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AN INVESTIGATION OF OVINE PNEUMONIA IN FOUR HERDS FROM CENTRAL NORWAY

II. RELATION BETWEEN PATHOMORPHOLOGY AND PRESENCE OF MICRO-ORGANISMS *

By

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NØSTVOLD, S. and T. BAKKE: *An investigation of ovine pneumonia in four herds from Central Norway. II. Relation between pathomorphology and presence of micro-organisms.* Acta vet. scand. 1982, *23*, 259—274. — Sheep lungs obtained at slaughter from 4 herds from Central Norway were subjected to a macroscopical examination. Gross pulmonary findings included normal lungs, fibrous pleurisy, verminous nodules, atelectasis and consolidations of lobular and lobar extensions. A selection of lungs representing the various gross findings was subjected to microbiological and histological examinations. Pleurisy was associated wtih pneumonic consolidations and extension of lesions. Verminous nodules indicative of Muellerius spp. migrations occurred irrespective of gross pulmonary lesions or microbiological findings. A subacute or chronic pneumonia of a mixed proliferative exudative type was demonstrated in consolidated tissue. An association was found between the isolation of Mycoplasma ovipneumoniae and the presence of pneumonic lesions and between the number of organisms and extension of lesions. Histological findings highly indicative of this organism were proliferative changes in the bronchiolar and bronchial epithelium and intrapulmonary lymphoid tissue. The intensity of these changes was correlated to the number of mycoplasma organisms present. Mycoplasma ovipneumoniae is suggested to be of primary etiological significance regarding subacute or chronic pneumonia in lambs at slaughter.

ovine pneumonia; Mycoplasma ovipneumoniae; gross and histological lesions.

In an earlier study of ovine pneumonia comprising numerous herds from Southern Norway (*Bakke* 1982), Mycoplasma ovipneumoniae was regularly recovered and incriminated to be of

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etiological significance regarding subacute or chronic pneumonia in lambs at slaughter.

A second investigation was designed to study the occurrence of possible respiratory pathogens and the prevalence of pneumonia within single herds (*Bakke & Nøstvold* 1982).

Encouraged by reports of both spontaneous and experimental mycoplasma pneumonia, claiming certain histopathological features to be highly indicative of this organism (*Sullivan et al.* 1973 a, b, *Foggie et al.* 1976, *Gilmour et al.* 1979, *Alley and Clarke* 1979), microbiological samplings were followed by histological examination.

The main purpose of the histological investigation was to study the validity of certain pathomorphological features in the diagnosis of ovine pneumonia in Norway with respect to possible etiologies.

MATERIAL AND METHODS

Information regarding description of material and microbiological and histological procedures is given in a preceding paper (*Bakke & Nøstvold* 1982).

Histological changes were evaluated in a semiquantitative way. A set of histological criteria was recorded as being absent, very mild, mild, moderate or severe. Sections were scored negative for the various traits if the changes were absent or very mild, and positive if the changes were mild, moderate or severe.

RESULTS

Gross pathological findings

Gross changes indicative of pneumonia consisted of small areas of lung collapse (atelectasis), true consolidations of a minor sublobar extension (lobular pneumonia), and consolidations covering an entire lobe or several lobes (lobar pneumonia).

Areas of collapse were most frequently seen in the anterioventral parts of the lung. They were often wedge-shaped with the base extending towards the margins or emerged as shallow depressed grooves. Atelectatic areas were soft in consistency, dull red in colour with a homogenous cut surface and smooth pleural lining. They were minor in extension but often several in number in each lung.

True consolidations distinguished themselves from atelectatic areas by extension, a grey to reddish-grey colour, a rather firm consistency and a finely granular mottled appearance. On the cut surface some mucoid exudate often oozed out from larger air passages. Consolidations were sharply delineated from normal tissue and occurred in the apical, cardiac and intermediate lobes. In a few instances the anterio-ventral parts of the diaphragmatic lobes were also affected. There was a tendency for the more extensive lesions to be firmer in consistency and pale grey.

Pleurisy was grossly evident in both pneumonic and normal lungs. It was seen in the chronic healed stage. The predilection sites for pleurisy seemed to follow that of pneumonia, except being more frequently noticed on the anterior aspects of the diaphragmatic lobes, probably dispersed from neighbouring lobes.

Macroscopical lesions indicative of lungworm migrations (*Jubb & Kennedy* 1970) were seen as small (< 1 cm) raised nodules located in the subpleural tissue. None of the lungs were heavily invaded. Only a small number of lungs contained more than 3 lungworm nodules.

Histological findings

Histology of lungs with gross atelectasis revealed either alveolar tissue collapse only, or also mild inflammation. The inflammatory changes were more of a proliferative than exudative type.

The histomorphological picture of true consolidations (lobular and lobar pneumonia) reflected a rather low-grade subacute to chronic infection with a mixed exudative proliferative inflammatory reaction (Fig. 1). There was evidence of a strong antigenic stimulation with peribronchiolar and peribronchial lymphoid proliferations (Fig. 2). These lymphoid proliferations were mostly of a follicular type and often encircled the air passages ("lymphoid cuffings") (Fig. 3). There were indications of chronic, largely non-destructive irritation of the air-way epithelium in form of bronchiolar and bronchial epithelial hyperplasia and some desquamation (Fig. 4). Exudative changes consisted of an increased amount of inflammatory cells in the alveolar space, the lumen of bronchioles and the peripheral bronchial branchings in decreasing order. The exudate was usually of a mixed type, the major part of the cells being mononucleated, mainly alveolar

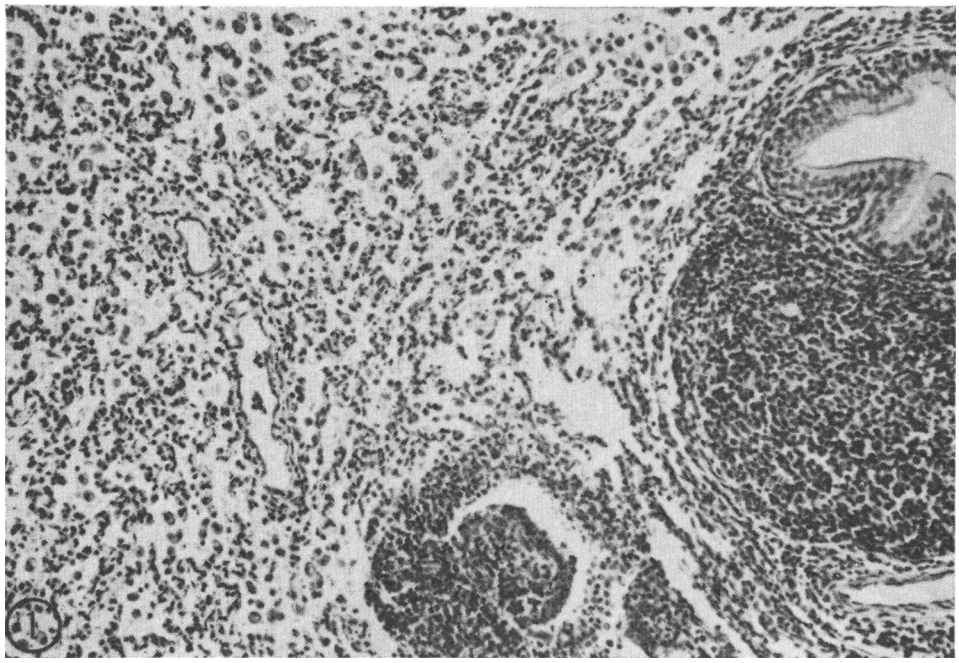

Figure 1. Proliferative exudative pneumonic changes showing epithelial and lymphoid hyperplasia together with increased numbers of mononucleated cells in the alveolar space. HE × 170.

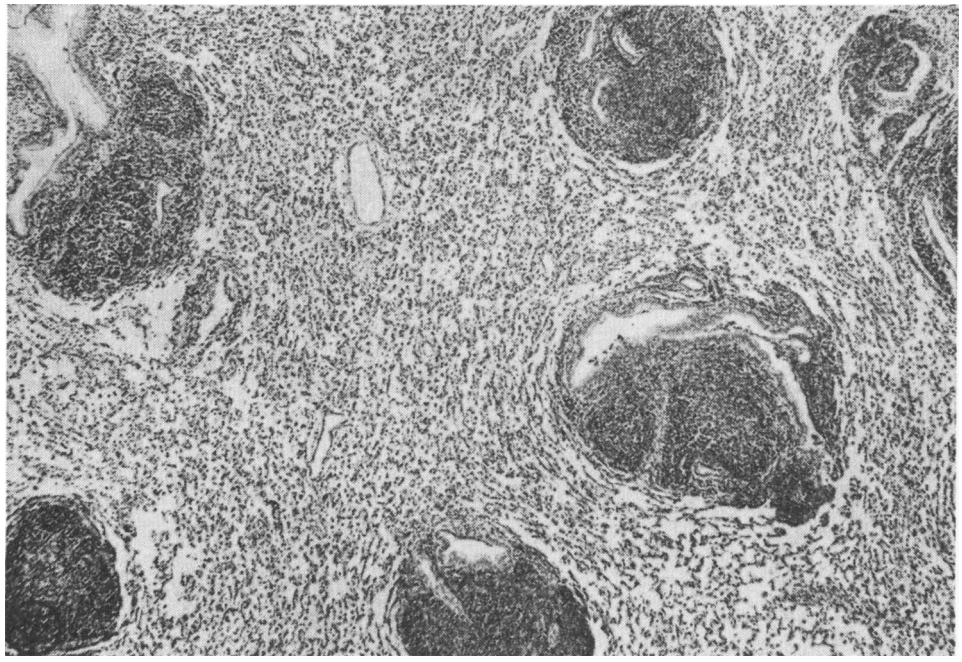

Figure 2. Section demonstrating follicular lymphoid hyperplasia neighbouring the smaller air passages and partly compressing their lumina. Slight alveolar collapse is evident. HE × 68.

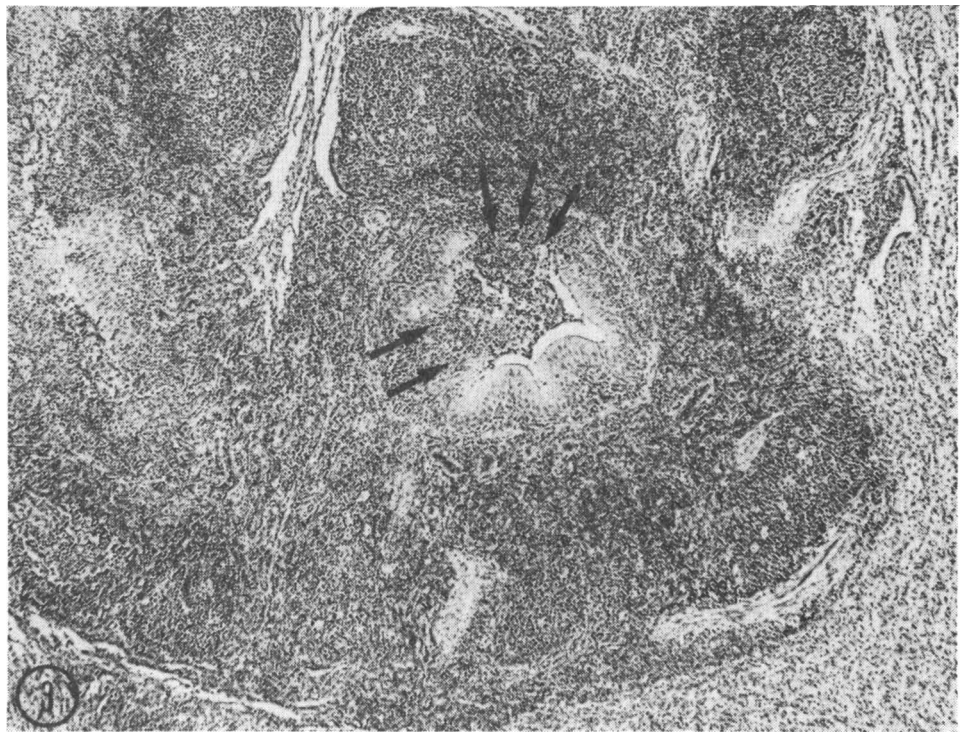

Figure 3. Extensive peribronchial lymphoid hyperplasia ("cuffing pneumonia") encircling the air passages. Focal destructions of the air-way epithelium with massive infiltration of lymphoid cells are evident (arrows). HE × 68.

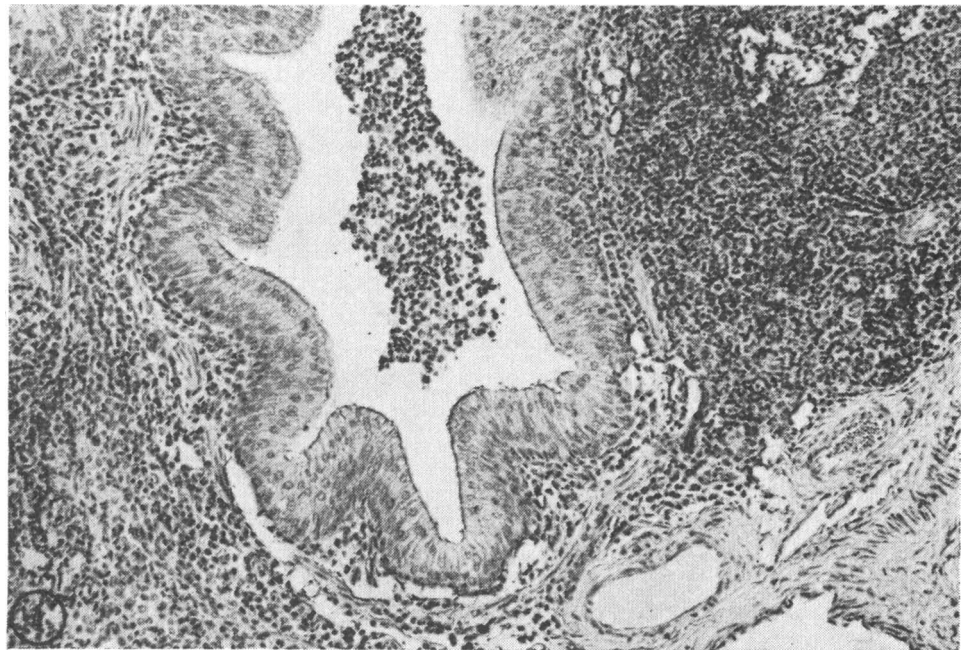

Figure 4. Section from consolidated tissue demonstrating bronchial epithelial hyperplasia, mixed cellular exudate in bronchial lumen and peribronchial lymphoid hyperplasia. HE × 170.

macrophages. Neutrophils were usually scattered among the mononucleated cells in the alveolar space. In the lumen of the terminal air-ways, however, they often formed pure aggregates ("neutrophilic plugs"). Proliferation of type II pneumocytes was regularly seen. They were often shed into the alveolar lumen and therefore difficult to distinguish from macrophages. Interalveolar septa and peribronchiolar interstitial tissue regularly demonstrated proliferative thickening, infiltration with mononuclear cells, some collagen formation (peribronchiolar "hyaline scars") but little evidence of myoproliferation.

The histomorphological changes described were largely lobular in distribution and seemed to originate from the surroundings of terminal bronchioles.

Histology of lungworm nodules revealed central necrotic masses enveloped by a granulomatous inflammatory reaction with a large number of eosinophils and some collagen formation. Parasites or bacteria were not encountered.

The relation between pathomorphological changes and microbiological findings

P l e u r i s y. The prevalence of pleurisy among the total number of lungs examined at the slaughter-line was 10 %. Of both 188 lungs without macroscopical changes and 74 lungs with gross atelectasis 5 % were affected. The corresponding figures for lungs exhibiting lobular (26) and lobar (12) consolidations were 26 and 75 %, respectively. Of the 86 lungs being selected for further microbiological and histological investigation, 19 (22 %) demonstrated pleuritic changes, all in association with pneumonic lesions. M. ovipneumoniae was recovered from all the pleuritic lungs, for 12 of them in combination with bacteria.

V e r m i n o u s n o d u l e s. Gross verminous lesions were more frequently encountered in normal than in pneumonic lungs, the difference, however, not being statistically significant. The anatomical distribution of verminous nodules were not associated with either pleuritic changes or pneumonic consolidations.

P n e u m o n i c l e s i o n s. In 26 out of 39 (67 %) lungs scored as normal by macroscopical and histological evaluations no micro-organisms were demonstrated (Table 1). The remaining third was almost equally divided into one group (15 %) with

Table 1. Microbiological findings in relation to patho-anatomical lesions.

Pulmonary lesions	Number of lungs (% in brackets) with isolation of				Total
	Mycoplasmas & bacteria	Mycoplasmas only	Bacteria only	Micro-organisms not isolated	
None	1	3	2	19	25
Atelectasis (histo —) *	4	3	0	7	14
All healthy lungs	5 (13)	6 (15)	2 (5)	26 (67)	39 (100)
Atelectasis (histo +) **	3	2	0	1	6
Lobular pneumonia	11	18	0	0	29
Lobar pneumonia	9	3	0	0	12
All pneumonic lungs	23 (49)	23 (49)	0 (0)	1 (2)	47 (100)

* Atelectasis (histo —): gross atelectasis without histological evidence of inflammation.

** Atelectasis (histo +): gross atelectasis with histological evidence of inflammation.

isolation of mycoplasmas only, and one group (13 %) with isolation of both mycoplasmas and bacteria. In lungs scored as pneumonic (47) mycoplasmas were almost constantly isolated (46), in half of the instances (23) bacteria were also demonstrated.

The relative occurrence and number of mycoplasma organisms in relation to sampling location of lungs exhibiting various macroscopical lesions are shown in Table 2. In lungs without macroscopical lesions, mycoplasmas were rather infrequently recovered, and solely with a low titre. Comparing the different sampling locations, the organism was most frequently isolated from the right apical lobe. This was also the predilection site for pneumonic consolidations. Among lungs exhibiting macroscopical atelectasis, the highest numbers of organisms were found in lungs with histological evidence of inflammation (not shown in table). In the two pneumonic groups (lobular and lobar pneu-

Table 2. Mycoplasma recovery rates and titres in relation to sampling locations and gross pulmonary lesions.

Gross pulmonary lesions	Sampling locations	Positive mycoplasma recoveries / total number examined	Mycoplasma titre* $\geq 10^7$	10^6	$\leq 10^5$
None	Consolidated tissue (C)**	4/25			4
	Diaphragmatic lobe (D)	1/25			1
Atelectasis	C***	12/20		2	10
	D	9/20		1	8
Lobular pneumonia	C	29/29	9	10	10
	D	23/29		2	21
Lobar pneumonia	C	12/12	6	2	4
	D	11/12		1	10

* organisms/g lung tissue; calculated from the highest dilution from which Mycoplasma ovipneumoniae could be cultured.
** in normal lungs from the right apical lobe.
*** includes both true atelectasis and minor consolidations judged histologically.

monia) mycoplasmas were constantly recovered from consolidated tissue. The corresponding recoveries for samplings from apparently normal tissue of the right diaphragmatic lobe were 79 and 92 %, respectively. The mycoplasma titres from consolidated tissue were usually very high ($\geq 10^6$), while samplings from the diaphragmatic lobe regularly displayed a lower titre ($\leq 10^5$).

Samplings from the immediate vicinity of pneumonic consolidations demonstrated practically the same recovery rates for mycoplasma as consolidated tissue. The titres were, however, generally lower (not shown in table).

In pneumonic lungs with a positive recovery of bacteria from consolidated tissue, the organism was additionally isolated from the diaphragmatic lobe in 52 % of the cases (not shown in table).

As shown in Table 3 exudative changes were virtually absent in non-consolidated tissue both in normal and pneumonic lungs. Exudation occurred, however, with some regularity in the vicinity to true consolidations. Interstitial septal thickening was almost constantly recorded in the anterior lobes irrespective of gross lesions. It was less frequent in the diaphragmatic lobe,

Table 3. The relative occurrence of some histological features in relation to sampling location asnd gross pulmonary lesions.

Gross pulmonary lesions	Sampling locations	% positive scores for some histological features			
		Exudation	Interstitial septal thickening	Bronchiolar epithelial hyperplasia	Lymphoid hyperplasia
None	C*	0	88	17	8
	N**	0	75	13	17
	D***	0	33	4	8
Atelectasis	C****	10	72	34	41
	N	9	73	18	73
	D	0	10	5	10
Lobular pneumonia	C	86	61	96	96
	N	13	60	57	70
	D	0	41	38	31
Lobar pneumonia	C	92	83	100	83
	N	58	100	83	75
	D	0	50	50	8

* C consolidated tissue (in normal lungs from the right apical lobe).
** N neighbouring tissue to consolidations (in normal lungs from the left cardiac lobe).
*** D grossly normal tissue of the right diaphragmatic lobe.
**** C includes both true atelectasis and minor consolidations judged histologically.

where macroscopical changes were totally absent. Hyperplasia of the bronchiolar and bronchial epithelium and intra-pulmonary lymphoid tissue was almost consistent features of consolidated tissue. These changes, however, were not solely restricted to pneumonic areas nor to pneumonic lungs only.

As seen from Fig. 5 polymorphonucleated leucocytes (PNL) and increased numbers of macrophages were almost absent in lungs with negative microbiological findings. A pure mycoplasma infection demonstrated the same frequency of positive PNL-scores as a combined infection, while having a somewhat lower prevalence of positive scores for alveolar macrophages. However, a stronger intensity of the PNL-reaction (scored "severe") seemed to be associated with the presence of bacteria (not shown in table), but not exclusively so. Proliferative changes of the bronchiolar epithelium and intra-pulmonary lymphoid tissue were very frequently given positive scores in lungs with positive

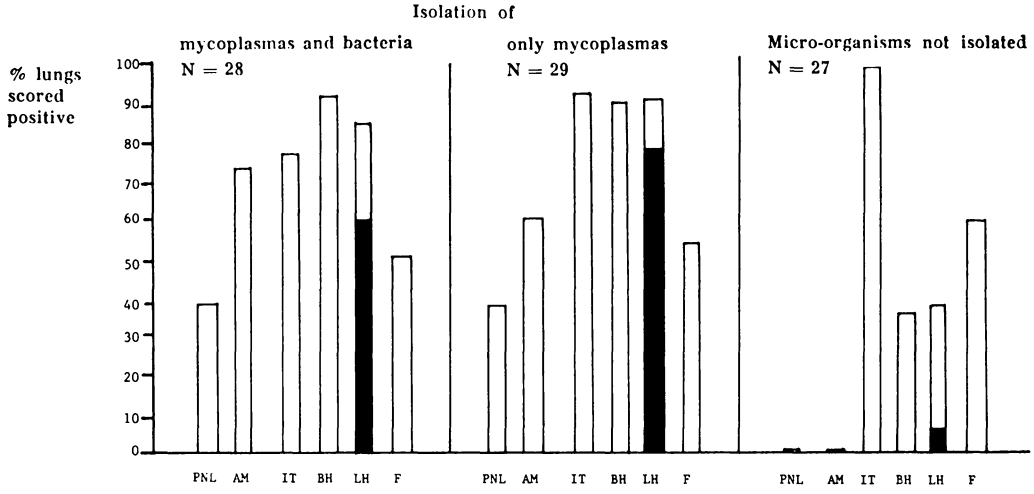

Figure 5. Prevalence of positive scores for some histological changes in relation to microbiological findings. *

N = number of lungs within each recovery group
PNL = polymorphonucleated leucocytes } in the alveolar space
AM = alveolar macrophages }
IT = interstitial septal thickening
BH = bronchiolar epithelial hyperplasia
LH = peribronchiolar lymphoid hyperplasia
F = interstitial fibroses
■ lungs scored "severe", i.e. "cuffing pneumonia"
* consolidations; in normal lungs the right apical lobe

microbiological findings irrespective of whether pure mycoplasma or combined infections were demonstrated (90 versus 40 % in "sterile" lungs). Interstitial septal thickening was almost constantly present regardless of microbiological findings. Slight interstitial fibrosis occurred in approximately half of the lungs of all 3 recovery groups.

When grading the intensity of lymphoid hyperplasia, "cuffing pneumonia" was almost consistently associated with the presence of mycoplasmas. Of 39 lungs with "cuffing pneumonia", 38 were positive for mycoplasmas, either alone (23) or mixed with bacteria (15). Extensive lymphoid hyperplasia was encountered in lungs exhibiting macroscopical atelectasis, consolidations of minor (lobular) or major (lobar) extension both in consolidated and closely neighbouring tissue. It was never seen in grossly normal lungs.

DISCUSSION

The spectrum of gross lesions found in the present investigation is in accordance with reports from similar surveys of lungs obtained from lambs at slaughter from other countries (*Sullivan et al.* 1973a, *Alley* 1975, *Kirton et al.* 1976, *Jones et al.* 1979). Progressive pneumonia (Maedi) or "enzootic pneumonia" of the acute pasteurella type (*Gilmour* 1978) were not encountered.

Pleurisy of the type described in this material is generally thought to be the result of a previous pneumonia and not a primary serositis (*Jubb & Kennedy* 1970, *McGowan et al.* 1978). Our findings strengthen this concept as pleuritic changes had the same predilection sites as pneumonia, and were most frequent in lungs exhibiting pneumonic lesions. There was also an association between the extension of pneumonic lesions and the occurrence of pleurisy. In lungs possessing the major extensions (lobar pneumonia) bacteria were most frequently isolated, indicating these organisms to be of etiological significance for the establishment of the pleuritic lesions. The presence of pleuritic changes in "sterile" lungs does not exclude the participation of such bacterial agents at an earlier stage. Pleuritic exudation in sheep is rarely resorbed before a fibrosing stage is reached, possibly due to a low fibrinolytical activity of the mesothelial lining (*McSporran & Russel* 1978). Reabsorption of intra-pulmonary pneumonic lesions might therefore leave fibrous pleurisy on an otherwise normal lung. Pleurisy is economically important due to time consuming manual trimming of the chest wall, high condemnation rates (*Alley* 1975) and trade restrictions of such carcasses in many countries (*Dysart* 1976).

The gross and histological picture of verminous nodules seen in this study indicates earlier migrations of Muellerius spp., which is moderately prevalent among sheep in Norway (*Helle* personal communication 1981). The lack of association between the occurrence of gross verminous lesions and either pneumonia, pleurisy or microbiological recoveries indicates a minor clinical significance of low-grade Muellerius invasions (*Soulsby* 1968).

The histomorphological features of pneumonia in the present investigation closely resembled descriptions of subacute or chronic non-progressive ovine pneumonias from other countries and possessing a number of designations: i.e. "atypical pneumonia" (*Stamp & Nisbet* 1963), "enzootic pneumonia" (*St. George &*

Sullivan 1973), "proliferative exudative pneumonia" (*Jones et al.* 1979, *Gilmour et al.* 1979) and "chronic non-progressive pneumonia" (*Alley and Clarke* 1979).

The close association between the occurrence of gross pneumonic lesions and the presence of mycoplasmas (Table 1), and the correlation between number of organisms and extension of lesions (Table 2) strongly indicate M. ovipneumoniae to be of primary etiological significance for subacute or chronic ovine pneumonia. This is in agreement with the conclusion of an earlier microbiological survey of ovine pneumonia in Norway (*Bakke* 1982) and is widely accepted abroad (*Sullivan et al.* 1973 a, b, *Jones et al.* 1979, *Alley & Clarke* 1980).

As found in the present and other studies, the organism may colonize the lung parenchyma without eliciting an inflammatory reaction (*Foggie et al.* 1976). This offers an explanation for the recovery of mycoplasmas from normal lungs (Table 1) and from non-consolidated tissue in pneumonic lungs (Table 2). The presence of mycoplasma organisms in normal tissue is probably an even more common feature in the upper respiratory tract in sheep of infected herds (*Jones et al.* 1979). However, isolation of mycoplasmas outside grossly pneumonic lesions was often associated with microscopical evidence of an inflammatory reaction. Outside macroscopical lesions, the various histological features were generally found to be less severe and more of a proliferative than exudative type. At these sites mycoplasma recoveries clearly demonstrated a lower titre. This indicates that the organism must be present in fairly high numbers before eliciting a proliferative exudative reaction strong enough to precipitate macroscopical consolidations, probably as a sequel to obstructive bronchiolar change (*St. George et al.* 1971).

The higher prevalence score for interstitial changes in "sterile" than in infected lungs (Fig. 5) merely reflects that minor changes are readily detected in well aerated tissue but easily lost in true consolidations. However, a high prevalence of interstitial changes has also been described in experimental infections with M. ovipneumoniae (*Sullivan et al.* 1973 b) and in extensive lesions of the natural disease (*Sullivan et al.* 1973 a, *Alley* 1975, *Alley & Clarke* 1979). To what extent some of the interstitial proliferative changes ("hyaline scars") observed were due to earlier larval migrations is unsettled, as low-graded invasions may not necessarily leave grossly evident remnant lesions. How-

ever, similar "hyaline scars" are reported in "atypical pneumonia" (*Jones et al.* 1978, *Gilmour et al.* 1979) with no reference to lung nematodes. It should be added that higher frequency of positive scores for interstitial thickening was demonstrated in the apical than in the diaphragmatic lobe in non-consolidated lungs (Table 3). These changes may therefore be remnant features of a mycoplasmal infection in its restituted phase (*McGowan et al.* 1978).

One of the most striking histological features of the present type of ovine pneumonia is the participation of the intra-pulmonary lymphoid system; still the influence of immune mechanisms in the pathogenesis of the disease is largely unrevealed. In the present histological survey, the proliferating lymphoid follicles of the pulmonary tissue contained few plasmoid or plasma cells. This probably indicates a cellular immune reaction being the major feature. Delayed hypersensitivity to endogenous lung antigens has been demonstrated in response to pulmonary tissue damage (*Cate & Burrel* 1974). A combined experimental infection with M. ovipneumoniae and Pasteurella haemolytica produced typical proliferative exudative pneumonia in conventionally reared and SPF lambs. In the latter instance, however, there was a marked absence of lymphoid hyperplasia and less extensive lesions. There are further reports that immunosuppressed laboratory animals challenged with their respective pathogenic mycoplasma spp. develop less severe lesions than controls (*Denny et al.* 1972, *Taylor-Robinson et al.* 1972). Whatever the nature of this lymphoid hyperplasia of pulmonary infections, an effect on extension of lesions is readily evidenced histologically. Proliferating lymphoid tissue is seen to compress the smaller air passages, which are narrowed even more by epithelial hyperplasia, leading to obliteration of the air-ways. To what extent various immune mechanisms should be credited for the clinical restitutions usually seen in this form of non-progressive pneumonia remains to be settled, as the organism probably somehow persists (*Jones et al.* 1979). However, colostral transfer of antimycoplasmal antibodies has been offered as one possible explanation for a rather low incidence of mycoplasma pneumonia in the early neonatal period (*Jones et al.* 1979).

Extensive intra-pulmonary lymphoid hyperplasia may be associated with a variety of pulmonary insults (*Jericho* 1977) and are not pathognomonic for any single agent. However, in the

present investigation these changes were strongly associated with the isolation of M. ovipneumoniae both in qualitative and quantitative terms. These findings strengthen the conclusion drawn previously (*Bakke* 1982), indicating M. ovipneumoniae to be of primary etiological significance regarding subacute or chronic ovine pneumonia in lambs at slaughter.

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SAMMENDRAG

En undersøkelse av ovin pneumoni i 4 besetninger fra midt-Norge. II. Relasjon mellom patomorfologiske forandringer og tilstedeværelsen av mikro-organismer.

Lunger fra samtlige dyr slaktet i 4 sauebesetninger ble undersøkt makroskopisk med henblikk på pneumoniske forandringer. De makroskopiske funn omfattet normale lunger, fibrøs pleuritt, lungeorm-knuter, atelektase samt pneumoniske fortetninger av lobulær og lobær utbredelse.

Et utvalg av lunger som representerte de ulike makroskopiske funn ble undersøkt nærmere mikrobiologisk og histologisk. Det ble

påvist sammenheng mellom pleuritt og pneumoni samt utbredelsen av pneumoniske lesjoner. Lungeorm-knuter som indikerte Muelleriusvandringer opptrådte uavhengig av andre makroskopiske forandringer eller mikrobiologiske funn. Det makroskopiske og histologiske bilde av pneumoniske fortetninger viste subakutte til kroniske betennelsesforandringer af en proliferativ eksudativ type. Det ble påvist sammenheng mellom pneumoniske fortetninger og tilstedeværelsen av Mycoplasma ovipneumoniae samt utbredelsen av lesjoner og veksttiter. Proliferative forandringer i bronkiole- og bronkie-epitel såvel som i det intrapulmonale lymfoide vev ble funnet å være sterke indikasjoner for tilstedeværelsen av M. ovipneumoniae. Videre ble det påvist sammenheng mellom intensiteten av histologiske forandringer og veksttiter. M. ovipneumoniae antas å være av primær etioolgisk betydning for subakutt eller kronisk pneumoni som finnes hos høstslaktede lam.

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