2.13. **Cannibalism.** In certain circumstances, abnormal behaviours can be observed in the mothers, which can bite the neonate's hooves, ears or tail (A-C). They are usually associated with mineral deficiencies during pregnancy or abnormal behaviour of unknown aetiology.

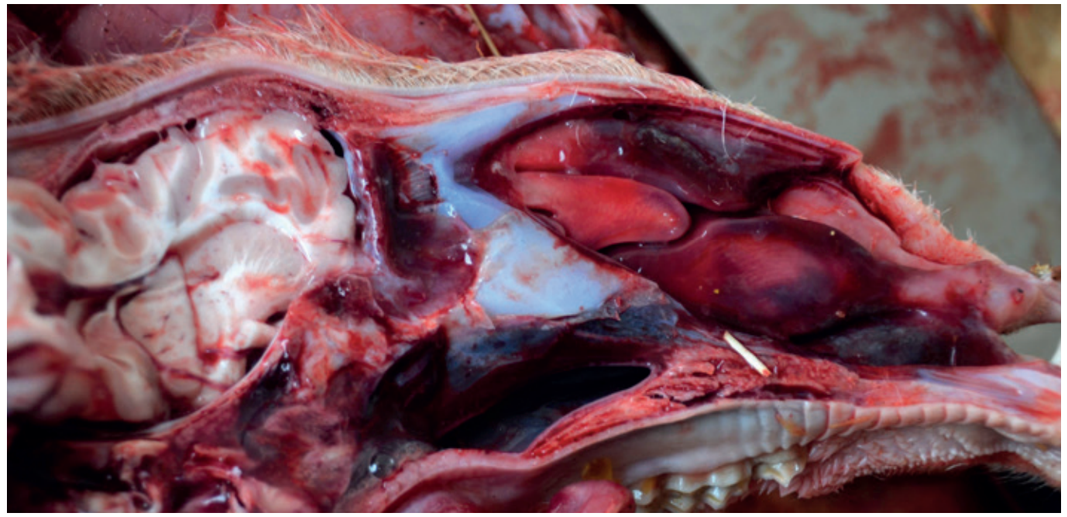
2.14. **Accidents.** When the facilities and materials are not well designed, young animals can get hooked and/or hang (A and B).

Other disorders that affect the neonate, such as pneumonia (*see chapter 3*), diarrhoea (*see chapter 4*), and enzootic ataxia (*see chapter 8, page 422*), will be seen more extensively in the corresponding chapters.

Respiratory disorders

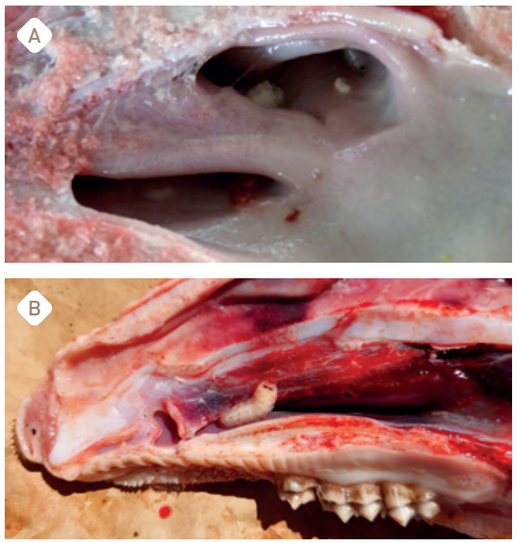
RESPIRATORY DISORDERS IN LAMBS AND KIDS

RHINITIS



3.1. **Clinical signs.** Rhinitis in lambs/kids is associated with ventilation problems due to a high concentration of irritating gases or environmental factors (temperature, humidity, etc.). It is a frequent lesion observed in the ovine respiratory complex.

3.2. **Oestrosis.** Although it is not as frequent as in adults, oestrosis can be diagnosed in lambs born and raised in the pasture. This never happens in housed lambs. The nostrils may be stained with bloody remains provoked by the muco-haemorrhagic discharge caused by the larva of *Oestrus ovis*.



3.3. **Oestrosis. Postmortem findings.** Different larval stages can be found in the nasal cavity (A and B).

TRACHEITIS

3.4. **Tracheitis.** Occasionally, tracheitis is observed in young animals. Its origin has not been well defined but seems related to infectious disorders or irritating gases due to poor ventilation, as described in rhinitis (A and B).

CLINICAL SIGNS OF LOWER RESPIRATORY TRACT DISORDERS

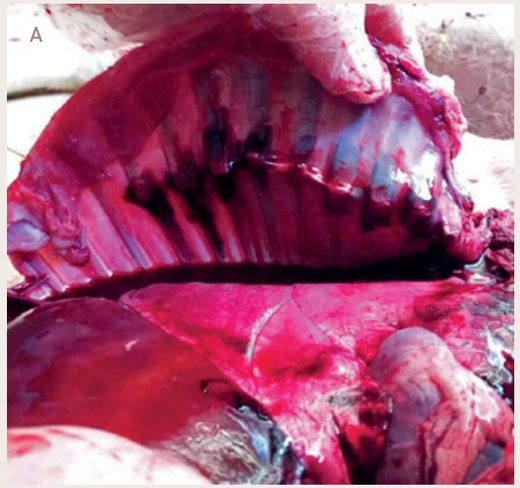
3.5. **Feeling cool.** Pneumonic animals, along with weak and hypothermic lambs or kids, seek the heat of the first rays of the sun (A). In this group, it is easy to differentiate those with respiratory disorders by their shrunken posture, feverish appearance and growth retardation when the process chronifies (B).

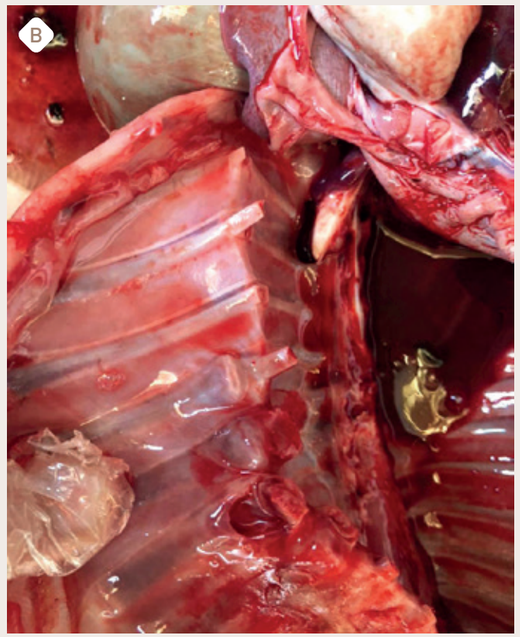
3.6. **External appearance.** Affected animals have sad faces, floppy ears, nasal secretions (rhinitis), and epiphora (A-C).

3.7. **Dyspnea and cough.** When they are severely affected and we force them to move quickly, they begin to show severe expiratory dyspnea, with streaking mouth breathing and cough.

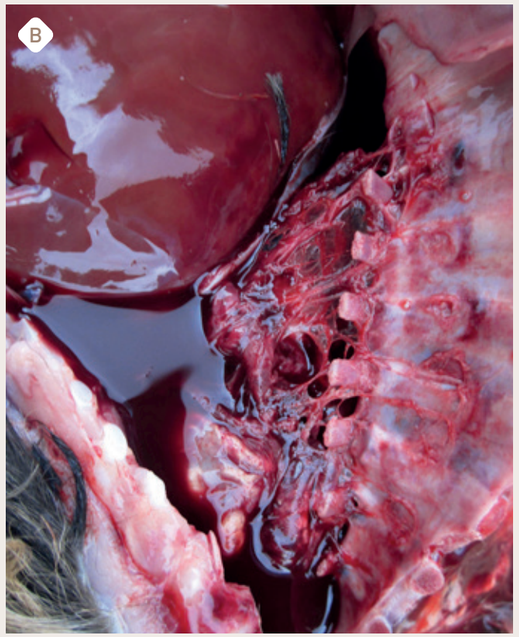
3.8. **Provoke coughing.** It is essential to differentiate if the cough is due to respiratory disorders or produced by the pressure of the feeders in the trachea when they are too high for the small size of lambs or kids.

RIB FRACTURES AND PNEUMONIA

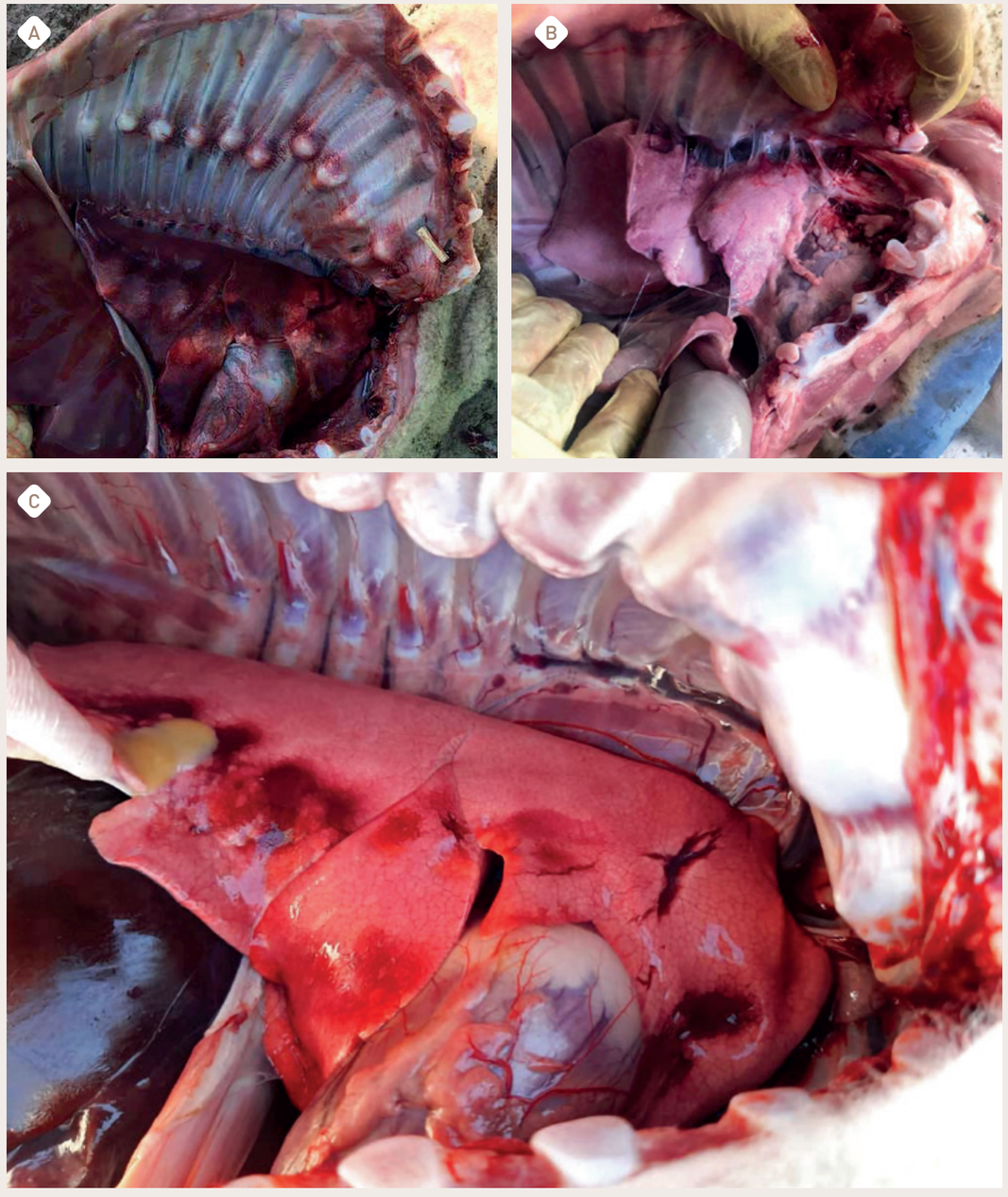




3.9. **Dystocia and rib fractures.** During the resolution of dystocic deliveries, fractures of different bones can occur, being the ribs the most frequently affected. In the most severe cases, chest haemorrhages can be present with peracute or acute pneumonia that can lead to the sudden death of the animal (A and B).



3.10. **Pneumonia.** When the animal survives, lung lesions can be developed, varying from localised foci caused by the sharp tips of the broken ribs in the lung parenchyma (A) to more extensive areas of pleurisy and fibrinous pneumonia (B).



3.11. **Bony callus formation and pneumonia.** When the process progresses, the rib fractures create voluminous bony calluses that usually lead to pneumonia (A). These can show pleural adhesions (B) or simple atelectasis caused by the pressure of the calluses in the lung parenchyma (C).

DEVIANT SWALLOWING AND GANGRENOUS PNEUMONIA

3.12. **Deviant swallowing.** Young lambs or kids, especially if they have low vitality, may suffer deviant swallowing of milk or colostrum, and in many cases, milk can be seen coming out through the nostrils (A). Lambs or kids born with cleft palate (palatoschisis) have a particularly higher risk, when they attempt to suckle from their mother or are bottle-fed. In addition to milk or colostrum, it is possible to find plant remains in the trachea (B).

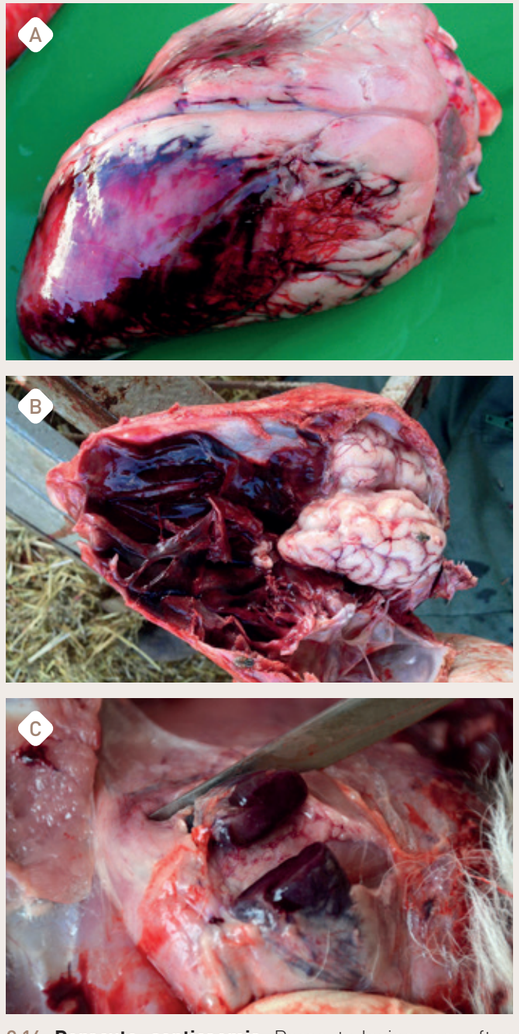
3.13. **Aspiration pneumonia.** The entry of milk into the lungs can cause suffocation of the animal or lead to the onset of inhalation pneumonia in the cranioventral lobes of the lung.

3.14. **Gangrenous pneumonia.** If the animal survives, the lung lesion with the ingested material and the microbial contamination evolves into gangrenous pneumonia. This can lead to significant lesions with growth retardation and a characteristic smell of the exhaled air.

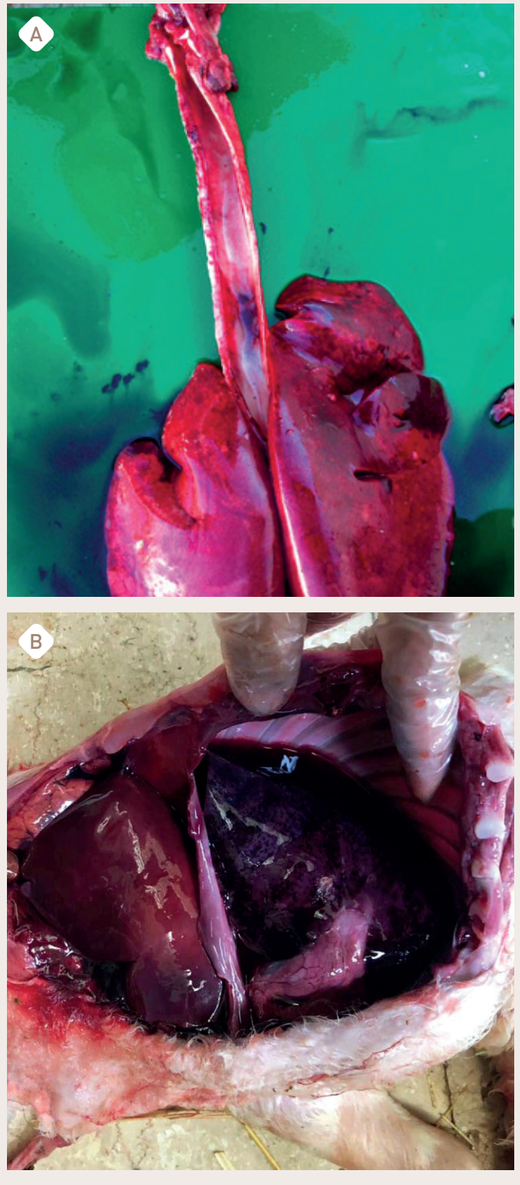
OVINE RESPIRATORY COMPLEX (ORC)

Ovine Respiratory Complex (ORC) is a complex disease process involving a range of host-pathogen-environment interactions (HPE), where host immunological and physiological mechanisms (host) interact with multiple aetiological agents, including bacteria (pathogen), plus environmental factors or stressors (environment). Several commensal microorganisms of the nasopharynx, tonsil and lungs of healthy sheep have been associated with ORC: *Mannheimia haemolytica, Pasteurella multocida, Bibersteinia trehalosi* and *Mycoplasma* sp., usually in mixed isolations.

3.15. **Peracute: sudden deaths.** The peracute presentation is frequent but not easily detected by the farmer. Its aetiology is mainly related to *B. trehalosi*. After sudden deaths, haemorrhagic foam can appear through the nostrils (A). The skin not covered by wool may show a soft red-violet colour (B).

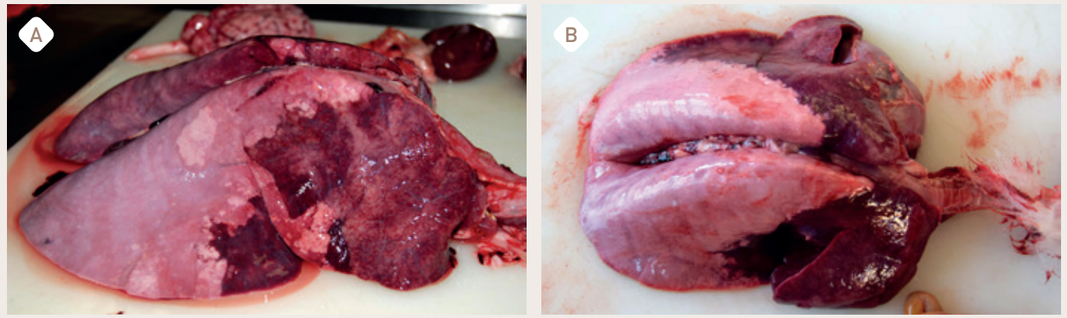


3.16. **Peracute: septicaemia.** Peracute lesions are often characterised by haemorrhages (A), especially in the nasal cavity (B), and pulmonary oedema. Retropharyngeal lymph nodes are usually congestive or haemorrhagic (C).

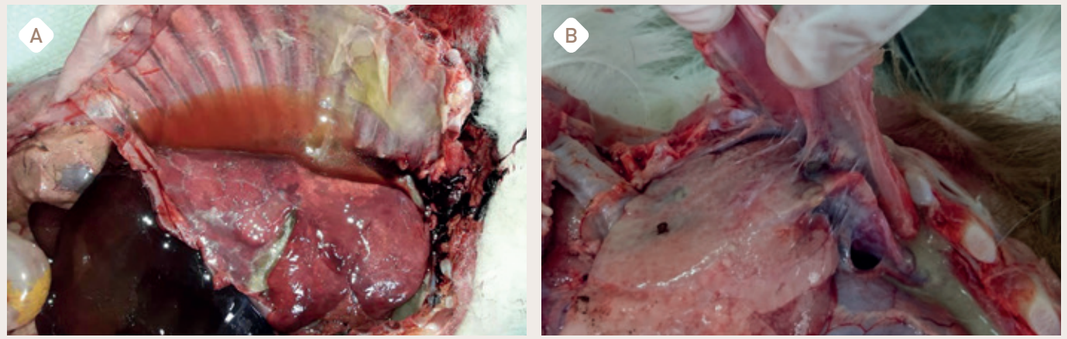


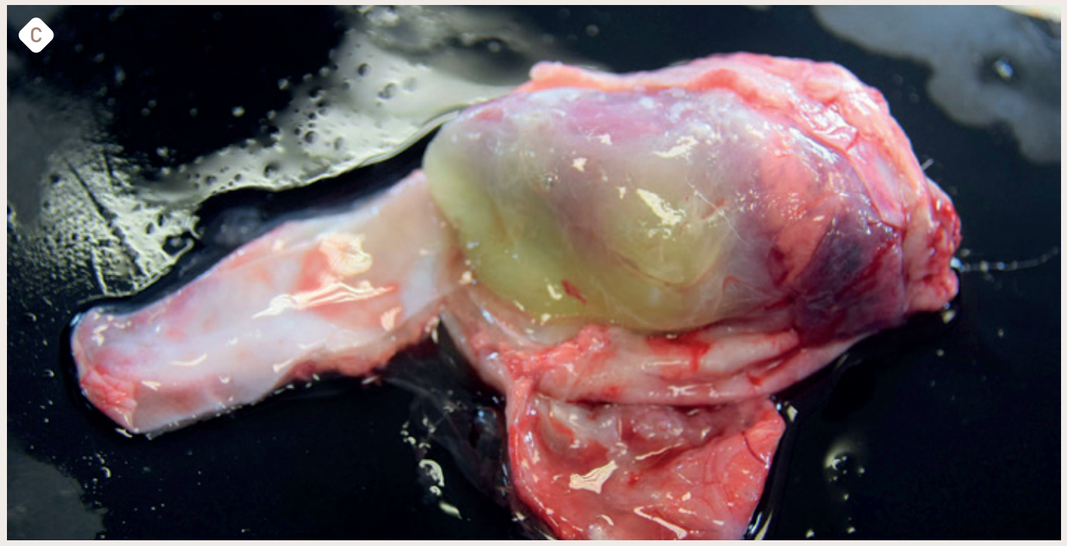
3.17. **Peracute: lung lesion.** In peracute cases of ORC, pulmonary oedema and congestion are seen, and foamy content can be present in the trachea (A). The lungs show severe congestion, sometimes accompanied by petechiae and ecchymosis, and serosanguineous fluid can be seen in the pleural cavity (B).

3.18. **Acute: clinical signs.** The main clinical signs in the acute ovine respiratory complex are fever, nasal discharge, anorexia, depression, lethargy and severe expiratory dyspnea (A). When the animal is severely affected, it cannot breathe properly and take an orthopneic position with the anterior limbs separated and the neck stretched out (B). Acute ORC is mainly associated with *Mannheimia haemolytica*, although mixed infections are common.



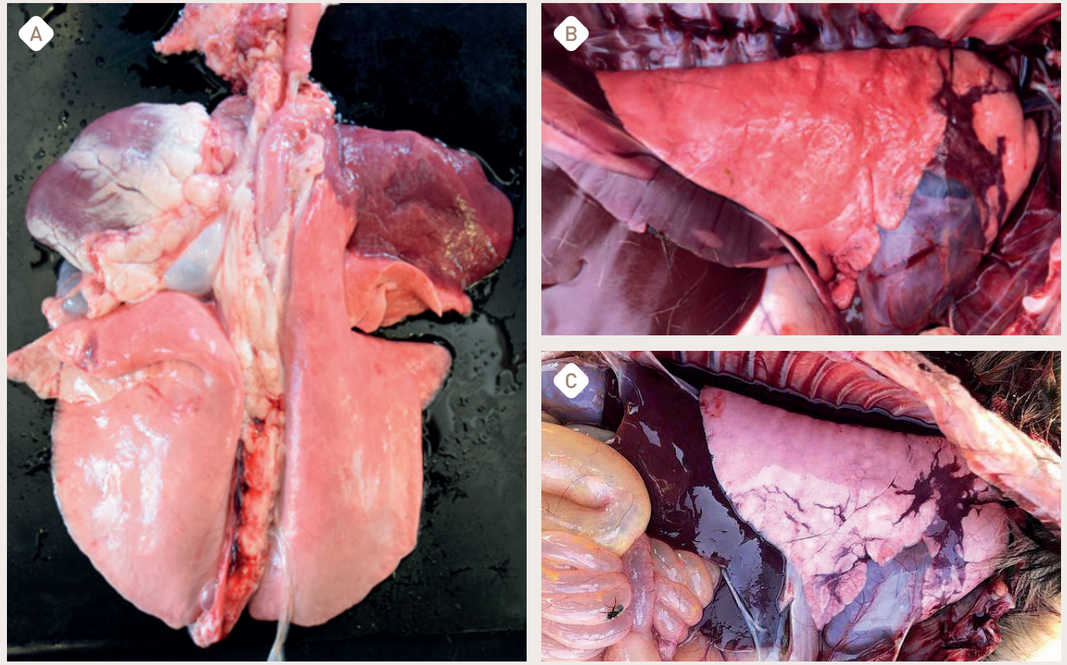
3.19. **Acute: postmortem findings.** The most common pattern observed in the postmortem examination of acute ORC is fibrinous or necrotic bronchopneumonia with variable degrees of vascular damage (A and B).



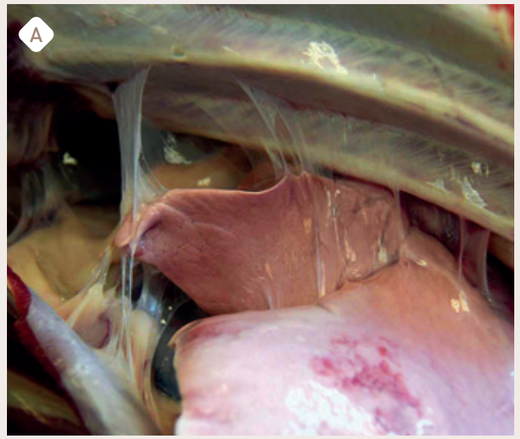


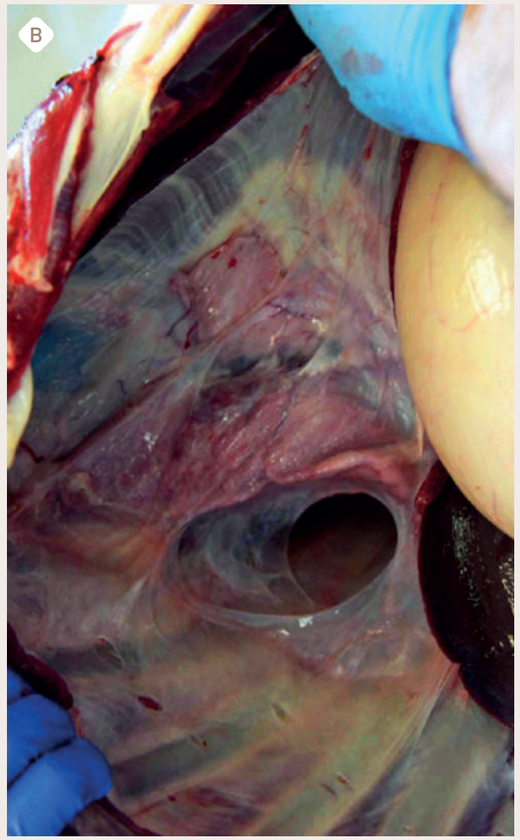
3.20. **Acute: fibrinous pleurisy.** Acute ORC is usually accompanied by fibrinous pleurisy, affecting parietal and visceral pleura (A and B). Fibrinous pericarditis is also occasionally seen (C).

3.21. **Chronic: clinical signs.** Chronic ovine respiratory complex causes mixed dyspnea, cough and chronic weight loss. Clinical signs can often go unnoticed or not be related to respiratory disorders. Chronic ORC is mainly associated with *Pasteurella multocida* and *Mycoplasma* sp.

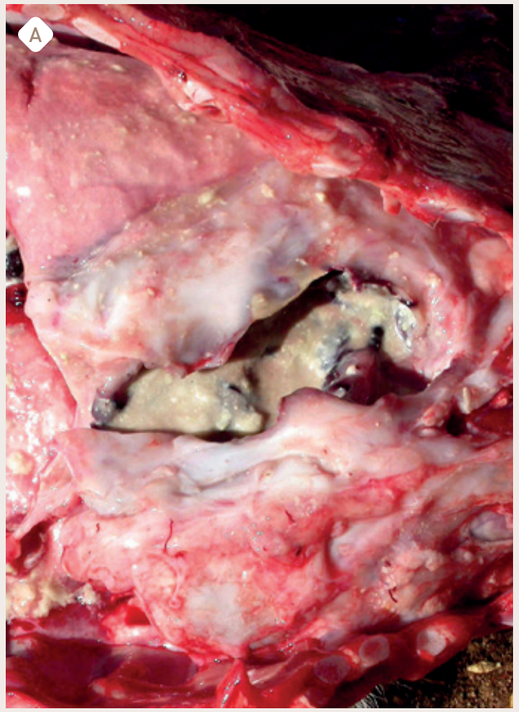


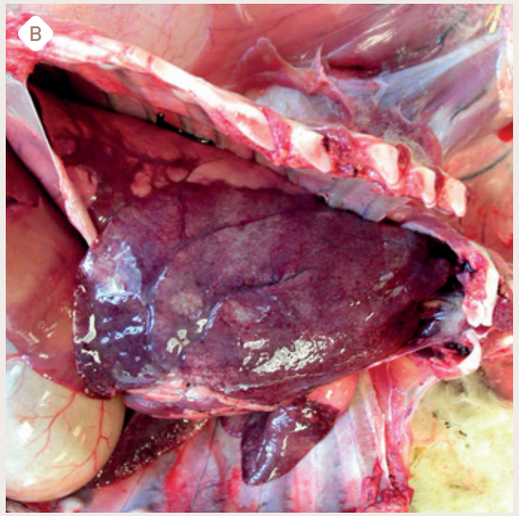
3.22. **Chronic: chronic suppurative pneumonia.** Chronic suppurative pneumonia, mainly located in the cranioventral lung lobes, is the most common lesion in chronic ORC (A). If enough time passes, these types of pneumonia may be found in the resolution phase (B). In some cases, more or less abundant hydrothorax is observed (C).





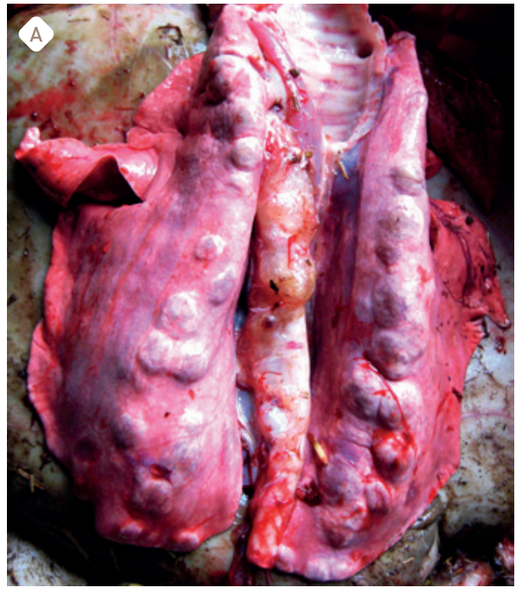
3.23. **Chronic: fibrous pleurisy.** Fibrinous pleurisy progresses to fibrous pleurisy with pleural adhesions (A and B).

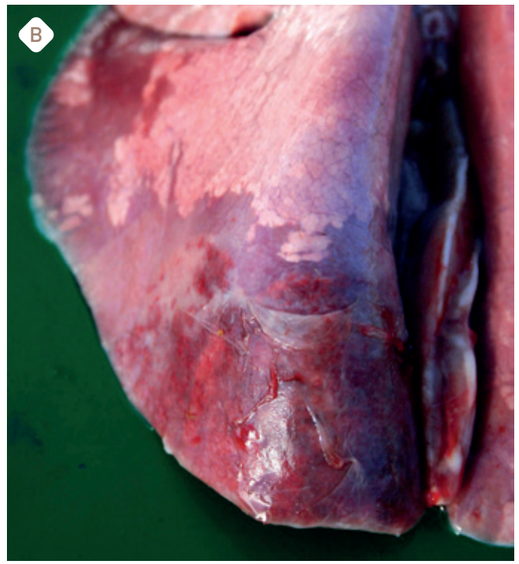




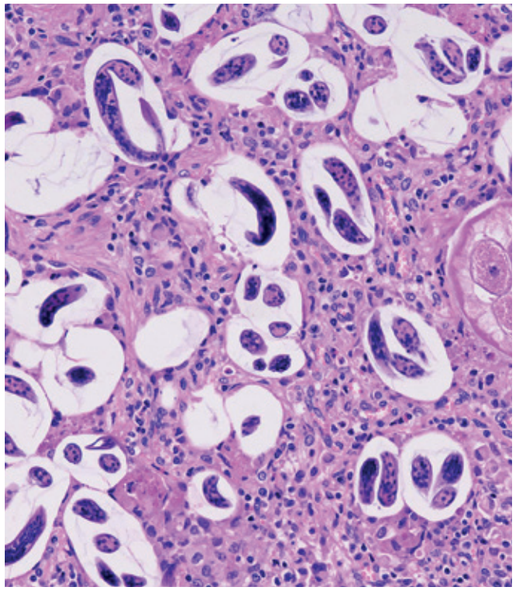
3.24. **Chronic: evolution of the process.** Occasionally, animals may end up presenting gangrenous pneumonia (A) due to difficulty in breathing, or a lesion exacerbation may occur (B).

VERMINOUS PNEUMONIA





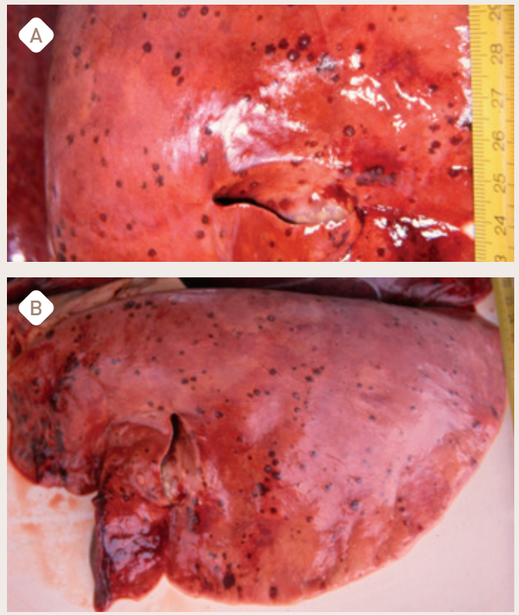
3.25. **Verminous pneumonia.** In grazing lambs or kids, same as in adults, parasitic nodules of small pulmonary strongyles can be found on the dorsal area of the lung (A). Likewise, atelectasis caused by *Dictyocaulus filaria* is observed in the diaphragmatic lobes (B).



3.26. **Histopathology.** Parasites can be seen in the histologic sections of the lung parenchyma. *Picture courtesy of Dr. J. A. Castillo.*

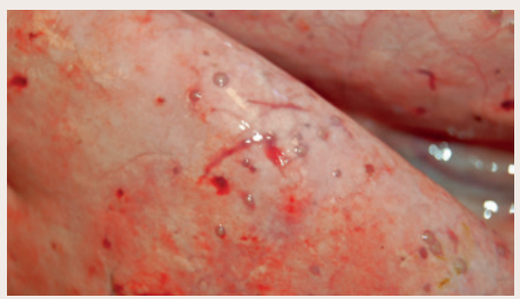
3.27. **Larvae in faeces.** The coprological test allows us to differentiate the larvae from the small and large lung strongyles since they present very different sizes. *Picture courtesy of Dr. J. A. Castillo.*

PULMONARY ASPERGILLOSIS



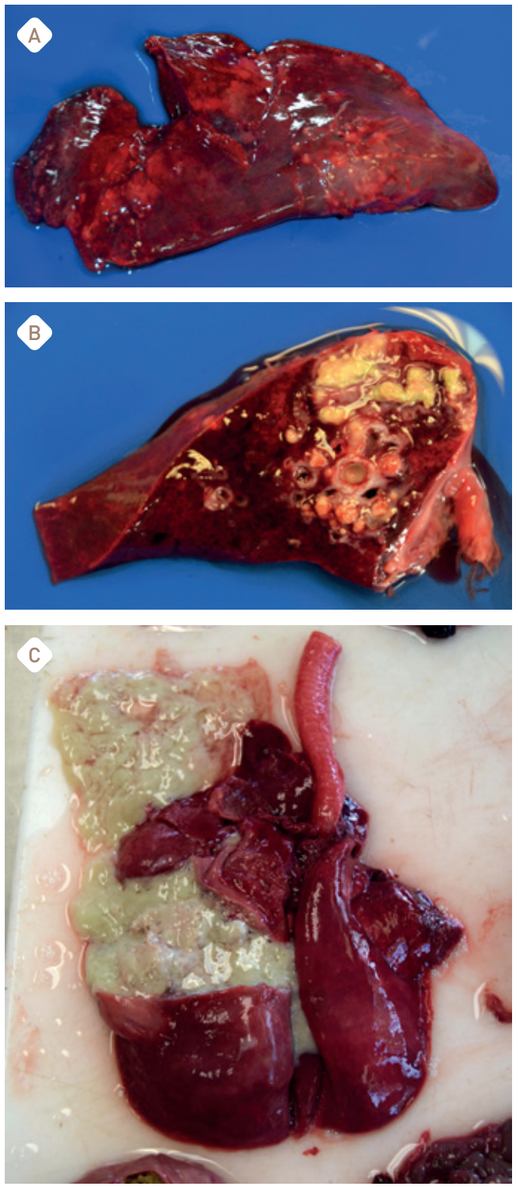
3.28. **Pulmonary aspergillosis.** Fungi of the genus *Aspergillus* can cause pneumonic lesions in lambs, especially in the youngest. Affected animals show mild/severe dyspnea. At necropsy, lungs appear with the surface covered with grey-whitish nodules and surrounded by a red halo (A). The size of the nodules is variable, and there may even be more extensive foci similar to atelectatic areas (B).

PULMONARY CYSTICERCOSIS



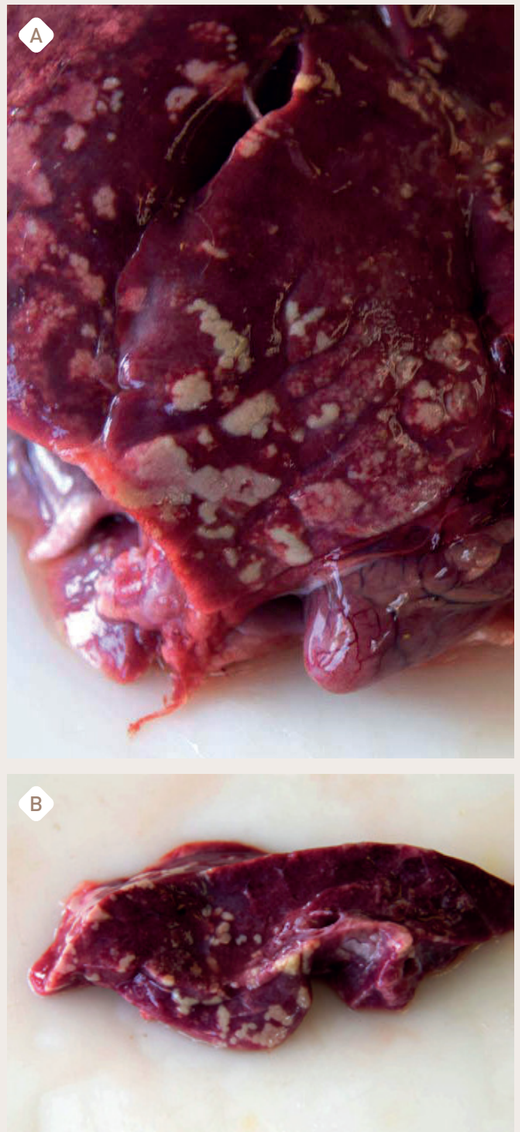
3.29. **Pulmonary cysticercosis.** Parasitic points and pathways of different sizes can be located on the surface of the lung in a subpleural location. Usually, this finding is complemented by similar or greater lesions on the liver.

PURULENT PNEUMONIA



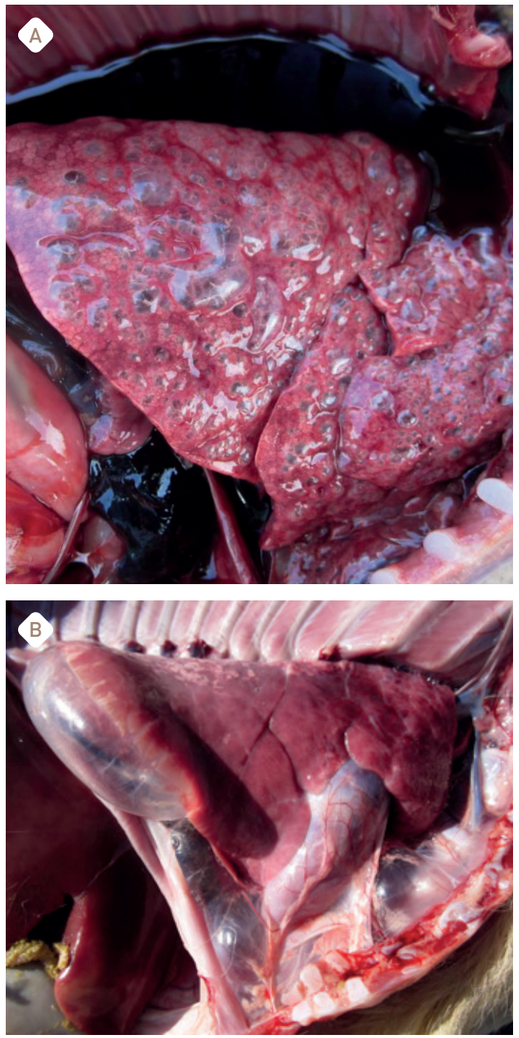
3.30. **Purulent pneumonia.** Contamination of the lung with pyogenic microorganisms produces abscesses that can be seen on the surface (A) or the section (B). Some of them can reach a relevant size (C).

PULMONARY NECROBACILOSIS



3.31. **Pulmonary necrobacilosis.** Infection with *Fusobacterium necrophorum* leads to necrobacilosis, forming necrotic well-delimitated areas with a dry appearance on section (A and B). These microorganisms can reach the lung from the umbilical cord, affecting also other organs.

EMPHYSEMATOUS BLEBS AND BULLAE



3.32. **Emphysematous blebs.** A pulmonary bleb is a small collection of air between the lung and the outer surface of the lung (visceral pleura), usually found in the upper lobe of the lung and caused by a rupture of alveoli (A). When a bleb ruptures, the air escapes into the chest cavity, causing a pneumothorax, resulting in a collapsed lung. If blebs become larger or come together to form a larger cyst, they are called bullae (B). Blebs or bullae are of congenital or acquired origin after ingesting toxic plants or severe pulmonary infections.