Pneumonia

Diseases of the lower respiratory tract of sheep are not considered a serious cause of lost productivity in Australian flocks because of the generally sporadic nature of serious respiratory disease and the sub-clinical nature of most cases of respiratory infection. The extensive conditions under which Australian sheep are usually managed do not favour transmission of pneumonic infectious agents and also make detection of the disease less likely.

The evidence from abattoir surveys in Australia and more detailed investigations in New Zealand and European countries suggest that we may be underestimating the effect that pneumonic diseases have on our sheep flocks. Pneumonic lesions are frequently seen at necropsy in sheep of all ages and clinical signs are frequently evident when flocks of sheep are driven. It is likely that, either alone or in combination with other disease conditions, respiratory diseases are a significant cause of loss to the Australian sheep industry. The extent of the problem in Australia, its economic impact or the success of any control measures have not been researched.

The important pneumonic conditions of sheep in Australia can be first classified as parasitic or microbiological. The latter classification is non-specific and encompasses conditions which vary between mild and severe, chronic and acute, and include a number of aetiological agents which can be viral, mycoplasmas, bacterial or any combination of these.

Of lesser importance are aspiration pneumonia and some bacterial infections which may or may not involve the lungs (CLA, tuberculosis, melioidosis).

The microbiological agents associated with pneumonia in sheep include:

- *Mycoplasma ovipneumoniae, M arginini*
- Viruses; *Parainfluenza 3 (PI3)* virus principally but also *ovine adenovirus type 6, bovine adenovirus type 7 and respiratory syncytial virus, type 3 reovirus, maedi, pulmonary adenomatosis*
- *Bordetella parapertussis*
- *Pasteurella haemolytica, P multocida*
- A number of other bacteria including *Actinobacillus lignieresii, Actinomyces pyogenes, Klebsiella spp, Escherichia coli, Histophilus ovis, Fusobacterium necrophorum and Neisseria spp*[1][2][3]

These and possibly other, as yet unidentified, agents cause a spectrum of pneumonic diseases which vary from mild to severe, acute to chronic, proliferative interstitial to exudative. The most severe, and those which cause mortalities and the most obvious clinical signs, are associated with bacterial infections, particularly *P haemolytica*. The mildest, which cause either no clinical signs or which cause only mild
Clinical signs after extended periods of development, are associated with mycoplasma infection and from which viruses may or may not also be isolated.

The spectrum of disease is such that pneumonic lesions and clinical signs may fall anywhere between the two extremes. It is evident, however, that the mycoplasmas, with or without viral assistance or induction, are the usual initiating infectious agents and bacteria the secondary invaders. The outcome of the initial infection is determined by a number of complex environmental, genetic and immunological factors. A number of classifications, in common usage but which do not particularly make the aetiology or severity of infection any clearer, are applied to different types of pneumonia in different types of sheep and in different countries.

The term *enzootic pneumonia* is applied by some authors to the whole spectrum of lower respiratory tract infections, from the mild, chronic proliferative interstitial pneumonia caused by mycoplasmas and PI3, to the sub-acute pneumonia associated with mycoplasma infection and secondary bacterial infection (commonly called *summer pneumonia*) involving a combination of proliferative and exudative reactions in the lungs, to the acute exudative pneumonia of pasteurellosis which has much in common, pathologically with shipping fever of cattle[2].

Other authors use the term *enzootic pneumonia* to describe only the most severe, acute and exudative forms of pneumonia which are associated with bacterial (usually *Pasteurella* spp) infection[4]. This restricted use of the term denies the almost certain connection to milder forms of pneumonia through the common mycoplasmal initiation and also loses the hint of consistency in terminology with the porcine disease associated with *M hyopneumoniae*.

In New Zealand, the term *chronic non-progressive pneumonia* or *CNP* is used to describe a sub-acute or chronic pneumonia which is enzootic in lambs of slaughter age[5]. This disease is associated with reduced growth rates of lambs[6] and with increased trimming and condemnation of carcasses at slaughter[7]. The disease appears to have much in common with *summer pneumonia* of Australian flocks, including the seasonal rise in incidence, but its impact is recognised in a different way because of the different management systems and production objectives of sheep flocks in the two countries. *Acute fibrinous pneumonia* is a progression from the milder form and associated with secondary infection with *Pasteurella* spp and other bacteria[8] or caused by *P haemolytica* alone. Both *CNP* and *acute fibrinous pneumonia* are regarded as forms of *enzootic pneumonia*.

Mycoplasma pneumonia

In Australia, outbreaks of pneumonia of lambs with high morbidity, low mortality and apparently significant deleterious effects on growth rate and exercise tolerance were recorded from Queensland[9] and south eastern Australia[10][11]. The condition in south eastern Australia became known as *summer pneumonia* because of an increase in the prevalence of the disease in the summer and early autumn months. Two types of *Mycoplasma* spp were frequently associated with the disease and these were subsequently identified as *Mycoplasma ovipneumoniae* and *M arginini*[12].

Experimental reproduction of the disease with pure cultures of *M ovipneumoniae* have shown that it is capable of causing a mild interstitial pneumonia and mild clinical signs, generally only observable by auscultation and not producing overt signs[13][14][5]. Other bacteria, which are incapable of causing pneumonia alone, subsequently infect the lungs and cause a more severe inflammatory response and clinical signs, including coughing and nasal discharge.

The natural history of infection with mycoplasmas appears to be as follows; in flocks in which *Mycoplasma ovipneumoniae* is endemic, lambs become infected within a few days of birth from ewes which are carriers. The disease is slowly progressive over a period of weeks and, at ages over 5 to 10 weeks, secondary bacterial infection occurs. The pneumonia then becomes more severe. Some lambs recover over the following few weeks but clinical signs in some lambs persist up to 6 months of age or more. The extra demand placed on the lungs for heat exchange by high atmospheric temperatures may be a precipitating
factor in the exacerbation of a mild condition and the appearance of obvious clinical signs in summer[9][13].

**Viral pneumonia**

Parainfluenza 3 virus is the only virus which has been isolated from the lungs of sheep with pneumonia[15]. In 1968 the prevalence of flocks in Australia with antibody to PI3 was estimated to be 87%[16].

The virus is capable of producing mild pneumonic lesions but, without secondary bacterial infection, is unlikely to produce pneumonia with observed clinical signs. It is commonly linked to outbreaks of pneumonia in Australian sheep, either by viral isolation or by serological evidence, but that association does not imply causation[15]. It is clear that prior PI3 infection is not necessary for the development of pneumonia because cases of pneumonia associated with *Mycoplasma* spp occur in animals serologically negative for PI3 or in animals that become infected with PI3 months after pneumonia commenced in the flock[11].

PI3 virus apparently can have a role in the development of pneumonic pasteurellosis. Experimentally, *P haemolytica* will proliferate in the lung following infection with PI3 to give rise to an acute exudative bronchopneumonia and pleurisy whereas it is usually cleared rapidly from lungs not infected with virus[17][18]. A vaccine specific for PI3 was used in field trials in New Zealand and reduced the prevalence of pneumonia in naturally exposed lambs but did not prevent all cases of pneumonia[19]. This indicates that agents other than PI3 are responsible for initiating at least some natural cases of pneumonia in lambs.

**Pneumonia caused by Bordetella parapertussis**

In one small study in New Zealand this bacterium was found to be present in the nasal cavity and bronchial washings of a high proportion of 6 to 10 month old lambs with lesions of CNP, but a low proportion of lambs without lung lesions[1]. On experimental intratracheal inoculation of colostrum-deprived one week old lambs, lesions resembling those of early naturally occurring CNP were observed. This organism was considered to be capable of initiating a mild and short-lived respiratory infection which, with subsequent secondary bacterial infection, could develop to CNP[20].

**Bacterial pneumonia**

Bacteria such as *Pasteurella haemolytica*, *P multocida*, *Actinobacillus lignierisii*, *Actinomyces pyogenes*, *Corynebacterium equi*, Klebsiella spp, *Escherichia coli*, *Fusobacterium necrophorum* and *Neisseria* spp have all been recorded from the lungs of sheep with pneumonia[2]. These bacteria act as secondary invaders but they are important in increasing the severity of the clinical and pathological signs of pneumonia. They effectively render sub-clinical pneumonia, induced by *Mycoplasma* spp or PI3, a clinical entity such as *summer pneumonia* or CNP. Without them the effect on productivity or mortality rates of the milder pneumonias would be much less significant.

*Pasteurella* spp (particularly *P haemolytica*) are also responsible for the most severe and acute forms of ovine pneumonia. The disease is an acute exudative pneumonia with septicaemia and very high mortality rates. Difficulties in reproducing the disease experimentally with pure cultures of *Pasteurella* spp suggest that a previous infection with a virus or mycoplasma is usual in field cases.

*Histophilus ovis* has been associated with embolic pneumonic infection, characterised histologically by a necrotising vasculitis and grossly by abscession.

**Chlamydial pneumonia**

Chlamydia have been considered a cause of pneumonia in sheep in Europe and North America and were initially suspected of contributing to ovine pneumonia in Australia because the organisms have been identified in faeces of pneumonic and healthy sheep[2]. Subsequent investigations have failed to identify them in cases of pneumonia here or in New Zealand and they are largely discounted as lung pathogens in
Effects on productivity

Graziers and research workers observing flocks with enzootic pneumonia have noted the inferior growth rate of lambs affected by pneumonia\(^{(11)}\). There has been no quantification of this loss in Australia and no studies of the effect of pneumonia on wool growth rates recorded in any country. One New Zealand study has estimated that moderate to severe pneumonia is responsible for losses in carcass weight of 1.5kg and a reduction in liveweight gain of 0.8 to 0.9 kg per month for every 10% of the lung surface grossly affected by pneumonia\[^{[21]}\]. An earlier and extensive study of lambs slaughtered from a flock with no evidence of clinical pneumonia found that 60% of the lambs slaughtered had lesions of pneumonia. Only 6.5% of lambs had moderate to severe lesions and the deleterious effect on carcass weight of such lesions was estimated to be 0.45kg\[^{[22]}\]\[^{[23]}\]. The economic consequence in the whole flock of this loss of carcass weight of lambs is approximately 0.2% only and, therefore, quite insignificant. This study has been criticised for underestimating the degree of weight loss and the prevalence of moderate to severe lesions\[^{[24]}\] but it remains the only attempt to put an economic cost on CNP of lambs in Australasia.

The disease in most flocks appears to be clinically mild or sub-clinical and self-limiting. Most lambs recover within the first year of life. In line with many other interruptions to weight gain in early life, compensatory gain in liveweight may afford a 'catch-up' within a few months of recovery. The loss of wool production, a more important source of economic loss in Merinos than weight gain, is probably not recovered but has not been measured. Increased mortalities, both from pneumonia and from increased susceptibility to other diseases are likely to be additional and significant sources of economic loss in some flocks.

Acute pasteurellosis

Pasteurellosis is regarded as the most important bacterial disease of sheep in many countries, particularly those in which sheep-rearing is practised intensively. It is of much less importance in countries where grazing conditions are extensive, as in Australia and New Zealand. In these countries, outbreaks are uncommon, sporadic and usually have a low morbidity.

\textit{Pasteurella haemolytica} type A exists in 13 different serotypes (1 - 17 except 3, 4, 10 and 15) and \textit{P trehalosi}, formerly \textit{P haemolytica} type T, exists in four serotypes (3, 4, 10 and 15). The differences are important in vaccine considerations. In the UK, \textit{P haemolytica} causes pneumonic pasteurellosis in cattle (mainly serotype A\(_1\)), pneumonia in goats and sheep (mainly A\(_2\) but, in decreasing order of frequency, A\(_6\), A\(_7\), A\(_9\) and A\(_1\)). \textit{P trehalosi} causes systemic pasteurellosis in sheep only. Sheep strains of \textit{P haemolytica} appear to be different from cattle strains even within serotypes, principally in the way the organism is able to use host haemopexin as a source of iron. \textit{P haemolytica} and \textit{P trehalosi} both occur in the nasopharynx of apparently normal sheep.

\textit{Pasteurella multocida} is an uncommon pathogen in sheep, except in tropical countries\[^{[25]}\].

There are two important epidemiological associations with acute pasteurella pneumonias. One is the probable initiating role of PI3 virus in most cases; the other is that with pasteurella mastitis in ewes. Ewes with pasteurella mastitis commonly infect their lambs and infected lambs may induce pasteurella mastitis in their dams.

Clinical signs

Outbreaks of acute pneumonic pasteurellosis often commence with sudden deaths before clinical signs are observed. As an outbreak proceeds, respiratory signs become more apparent, particularly in older sheep rather than in lambs. Signs then include dullness, anorexia, fever, dyspnoea or hyperpnoea. On auscultation, respiratory sounds are loud and prolonged. Affected sheep froth at the mouth, cough and have a serous nasal discharge. In acute cases, death occurs in 1 to 3 days.
Necropsy findings

The findings at necropsy vary with the chronicity of the condition before death. In acute cases, the lungs are swollen and heavy with bright purple-red patches which are solid and exude a frothy haemorrhagic fluid when incised. Consolidation is evident and some areas may contain greenish-brown areas of necrosis surrounded by dark, haemorrhagic zones. In less acute cases, areas of the cranial lobes are greyish-pink, raised and consolidated. On cutting, the tissue is dense and the septa thickened and prominent. The bronchial lymph nodes are enlarged and may have petechial haemorrhages[25].

Diagnosis

Generally, the only other 'outbreaks' of acute pneumonia in sheep in Australia are associated with aspiration pneumonia, following careless drenching or dipping. History is important in connecting clinical signs of pneumonia to some recent management event, but pasteurellosis is often precipitated by stressful events as well, particularly in young sheep. The post-mortem diagnosis of acute pneumonia must be made with caution in animals which have died naturally because post-mortem change in the lungs and following some clostridial diseases can grossly resemble acute pasteurella pneumonia. Histopathology and bacteriology of freshly dead specimens are necessary to confirm the diagnosis.

Treatment and control

Vaccination is practised successfully in a number of countries, including the UK. One treatment with long-acting oxytetracycline intramuscularly at 20mg/kg (Terramycin LA, Pfizer, at 1 ml/10kg) is effective in controlling the development of pneumatic pasteurellosis for 4 days in animals with early clinical signs or animals which are post-exposure but pre-clinical[26]. Retreatment after 3 to 4 days is advisable because relapses occur. Feeding of broad-spectrum antibiotics, especially tetracyclines, to feedlot lambs is often done for recently weaned lambs, in an effort to reduce the incidence of acute pasteurellosis.

Tuberculosis

The disease is rare in sheep in Australia. It can be caused by M bovis and M avium[27][28].

Melioidosis

Melioidosis is caused by Pseudomonas pseudomallei. It occurs in Australia but outbreaks are restricted to tropical zones. The chief source of infection are rodents, which develop protracted infections during which organisms are excreted in the faeces. after the 'wet' season in northern Australia, climatic conditions are conducive to the growth of the organism and it can be found in surface soil and water[29]. Outbreaks occur in all farm animals, particularly sheep. Infection occurs through ingestion, insect bites, abrasions and inhalation. The disease is highly fatal in humans.

Clinical signs in sheep include lameness, respiratory distress, weakness and recumbency and death in 1 to 7 days. Chronic forms of the disease are more common in goats, which can be infected without showing clinical signs. At necropsy, multiple abscesses are found in most organs, including lungs, spleen, liver, subcutaneous tissues and lymph nodes, particularly nodes of the thoracic cavity. The pus in the abscesses resembles that of CLA.

Control of the disease involves the elimination of infected farm animals, reduction of rodent infestations and disinfection of premises.

Parasitic pneumonia

Dictyocaulus filaria is the only significant lungworm of sheep in Australia. Adult parasites live freely in the bronchi and the exudative bronchitis they induce can lead to bronchiolar obstruction and areas of atelectasis. Rarely are the affected areas extensive so dyspnoea is not evident but coughing is the predominant clinical
sign. Occasionally, very heavy infestations occur and clinical signs of dyspnoea, moist rales on auscultation and even death result. Additionally, secondary bacterial infections can exacerbate the verminous pulmonary damage and the clinical signs.

*Protostrongylus rufescens* causes similar clinical signs but infestations are usually light and lung lesions only ever significant in lambs.

*Muellerius capillaris* adults live in fibrous nodules in the interstitial tissue of the lungs and rarely cause any clinical signs or deleterious effects. (In goats, nodule formation is not a characteristic and an interstitial pneumonia can develop.)[30]

**Aspiration pneumonia**

In sheep, most cases of aspiration pneumonia occur from careless or improper drenching technique or from prolonged submersion in plunge dips - either through weakness of the sheep, crowding or overzealous dunking by the operator. Isolated cases of aspiration pneumonia also occur following vomition and inhalation of rumen contents, paralysis of larynx, pharynx or oesophagus, or rupture of a pulmonary or pharyngeal abscess.

Clinical signs vary with the dose and nature of the aspirant. Significant amounts of infective material can lead to acute, severe pneumonia and toxaemia with death in 1 or 2 days. Lesser amounts or less noxious material may lead to chronic pneumatic lesions, pulmonary abscessation, pleurisy and a prolonged period of ill health. Gangrenous pneumonia occurs sometimes following aspiration and is marked by a putrid odour on the breath.

**Sheep pulmonary adenomatosis (SPA, Jaagsiekte)**

This is a chronic progressive pneumonia of sheep marked histologically by adenomatous ingrowths of the alveolar walls and clinically by the production of a profuse watery mucus from the lungs which is discharged from the nose. It is caused by a retrovirus and is one of the three 'slow virus' infections of sheep with maedi-visna and scrapie. The disease occurs in Britain, continental Europe, South Africa, Israel, Asia and Iceland.

**Epidemiology**

Only sheep are infected in natural cases. Transmission is presumed to occur by inhalation of infected droplets and by vertical transmission to the foetus. Close housing in winter, such as occurs routinely in Iceland, promotes transmission but is not essential for flock outbreaks. The incubation period is 1 to 3 years.

**Clinical signs**

The adenomatous ingrowths encroach on the alveolar space and lead to anoxic anoxia. Coughing occurs but is not a prominent sign. Emaciation, dyspnoea and panting after exercise, profuse watery discharge from the nose are characteristic signs. Moist rales are audible over affected areas of lung. The disease is inevitably fatal.

**Necropsy**

The lungs are enlarged, heavy, consolidated and there is frothy fluid in the bronchi. Histopathology is characteristic.

**Treatment and control**

There is no treatment. A vaccine is used successfully in Kenya.
Maedi

Maedi-visna is a chronic, progressive viral infection characterised by a prolonged incubation period and predominantly two clinical manifestations; pneumonia (Maedi means dyspnoea) and encephalomyelitis (Visna means wasting in Icelandic). The virus also infects the udder causing a chronic mastitis and reduced milk yield. Generally, only one form of the disease occurs in one animal and often one form predominates in any one flock. The disease does occur in goats but transmission between sheep and goats does not usually occur in field cases.

The disease occurs widely throughout the world but does not occur in Australia or New Zealand. It was inadvertently introduced into Iceland in 1933, and, before the disease was eradicated in 1965 by a slaughter and restocking program, 650,000 sheep had been slaughtered and over 100,000 sheep had died of the disease.

Epidemiology

Vertical transmission, by the excretion of virus-infected leucocytes in colostrum and milk, is the main form of spread of Maedi-visna in flocks. Transmission to the foetus in utero occurs but is rare. It is believed that transmission via ova or sperm does not occur. Horizontal transmission occurs, chiefly by the inhalation of infected respiratory secretions from infected sheep. Infection is effectively always introduced into clean flocks by horizontal transmission from an introduced, infected sheep[31]. The incubation period is very long; often 2, 3 or 4 years or more. The virus does not survive for more than two weeks outside the host.

Pathogenesis

Following infection, viral replication is restricted for a prolonged period, during which the viral genetic material resides in infected cells as proviral DNA. The infection progresses and clinical signs are associated with the chronic progressive proliferation of lymphoid tissue in lungs, brain, udder and joints. The pulmonary lesions are effectively an interstitial pneumonia with a restriction of the alveolar space leading to anoxia. Many sheep remain in a sub-clinical phase of the disease. The variation in expression of the disease as primarily respiratory, nervous or mastitic is presumed due to different tissue tropism of the strains of the virus, differences between breeds and flocks of sheep in their susceptibility and the effects of different management factors.

Two characteristics of the disease are particularly important in the diagnosis and the epidemiology of the disease. Firstly, infected animals may not develop any detectable antibodies against the infection, or may take months or years to do so. These animals, which may or may not be presenting clinical abnormalities, are still capable of infecting other animals. Secondly, even in cases where the animals do mount an immunological response, the disease is progressive and virus multiplication continues.

Clinical signs

Animals with maedi are listless, emaciated and dyspnoeic. Respirations are laboured and rapid; 80 per minute or higher. There is coughing and nasal discharge but most affected sheep retain their appetite. Udder induration, hind-limb paralysis and, in some cases, swollen joints with or without lameness, may also be present in the flock. Clinical signs last for 3 to 12 months but the disease is inevitably fatal.

Diagnosis

Sheep pulmonary adenomatosis (Jaagsiekte) can produce similar clinical signs with similar flock history and, in some countries, can be simultaneously present in the same flock and the same sheep. Pulmonary adenomatosis is characterised by a profuse nasal discharge and a shorter clinical course. The two diseases can be readily differentiated histopathologically. Parasitic pneumonia and melioidosis also have signs of chronic respiratory disease.
Clinical pathology can aid in diagnosis, either by one of a number of techniques for virus identification or by serology. Serology is used as a flock diagnosis but negative serology in individual cases is not reliable evidence of freedom from infection. The time between infection and sero-conversion is variable and may be as long as one or more years. Some infected animals remain seronegative.

**Control**

There is no treatment for maedi-visna. National control programs in endemically infected countries vary in their approach but are based largely on the identification of clean flocks by serological testing; separation of lambs from ewes and artificial rearing of lambs in flocks with low prevalence of infection or complete destocking and replacement in flocks with moderate or higher levels of infection.

**Recommended reading**

St George TD (1972) *Investigations of respiratory disease of sheep in Australia* Aust Vet J 48 p 318

Sullivan ND, St George TD and Horsfall N (1973) *A proliferative interstitial pneumonia of sheep associated with mycoplasma infection 1. Natural history of the disease in a flock* Aust Vet J 49 p 57

Sullivan ND, St George TD and Horsfall N (1973) *A proliferative interstitial pneumonia of sheep associated with mycoplasma infection 2. The experimental exposure of young lambs to infection* Aust Vet J 49 p 63


[2] St George TD and Sullivan ND (1973) *Pneumonias of Sheep in Australia* The University of Sydney Post-Graduate Foundation in Veterinary Science, Sydney, Veterinary Review No 13


[15] St George TD (1969) *The isolation of Myxovirus parainfluenza* type 3 *from sheep in Australia*

[16] St George TD (1971) *A survey of sheep throughout Australia for antibody to Parainfluenza type 3 virus and to Mucosal disease virus* Aust Vet J 47 p 370


[27] Barton and Acland HM (1973) *Mycobacterium avium* serotype 2 infection in a sheep* Aust Vet J 49 p 212

