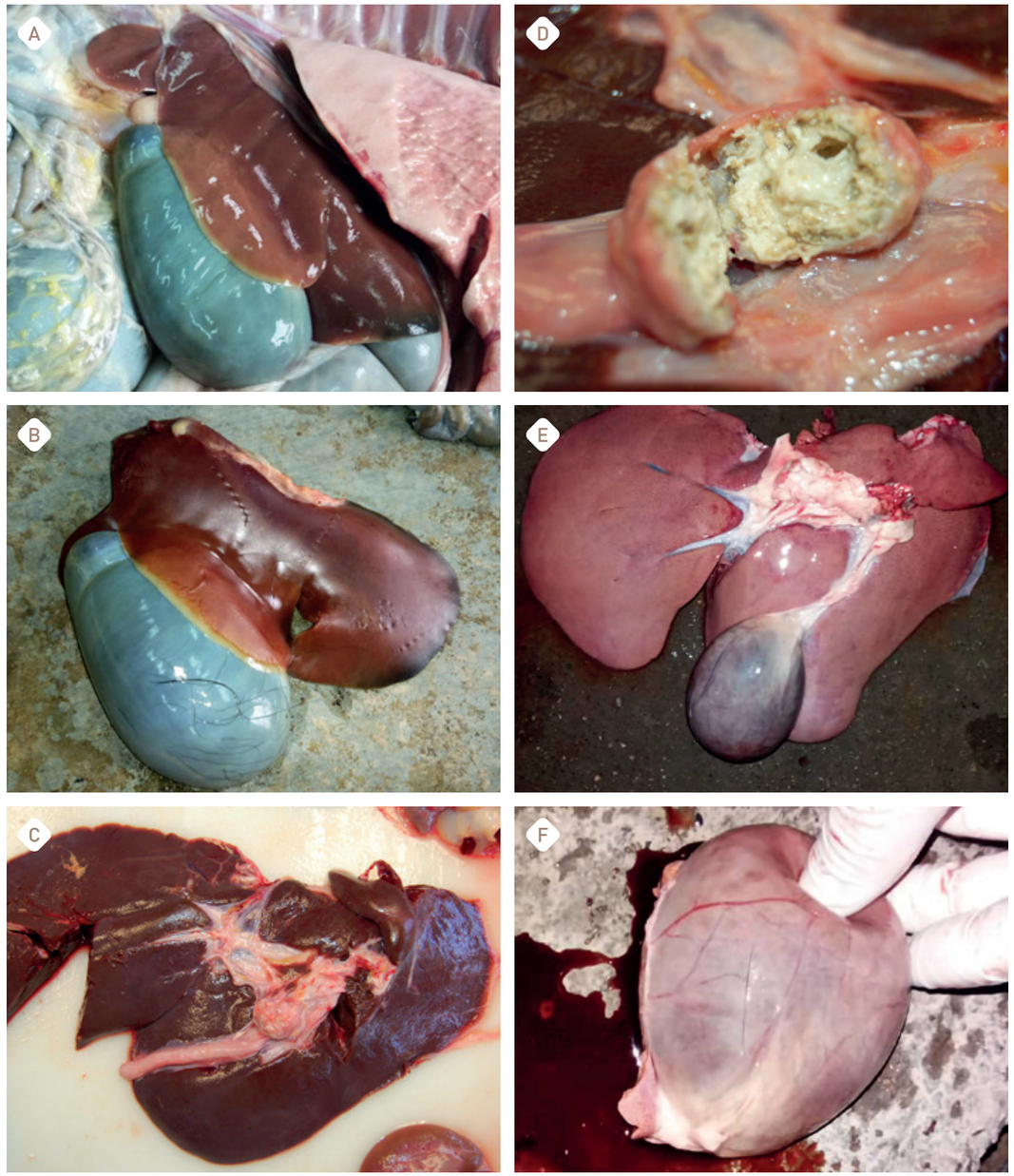


4.316. **Hydatid cysts.** Hydatid cysts are mainly caused by *Echinococcus granulosus* tapeworm, although similar cystic diseases can also be caused by *E. multilocularis*, *E. oligarthrus*, and *E. vogeli*. The same animal can have hydatid cysts in different organs (A) and be found in various numbers and stages of evolution (B-D).

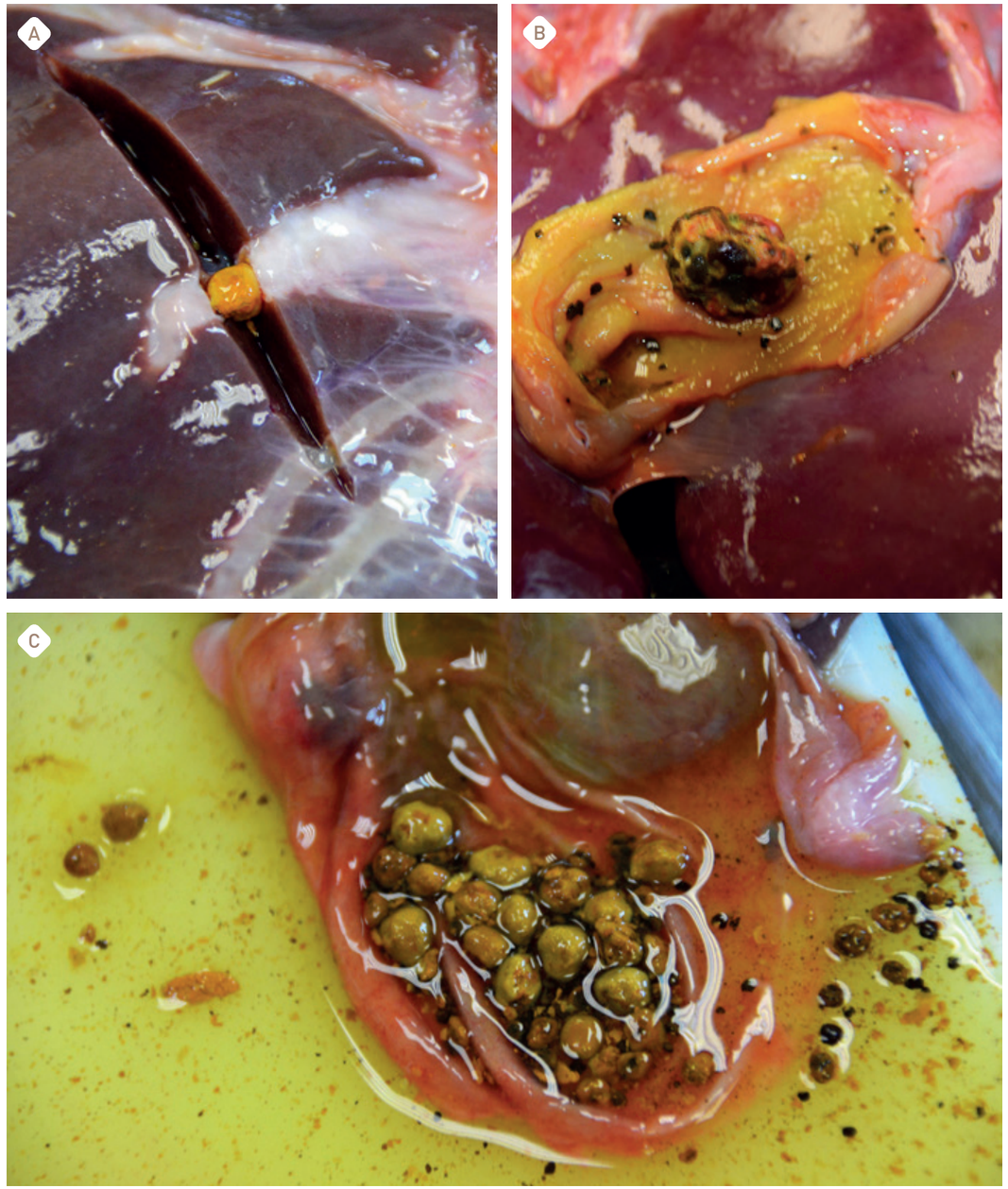


4.317. **Hydatid cyst.** The liver section with hydatid cysts allows us to observe uni- or multilocular cysts in different stages of development (A and B). We can obtain some cysts (C) and easily differentiate the germ layer from the endocyst (D). Others may be calcified (E).

BILIARY OBSTRUCTION



4.318. **Gall bladder obstruction.** Various processes can obstruct the gall bladder and produce bile retention (A-D). In some cases, haemorrhagic cholecystitis is found (E and F).

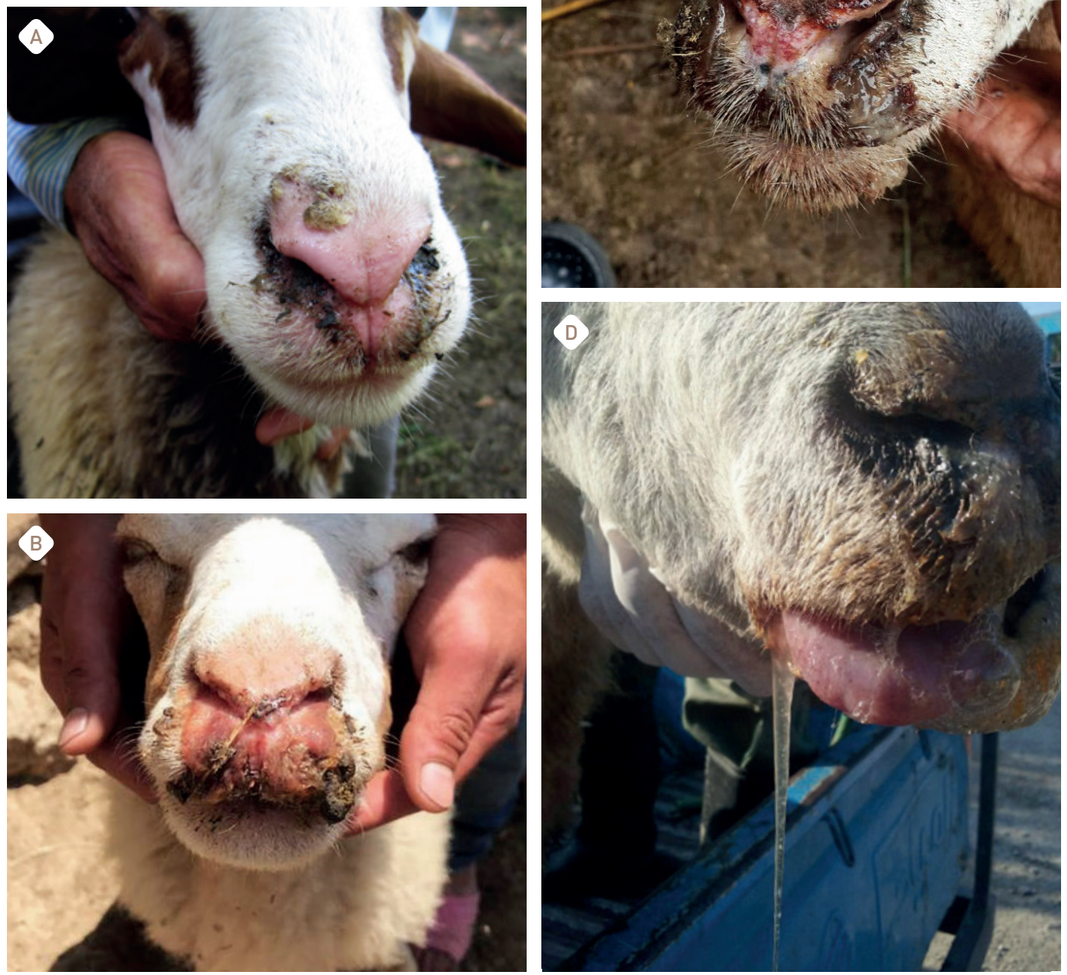


4.319. **Gallstones.** When opening the bile ducts, it is possible, in certain cases, to find abundant gallstones (A and B). Residues or gallstones can also be found in the gall bladder (C), sometimes associated with the presence of *Dicrocoelium dendriticum*.

INFECTIOUS DISEASES

BLUETONGUE (BT)

Bluetongue is an infectious and noncontagious arthropod-borne disease that primarily affects sheep and goats, and it is caused by an Orbivirus. The disease occurs in epidemic areas when climatic conditions allow the expansion of vector occurrence or when naïve animals are introduced into an endemic area.



4.319. **Clinical signs.** After an incubation period of less than a week, a severe febrile reaction with a maximum temperature of 41 °C is usually seen. The fever continues for 5 or 6 days, during which time nasal discharge and salivation become evident, accompanied by reddening of the buccal and nasal mucosae. The nasal discharge in Bluetongue is mucopurulent and often blood-stained, indicating severe inflammation of the nasal passages, and the saliva is typically frothy.



4.320. **Clinical signs.** Swelling and oedema of the lips, face, gums, dental pad, and tongue usually occur in sheep and goats affected by BT virus (A-E).

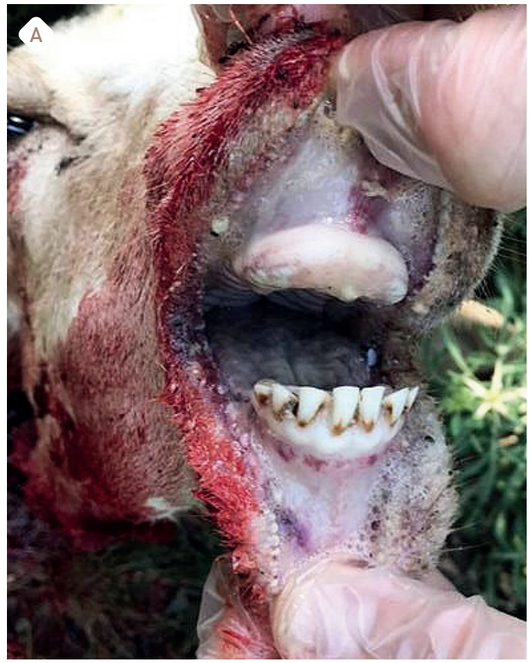



4.321. **Lesions.** When an excoriation of the buccal mucosa occurs, the saliva becomes blood-stained (3A), and the mouth has an offensive odour (3B). *Picture A courtesy of Dr. S. Rouhani.*






4.322. **Lesions.** Lenticular necrotic ulcers are developed, particularly on the lateral aspects of the tongue, which may be swollen and purple in colour (A and B). Hyperaemia and ulceration are also common at the commissures of the lips, on the buccal papillae (C and D). *Pictures A and B courtesy of Dr. M. Sarafraz.*




4.323. **Postmortem findings.** The oral mucosa is usually cyanotic or haemorrhagic, with erosions and ulcers commonly affecting the tongue and dental areas (A and B), sometimes extending to the rumen (C).




4.324. **Postmortem findings.** A very characteristic lesion of bluetongue is a haemorrhage at the base of the pulmonary artery.

FOOT AND MOUTH DISEASE (FMD)

Foot and mouth disease is a viral and highly contagious disease of sheep and goat, caused by an *Aphthovirus* of the *Picornaviridae* family. Although the disease has low mortality in adult animals, it can cause high mortality (up to 50%) in lambs and kids.





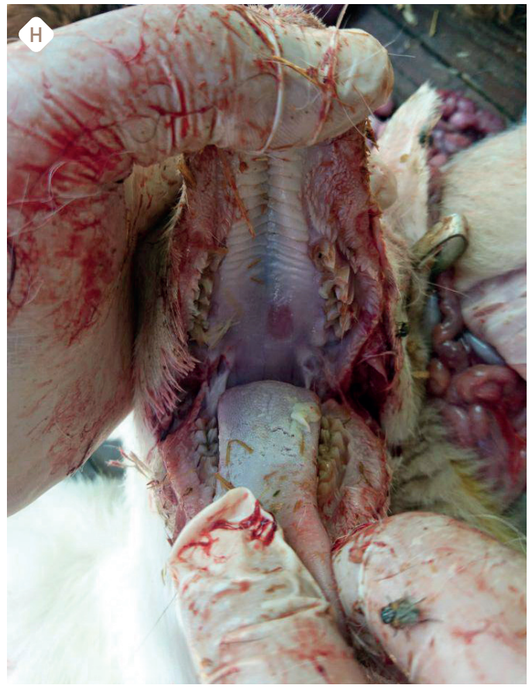












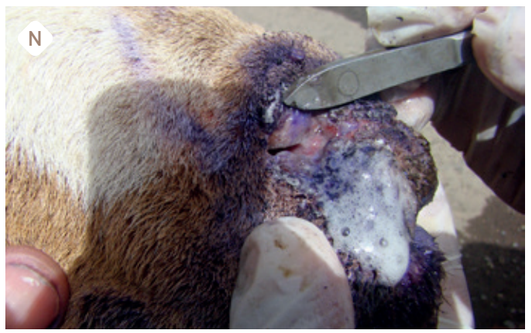












4.325. **Erosions in the mouth.** Since superficial epithelium in sheep and goat is very thin and easily ruptured, it is unusual to see fluid-filled vesicles in the mouths of these species. Erosions may be seen on the lips, gums (A-C), dental pad (D-G), hard and soft palate (H and I) and the tongue (J-L). Occasionally, they may be found in the nostrils (M and N).



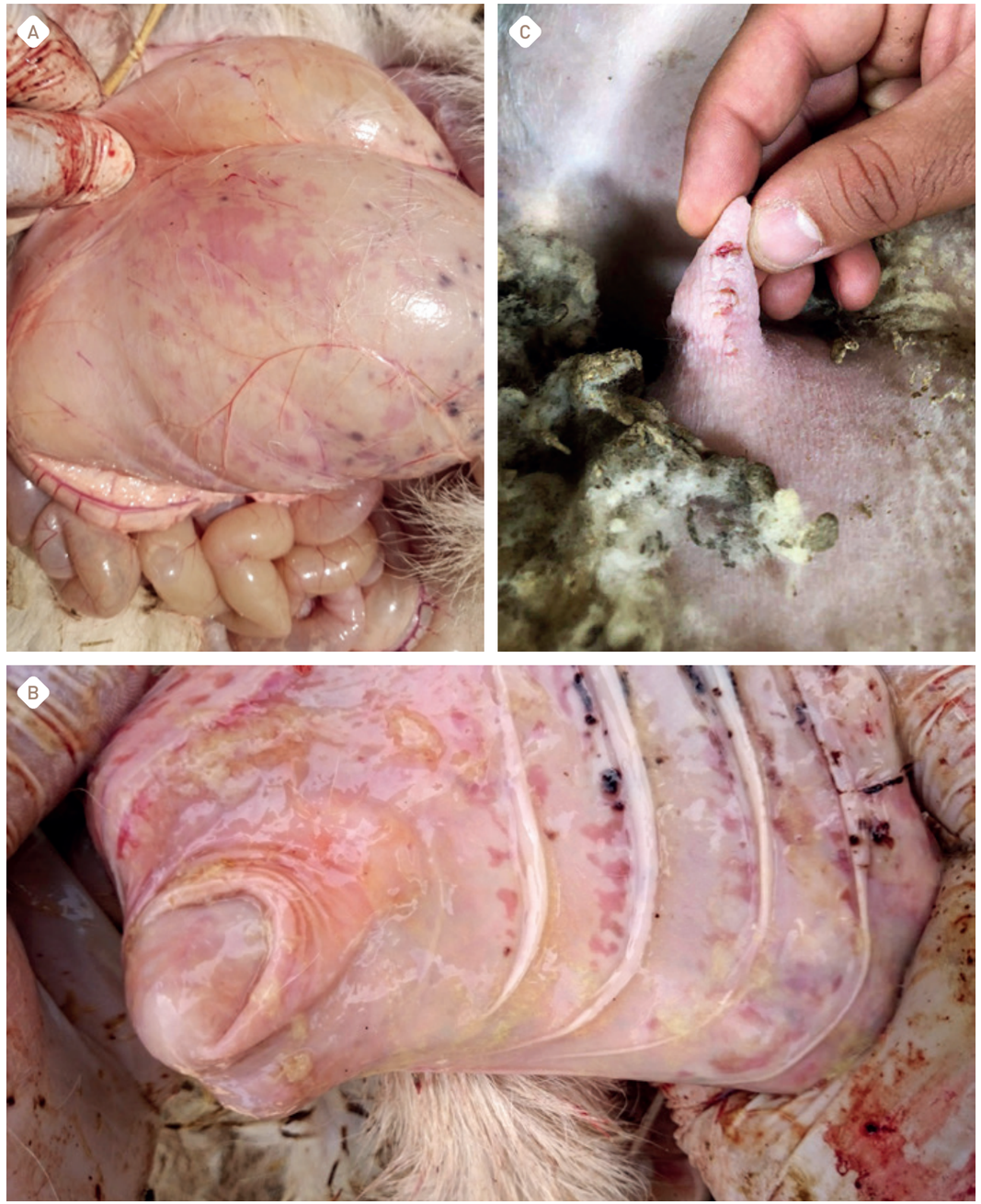
4.326. **Scars.** Erosions tend to turn into scars in 2 or 3 days (A-D).



4.327. **Tiger heart.** FMD virus has a high affinity for the cardiac tissue of kids and lambs, and causes their death through necrosis of heart tissue and heart failure. Some neonates who do not die from heart disease do not grow well due to the presence of necrotic tissue in the heart and chronic heart failure. In most cases of neonatal mortality there are not any vesicles or erosions in the oral cavity, but the tiger heart, due to necrosis of the heart muscle, is observed (A-C). Mortality up to 55% in kids has been observed even with no clinical signs in adult animals (D).



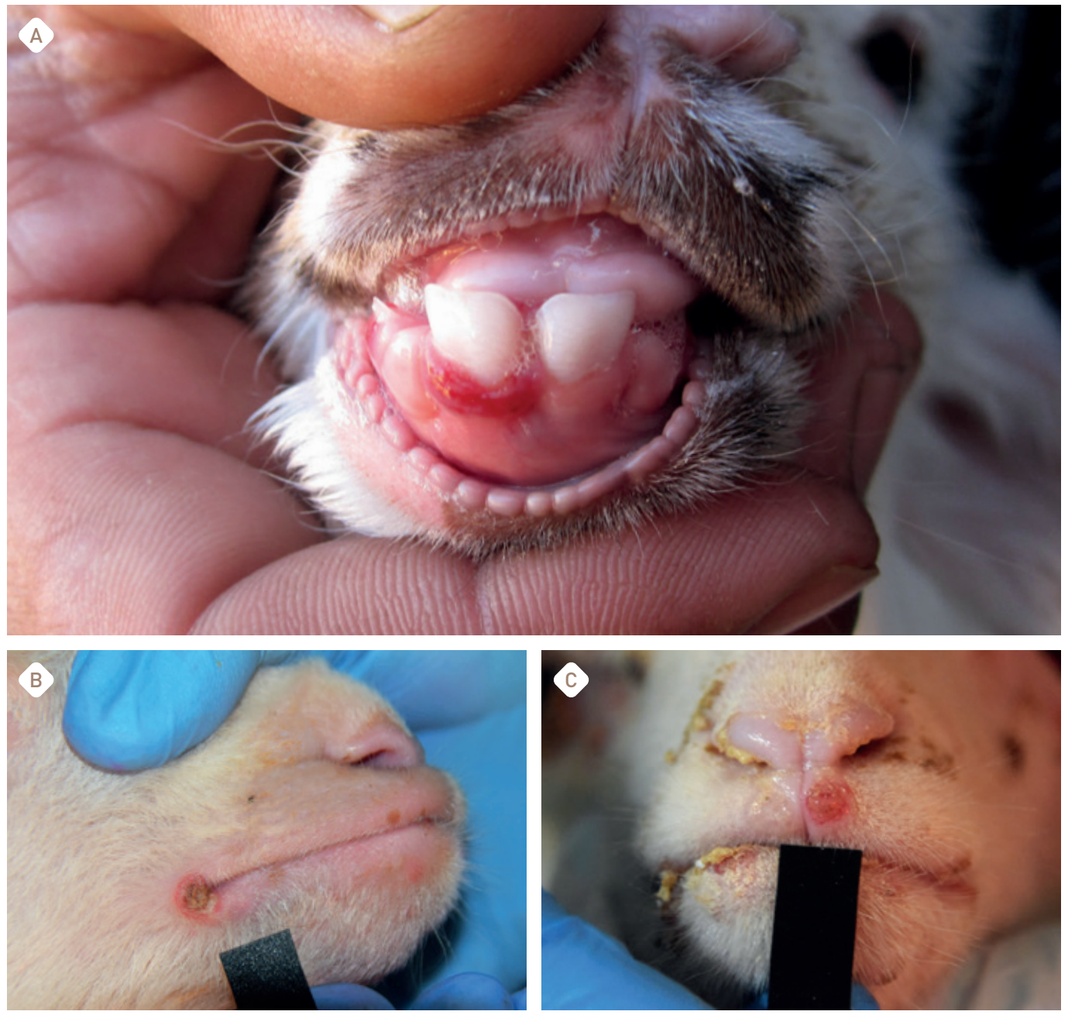
4.328. **Lameness in adults.** If it is not the lambing season, usually the first clinical sign in FMD affected flocks is lameness in adult animals, which quickly involves an increasing number of animals. Vesicles may be found along the coronary band, in the interdigital space, and above the heels (A-E).



4.329. **Other locations.** FMD lesions can be seen in the abomasum and udder tissue (A-C).

CONTAGIOUS ECTHYMA (ORF)

Contagious ecthyma, also known as orf, is a highly contagious eruptive skin condition of sheep and goats. Orf virus belongs to the genus *Parapoxvirus,* family *Poxviridae,* sub-family *Chordopoxvirinae.* Orf disease usually shows high morbidity and low mortality and affects mainly young animals in the first year of their life, with severe outbreaks generally associated with intensive sheep husbandry.
Clinical presentation in lambs or kids includes the formation of vesicles, papules, pustules with a yellowish creamy and/or proliferative appearance, then scabs that finally become dry and shed, usually with no scar remaining. Although the most common lesions can be found in the oral cavity, these can be extended to the skin of the face, ears, mammary gland, feet, flanks, scrotum and perianal area.



4.330. **Oral presentation in young animals. Early stages of the lesions.** Primary lesions usually begin on the lips or in the mucous membranes of the gums and oral cavity (A-C).



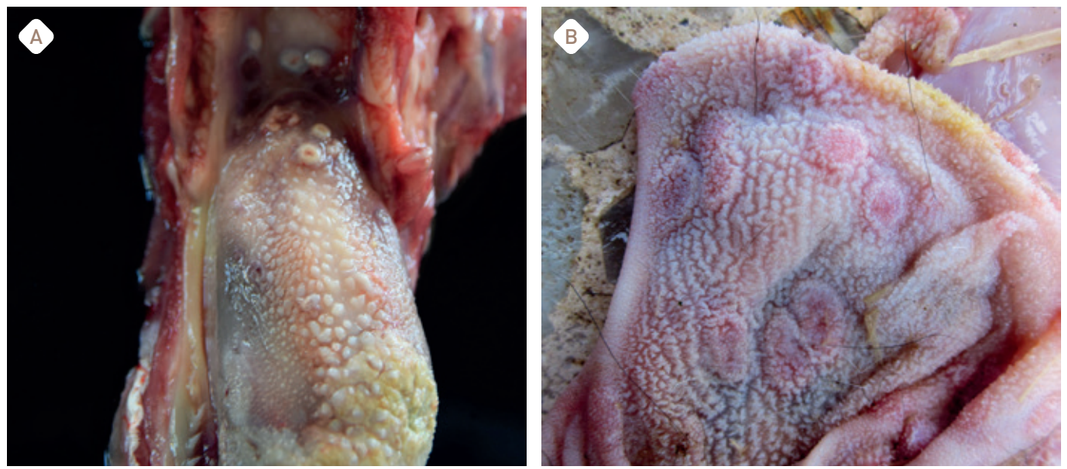
4.331. **Oral presentation. Second stage.** Vesicles turn into pustules, which rupture, and scabs and proliferative lesions begin to appear (A-D).



4.332. **Oral presentation. Third stage.** The proliferative lesions can become so severe that they can be observed without opening the mouth (A and B). Secondary infections generally occur (C and D).



4.333. **Oral presentation. Spread of the infection to the head and neck.** In this presentation, which is seen mainly in older than two month kids, the lesion starts in the upper and lower eyelids, then without affecting the mouth until the end of the illness course, it extends to the areas around the lips and muzzle (A and B), and finally to other areas of the face, ears and neck skin (C) and from there to the rest of the body (D).



4.334. **Internal spread.** Orf lesions are also seen in the pharyngeal mucosa and gastrointestinal tract (A and B).



4.435. **Mammary presentation.** Orf lesions appear in the teats and udders of nursing dams. The painful lesions that affect the mammary gland cause the ewe to prevent the lamb from nursing normally. The hungry lambs or kids try to find the milk in other dams, contributing to the spread of the disease.



4.436. **Foot presentation.** Foot ecthyma is found less frequently than in the previous forms. Lesions are usually located in the transition zone from the skin to the hoof (A-C).



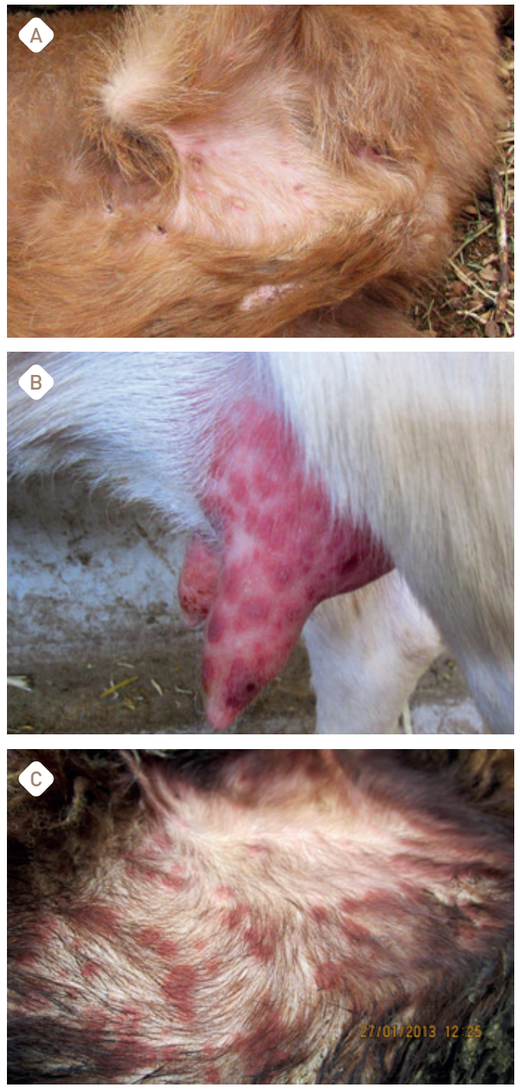
4.437. **Oral orf in adult animals.** Orf lesions in adults are seen sporadically, although occasionally may have high morbidity in the affected flock (A). Sometimes large masses of granulation tissue develop under the scabs, which frequently harbour secondary infections (B) or even secondary myasis. Occasionally, severe facial oedema can occur in association with oral lesions (C).



4.438. **Human orf. Zoonosis.** The causative virus of the disease may establish painful lesions in humans, usually in the form of a proliferative ulcer (A and B). An infected human can transmit the virus from one animal to another.

SHEEP AND GOAT POX

The causative agent of sheep and goat pox is a DNA virus that belongs to the *Poxviridae* family and *Capripoxvirus* genus. Small ruminants can be infected at any age, but young and lactating lambs and kids are more prone to suffer the disease. Pox is a highly contagious disease in sheep and goats with a morbidity rate ranged 75-100%, depending on the strain of the virus.



4.439. **Macular red lesions.** They are 1-3 cm in size. This form appears on the skin of the animal 2-5 days after the onset of fever, especially in hairless areas (A-C).



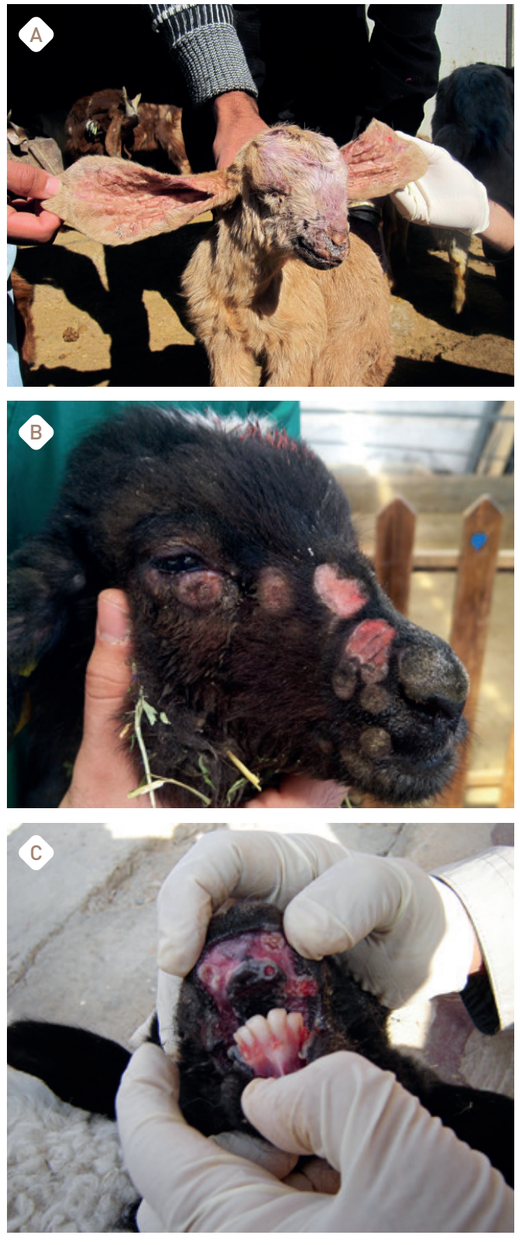
4,440. **Vesicles affecting the tail and vulva.** Lesions in the vulva quickly become infected and ulcerate (A and B). *Picture B courtesy of Dr. H. Ghasemifard.*



4.441. **The stage of vesicle and pustule formation.** Note the pus inside the vesicles. The vesicles and pustules turn into scabs (A-C). In the benign form of the disease, which is more common in adults, lesions are seen only on the skin, especially under the tail.



4.442. **Stonepox.** A nodular form of sheep and goat pox is called stonepox, which is similar to lumpy skin disease in cattle. Vesicles and pustules are absent, and there is no cross-immunity with other goat pox or orf viruses. *Picture courtesy of Dr. M. Mombeini.*





4.443. **Young animals.** Pox is more severe in young animals and develops a generalised disease, which affects the entire skin and the respiratory and gastrointestinal mucosa (A-C). *Picture A courtesy of Dr. M. Moghaddasi.*



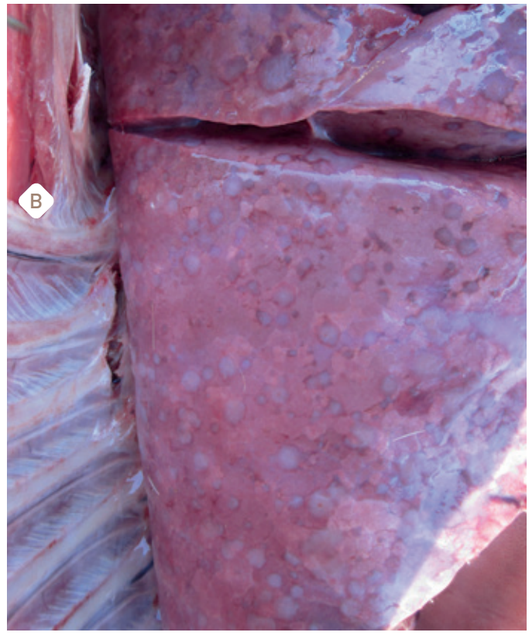


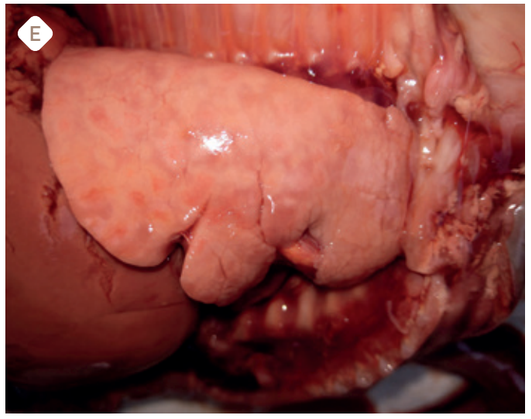
4.444. **Mammary and reproductive pox.** The pox lesions can be observed on the mammary gland (A and B), the prepuce (C), scrotum (D) and testis (E). Abortion occurs as a result of natural disease or after applying a live attenuated vaccine to sensitive breeds, such as the Saanen goat (F and G).





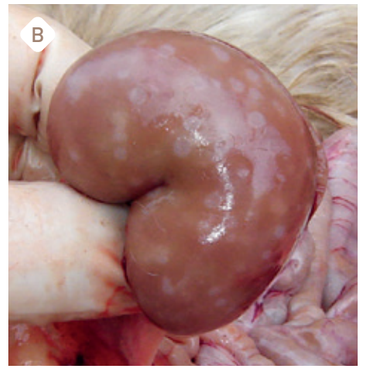






4.445. **Respiratory and alimentary pox.** Oral lesions on the tongue and gums tend to ulcerate (A). Animals that die after natural infection frequently have lesions in the lungs (B) and in the digestive tract (C and D). It can also be seen in lambs born to mothers vaccinated with live vaccines (E).







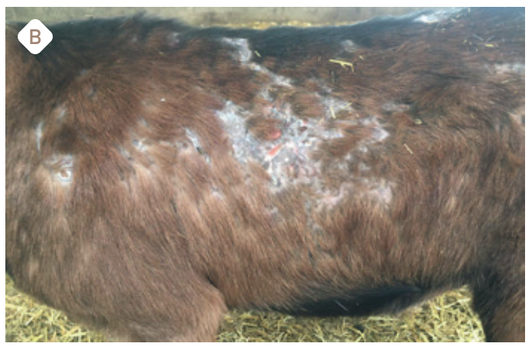
4.446. **Other lesions.** Pox lesions can also appear in the heart (A), urinary system (B) or even in the eyes (C).





4.447. **Pox bumps presentation.** Pox bumps affecting all the skin (A). In this clinical presentation, vesicles and pustules are not formed. A full milk udder indicates that the goat is recovering from the disease and is beginning to produce milk normally (B).





4.448. **Recovery.** The animals recover, but the scars remain (A and B). Pox lesions join together during the recovery period and cause loss of skin epithelium in some areas.

PESTE DES PETITS RUMINANTS (PPR)

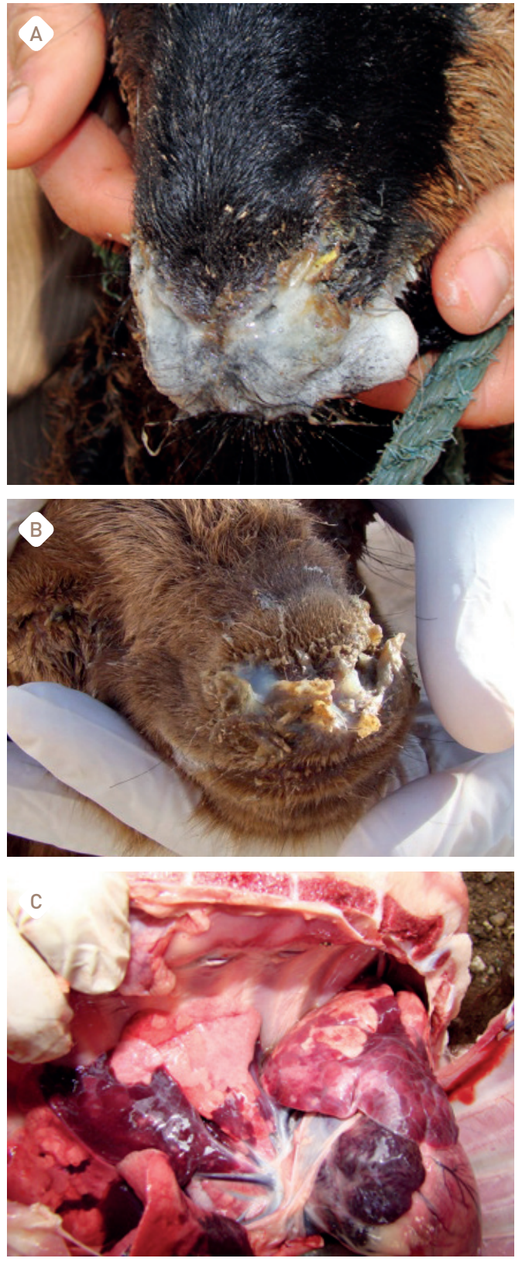
Peste des petits ruminants (PPR) is caused by a *Morbillivirus* closely related to the Rinderpest virus, and affects goats, sheep, and some wild relatives of domesticated small ruminants, as well as camels. Mortality rates are also much higher in goats (55-85%) than sheep (less than 10%).



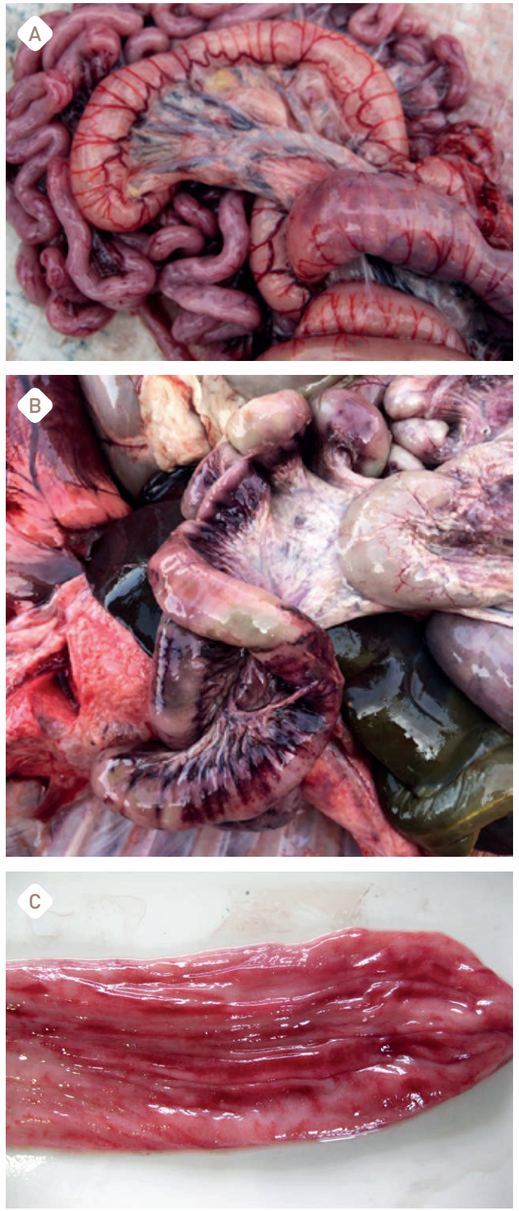
4.449. **Initial clinical signs.** Clinical signs usually appear 3 to 6 days after exposure to the virus in the form of high fever, dullness and serous ocular and nasal discharge (A-D). *Picture A courtesy of Dr. M. Mombeini.*



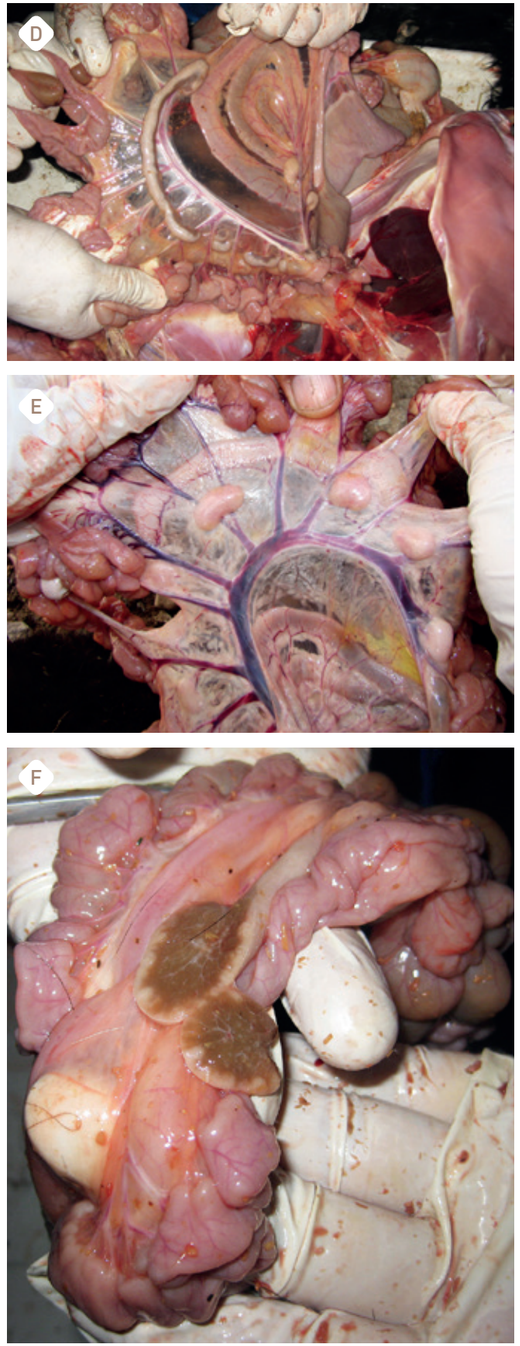
4.450. **Clinical signs.** A couple of days later, necrotic lesions appear in the oral cavity and expand over the entire oral mucosa, forming diphtheritic plaques (A-E).



4.450. **Respiratory.** Respiratory clinical signs follow the onset of the disease, which is aggravated by secondary bacterial pneumonia (A-C).



4.451. **Intestinal ulceration.** Haemorrhagic ulceration is marked in the intestines, which may produce typical "zebra lines" (A-C). Regional lymph nodes are usually enlarged and show a wet appearance on the section (D-F). *Picture F courtesy of Dr. H. Bazyar.*





4.452. **Diarrhoea and erosions.** Diarrhoea develops within 3 to 4 days after fever onset (A), and some erosions can be observed in the vulva and prepuce (B).