AN OUTBREAK OF CONTAGIOUS BOVINE PLEUROPNEUMONIA IN THE OWAMBO MANGETTI AREA OF SOUTH WEST AFRICA/NAMIBIA: MICROBIOLOGICAL, IMMUNOFLUORESCENT, PATHOLOGICAL AND SEROLOGICAL FINDINGS

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ABSTRACT

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An outbreak of contagious bovine pleuropneumonia (CBPP) in the Owambo Mangetti area of South West Africa/Namibia in 1982 led to the slaughter of 3 153 cattle. The lungs of 266 of these animals were scrutinized for gross lesions of CBPP and samples of lung tissue, sequestral contents or pleural fluid were submitted to the Veterinary Research Institute (VRI), Onderstepoort for microbiological, immunofluorescent and pathological examination.

Immunofluorescence proved to be the most successful method of diagnosis producing 96 % of positives, while mycoplasma isolations were only positive in 64 % of the 55 specimens processed in parallel. This clearly demonstrated the value of the former technique in the accurate as well as rapid diagnosis of CBPP. The impression smear technique employed, using Eriochrome black counterstaining proved most satisfactory and easy to interpret. The isolation of *M. mycoides* was influenced by the transit times and temperature of the samples on arrival at the VRI. No pathogenic bacteria were found in routine aerobic bacterial cultures from 27 of the samples submitted. Complement fixing antibodies were present in the sera of 16 cattle and titres varied between 10 and 320. Animals in which antibodies were absent included those with early lung lesions and some with sequestra.

INTRODUCTION

Contagious bovine pleuropneumonia (CBPP) or lungsickness, caused by *Mycoplasma mycoides* subsp. *mycoides*, SC., had been prevalent in Europe for centuries before it was introduced into southern Africa by way of a Friesland bull, imported from Holland and landed at Mossel Bay in 1854 (Henning, 1956). Although legislation dealing with the disease in the Cape Colony had been passed during the previous year, CBPP was spread rapidly by trek oxen, causing the death of over 100 000 cattle within 2 years. Lungsickness prevailed as the most destructive disease of cattle in South Africa until 1896, when it was completely overshadowed by rinderpest. After the eradication of rinderpest, the disease again assumed serious proportions. At the turn of the century, a strict policy for the extirpation of lungsickness was adopted, and since 1921 the Republic of South Africa (RSA) has remained free of the disease (Henning, 1956).

Contagious bovine pleuropneumonia spread to South West Africa (SWA)/Namibia in 1856 from the Cape Province, but stringent control measures confined it to a localized outbreak which was soon eradicated. It was reintroduced, however, in 1859, when it spread throughout the country. In 1919, the disease was eradicated from the central, commercial farming areas of SWA/Namibia. Lungsickness is, however, still endemic in the communal farming areas in the Kavango and Owambo districts on the northern borders of SWA/Namibia (Fig. 1), as well as in Angola (FAO-WHO-OIE, 1986; Schneider, 1987). The true incidence of the disease in these areas is difficult to determine. Two confirmed outbreaks, involving 14 animals, were recently reported from Kavango, and 2 foci, each involving 1 animal, were confirmed in Owambo (Anon, 1987). The Owambo Mangetti area and Kaokoland are regarded as risk areas. Control measures in these areas include restriction on movement of cattle and vaccination (Anon, 1987).

Relatively few reports of natural outbreaks of CBPP have been recorded and these were generally concerned with field control methods or clinical, serological and pathological data (Bygrave, Moulton & Shifrine, 1968). An outbreak of CBPP in the Owambo Mangetti area in 1982 provided an opportunity to record microbiological, immunofluorescent, pathological and serological findings in naturally occurring cases of the disease.

HISTORY OF OUTBREAK

The Owambo Mangetti area in which the outbreak occurred, lies between longitudes 17°–18° E and latitudes 18°–19° S. It consists of sandy savannah bushveld with a warm climate and an annual summer rainfall of between 500–550 mm.

During March 1981, 526 head of indigenous Sanga type cattle were moved from Kaokoland to the Owambo Mangetti area in SWA/Namibia (Fig. 1). In October 1981 several animals from this herd were placed in a camp adjacent to an established herd of 2 627 *Bos taurus* type cattle, which included 770 calves. Communal watering troughs, which served both camps on either side of a fence, were the means of contact between the 2 extensively kept herds. Approximately 3 months later, some of the Sanga type cattle showed signs of pneumonia. On testing, a few of these animals had complement fixation antibodies against CBPP. On further examination, several of the animals in the already established herd were also affected, and a group of 296 *Bos taurus* type cattle, some being serologically positive and others clinically ill, were slaughtered. Gross lesions of CBPP were evident in the majority of these animals, including 69 out of 70 2-9-month-old calves which, 42 days previously, had been serologically negative. It was decided to slaughter the remainder of the 2 herds, comprising 3 153 cattle, at the Oshakati abattoir in SWA/Namibia.

MATERIALS AND METHODS

The lungs of 266 randomly selected cattle were thoroughly examined at slaughter for gross lesions of CBPP. Specimens were collected for microbiological, immunofluorescent, pathological and serological examination.

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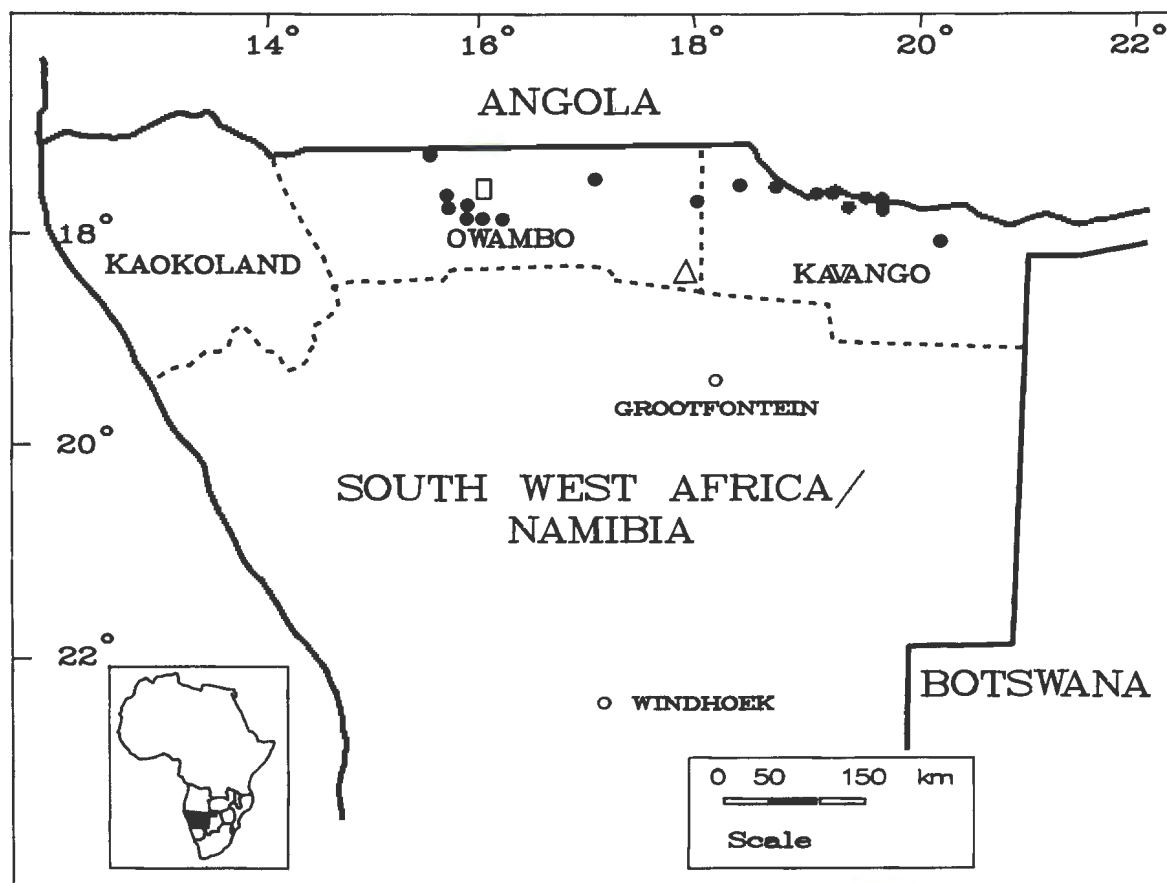
Legend:
———— International borders
····· District boundaries
△ Owambo Mangetti outbreak
□ Oshakati
● Confirmed outbreaks of CBPP 1985 — 1988

FIG. 1 Map to indicate confirmed outbreaks of CBPP

Microbiology and immunofluorescence

Specimens

Four batches of samples from 77 cattle with lesions of CBPP in various stages of development were submitted to the VRI, Onderstepoort, for the isolation of mycoplasma (Table 1). In order to maintain a temperature of 4–8 ° C during transport to the laboratory over a presumed 24 h period, samples of hepatized lung, sequestra or pleural fluid were packed in isothermic polystyrene holders containing prefrozen packs.

Media employed

Hayflick's agar and Hayflick's broth (Hayflick, 1965), containing 0,5 mg/mℓ ampicillin, were used for mycoplasma culture media.

Processing of samples

A few drops of pleural fluid were plated onto agar as well as in broth. Small portions of sequester and lung material were fragmented, to which an equal volume of Hayflick's broth was added, mixed well on a test tube shaker and filtered through gauze, before being plated on agar. In addition, other portions of sequester and lung material were suspended in equal volumes of broth, homogenized and passed through a 0,65 µm millipore filter. A few drops of the latter filtrate were then used to seed further broth cultures.

Impression smears

Impression smears for immunofluorescent studies were made from 14 specimens from Batch 2 and all the specimens from Batches 3 and 4 (Table 1), using the following method: glass microscope slides were used to make impression smears from lung and sequestral contents. In the case of pleural fluid, a drop was placed on a glass slide and evenly spread over 2 cm². All these specimens were allowed to dry at room temperature and heat fixed.

The impression smears were then stained by the fluorescent antibody technique (Del Giudice, Robillard & Carski, 1967; Baas & Jasper, 1972) for 30 min at room temperature and counter stained with a 20 % Eriochrome black solution for 20 s to reduce non-specific fluorescence (Hall & Hansen, 1962).

As a control, 30 randomly selected bovine lung specimens, which included normal as well as pathological material, were obtained from the abattoir and the Section of Pathology, VRI, Onderstepoort. These specimens were examined by making impression smears, as described, and fluorescent antibody tested.

The following monospecific hyperimmune rabbit antisera were used: *Mycoplasma mycoides* subsp. *mycoides* PG1, *Mycoplasma bovirhinis* PG43, *Mycoplasma arginini* G230, *Mycoplasma bovis* PG45 (Do-

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TABLE 1 The effect of temperature and transit time on positive isolation and immunofluorescence in CBPP

Batch No.	Days in transit	Temperature on arrival	Specimens for *Mycoplasma* isolation and immunofluorescence		
			Submitted	Positive isolation	Positive immuno-fluorescence
1	4	12 °C	11	6	Not done
2	3	12 °C	25	15	14/14
3	3–4*	24 °C	22	7	20
4	1	4 °C	19	17	19

* Specimens were collected on 2 consecutive days

TABLE 2 Serology, microbiology, immunofluorescence and pathology in 24 cases of CBPP

Case No.	CFT titre	Specimen submitted for isolation and immunofluorescence	Isolation	Immunofluo-rescence	Macroscopical pathology of lung	
					Stage of lesions	Number; and approximate size* of lesions (*large: more than 50 mm in diameter) (*medium: 15–50 mm in diameter) (*small: 5–15 mm in diameter)
1	320	Thoracic fluid	+	+	Acute pneumonia	Major portion of one lung
2	320	Lung tissue	+	+	Acute pneumonia	Major portion of one lung
3	160	Lung tissue	+	+	Acute pneumonia	Major portion of one lung
4	10	Lung tissue	+	+	Acute pneumonia	One; large
5	–	Lung tissue	+	+	Acute pneumonia	One; small
6	–	Lung tissue	+	+	Acute pneumonia	One; small
7	40	Lung tissue	+	+	Subacute pneumonia	One; large
8	80	Lung tissue	+	+	Subacute pneumonia	One; medium
9	320	Lung tissue	+	+	Subacute pneumonia	One; medium
10	10	Lung tissue	+	+	Subacute pneumonia	Two; small
11	–	Lung tissue	+	+	Subacute pneumonia	Two; small
12	–	Lung tissue	–	+	Subacute pneumonia	Two; small
13	80	Lung tissue	+	+	Subacute pneumonia	One; small
14	–	Lung tissue	+	+	Subacute pneumonia	One, small
15	320	Sequestral contents	+	+	Sequestrum	One; large; involving more than 1 lobe
16	320	Sequestral contents	+	+	Sequestra	Several; large
17	80	Sequestral contents	+	+	Sequestra	Two; large
18	180	Sequestral contents	+	+	Sequestrum	One; large
19	80	Sequestral contents	+	+	Sequestra	Several; medium
20	10	Sequestral contents	+	+	Sequestrum	One; medium
21	20	Sequestral contents	–	+	Sequestrum	One; small
22	–	Sequestral contents	+	+	Sequestrum	One; small
23	–	Thoracic fluid	+	+	Fibrotic scar	One; small
24	–	Lung tissue	–	+	Fibrotic scars	Two; small

netta), *Mycoplasma bovoculi* M165/69, and *Acholeplasma laidlawii* PG8 (Sewage A). These type cultures were obtained from the National Collection of Type Cultures, London.

Identification of Mycoplasma mycoides *isolates*

The mycoplasma isolates on agar were identified by the direct IFT, as described (vide supra), and the broth cultures by the growth inhibition test (Dighero & Bradstreet, 1970). The latter technique involved the seeding of agar plates with 0,5 mℓ of 10^6–10^8 cfu cultures and these were allowed to dry. One well, 5 mm in diameter, was then made in the centre of each agar plate and a drop of hyperimmune serum deposited in each.

Bacterial isolations

Isolations for aerobic bacteria by routine bacteriological methods were performed on 27 of the samples submitted.

Pathology

Representative pieces of hepatized lung or sequestra from 24 randomly selected cattle (included in batches 3 & 4) with lesions of CBPP in various stages of development were collected at slaughter for light microscopy (Table 2). Samples were fixed in 10 %

buffered formalin, routinely processed and stained with haematoxylin and eosin (HE).

Serology

The complement fixation test (CFT) was performed on the sera of the above-mentioned 24 animals, according to the method of Campbell & Turner (1953), using 2,5 units of complement (Table 2).

RESULTS

Microbiology and immunofluorescence

The results are summarized in Tables 1 & 2. The transit times of the batches of specimens ranged from 1–4 days and the temperatures in the containers on arrival at the VRI, Onderstepoort, varied between 4° C and 24 °C.

Mycoplasma mycoides was isolated from 45 of the 77 specimens that were submitted (Fig. 2). From the 55 samples on which isolations and immunofluorescence were performed in parallel, *M. mycoides* was cultured from 35 cases (64 %) while 53 cases (96 %) were positive with immunofluorescence (Fig. 3 & 4). Batch 2 consisted of 11 specimens from adult cattle and 14 specimens from 2–9-month-old calves. *Mycoplasma mycoides* was cultured from 4 of the adult

FIG. 2 Colonies of the field strain of *M. mycoides* as cultivated on Hayflick agar medium. Note pleomorphism
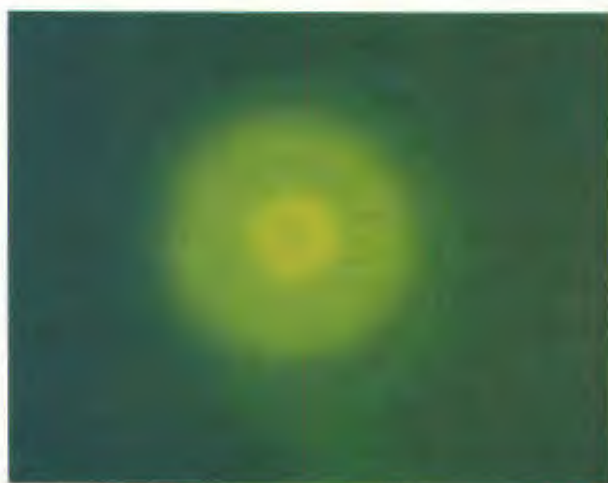

FIG. 3 Immunofluorescence of a single colony of *M. mycoides* field strain, cultivated on Hayflick agar medium
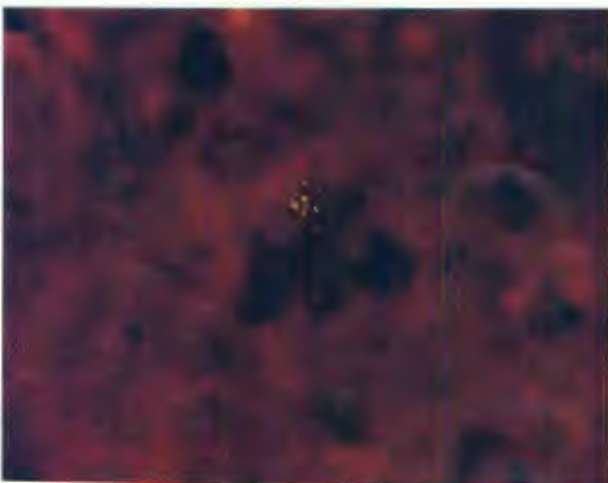

FIG. 4 Immunofluorescence of the *M. mycoides* field strain as seen on an impression smear

cattle and from 11 of the calves. Immunofluorescence was also done on the 14 calf specimens, which all proved positive.

The 30 lung specimens which served as controls were negative by immunofluorescence.

Routine bacteriological isolations consisted mainly of *Lactobacillus* spp. and other non-pathogenic bacilli with *Aeromonas hydrophila* and *Proteus* spp. as contaminants.

Pathology

In the 24 animals from which tissues were collected for pathology, the lesions in the lung were arbitrarily classified as acute (Cases 1–6), subacute (Cases 7–14) or chronic (Cases 15–24; Table 2). The more prominent features of the lesions in each group are described.

Macroscopical pathology

Acute lesions: In Cases 1–4 there was an extensive serofibrinous to fibrinous pleuritis and the pleural exudate was copious in one animal. Areas of acute pneumonia affected the major portion of one lung, the right lung being more commonly involved (Fig. 5). On cut surface, the lobules showed varying degrees of red and grey hepatization, which gave the lung a marbled appearance (Fig. 5). The marbled effect was further accentuated by distension of the interlobular septa and interstitium surrounding the vessels and airways with a yellowish serofibrinous exudate. Thrombosis was evident in some blood vessels and lymphatics, whilst haemorrhages were sometimes present in the lobules and occasionally in the septa. The walls of the bronchi in affected areas were thickened by oedema, their lumina often containing fibrinous material.

In each of Cases 5 & 6, a small, well-circumscribed reddish area of consolidation was apparent in one lung, but no pleuritis was evident.

Subacute lesions: Lesions grouped as subacute were characterized by necrosis, organization within lobules and interlobular septa and early sequestrum formation. One or sometimes 2 lesions of variable sizes were present, particularly in the right lung.

Necrosis of a single lobule or groups of lobules was a prominent feature in the majority of the subacute cases (Fig. 6). Necrotic areas were initially demarcated from adjacent tissues by irregular yellow-grey zones and later became sequestrated by granulation tissue.

Chronic lesions: Cattle with lesions classified as chronic were those with well-defined sequestra in various stages of organization or liquefaction, or those with resolved lung lesions where only fibrotic scars were evident. Sequestra, present in 8 animals, varied in size from 15 mm or less (Cases 21–22) to very large, involving more than one lobe of a lung (Case 15; Fig. 7). The sequestra were often surrounded by a thin irregular fibrous capsule which followed the bronchial ramifications and contained reddish-pink to yellowish-grey necrotic tissue. The normal pulmonary architecture was still visible in some of these sequestra. In others, the fibrotic capsule was thick and enclosed yellowish liquefactive material. In a few cases, the sequestral content had been expelled via either the bronchial tree or into the thoracic cavity.

The lungs of cases 23 & 24 contained small fibrotic scars, but no necrotic sequestra.

Other organs: In many cases the bronchial lymph nodes were markedly enlarged, whilst in one animal, a multifocal interstitial nephritis was evident.

Microscopical pathology

Acute lesions: Different stages of inflammation which varied between as well as within lobules of the

FIG. 5 Acute case of CBPP. The major portion of the right lung is enlarged. On the cut surface there is red and grey hepatization, haemorrhage of lobules and distension of interlobular septa
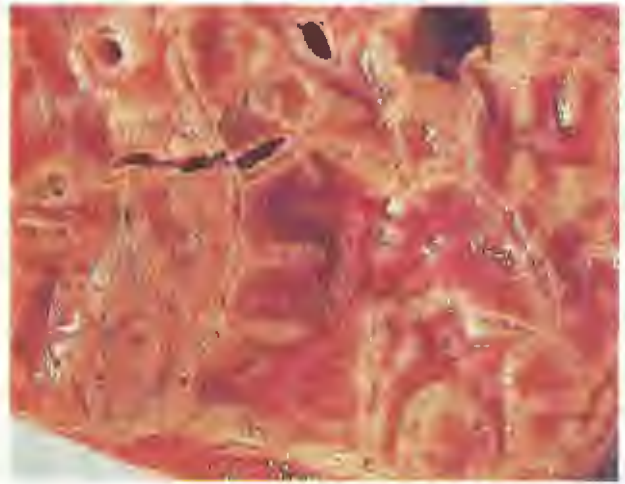

FIG. 6 Subacute case of CBPP. Cross-section of lung showing the marbled appearance and thickening of interlobular septa. Several lobules are necrotic and early sequestrum formation is evident

FIG. 7 Chronic case of CBPP. Large sequestrum containing yellowish-grey necrotic material

same lung were noticeable in Cases 1–4. Initially there was severe hyperaemia of alveolar capillaries, and the alveoli were flooded with a serous, serosanguineous to serofibrinous exudate which became increasingly proteinaceous. As the lesions progressed, variable numbers of neutrophils, which became necrotic early in the infection, infiltrated the alveoli and were followed by macrophages (Fig. 8 & 9). There were often haemorrhages within the alveoli.

The interlobular septa were distended by the accumulation of a serofibrinous exudate in the interstitial connective tissues and within the lymphatics, resulting in marked dilation of the latter. Necrotic neutrophils accumulated along the inner walls of the lymphatics and in particular along the margins of the septa, where a distinct inflammatory zone was frequently appreciable (Fig. 10). The walls of the lymphatics were often necrotic and indistinct, and fibrin thrombi were common.

An exudate similar to that in the alveoli was frequently present in the lumina of the bronchi and the bronchioli of affected lobules, and often the lumina were occluded by the exudate (Fig. 8). The epithelium was necrotic in some areas. The bronchial and
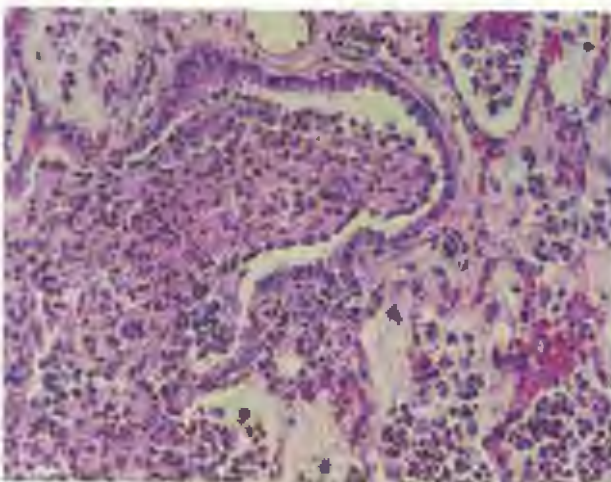

FIG. 8 Acute case of CBPP. Note inflammatory exudate within a bronchiolar lumen, and proteinaceous fluid and neutrophils in the alveolar spaces. HE × 250.
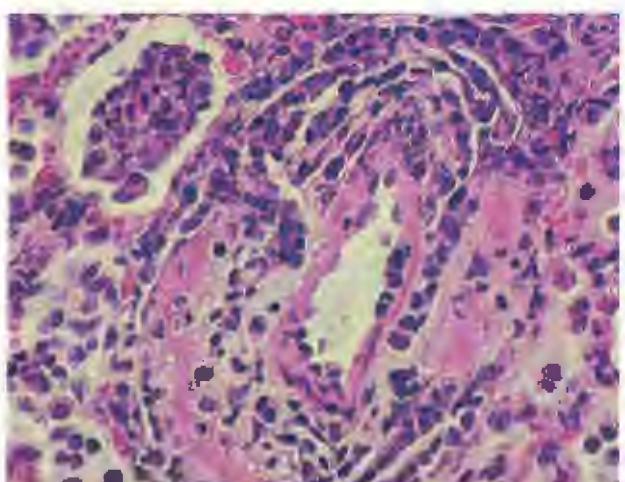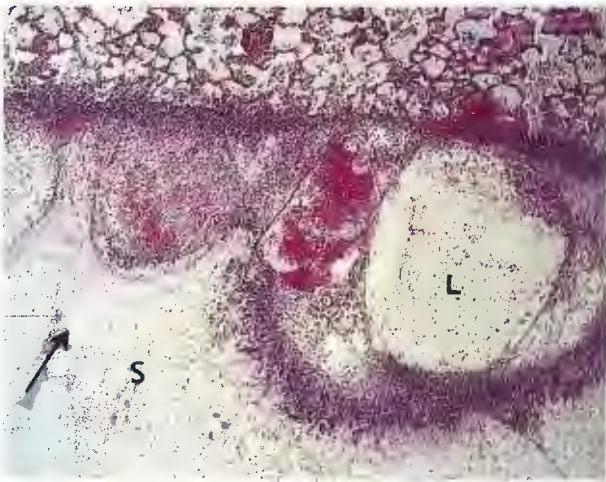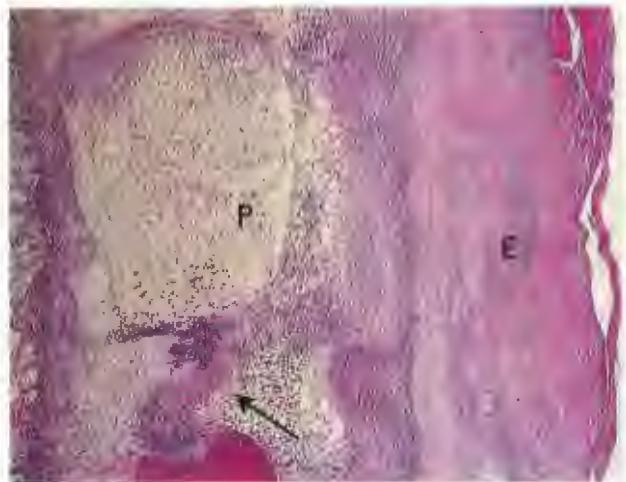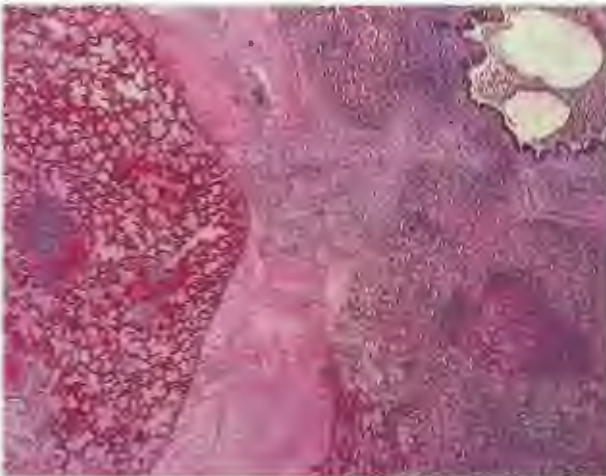

FIG. 9 Acute case of CBPP. Vasculitis (arrow) and exudation of fibrin, neutrophils and macrophages in perivascular and alveolar spaces. HE × 300
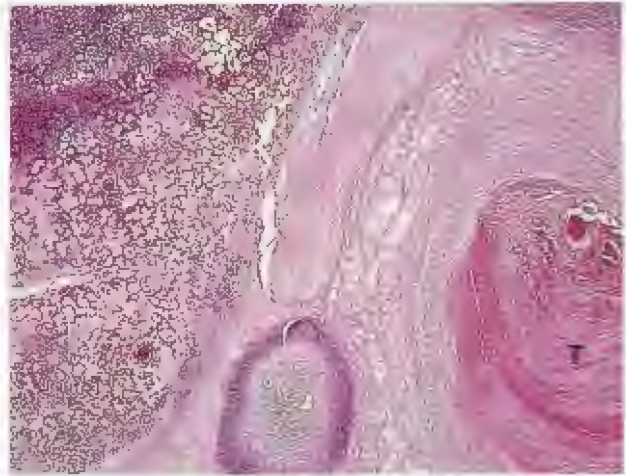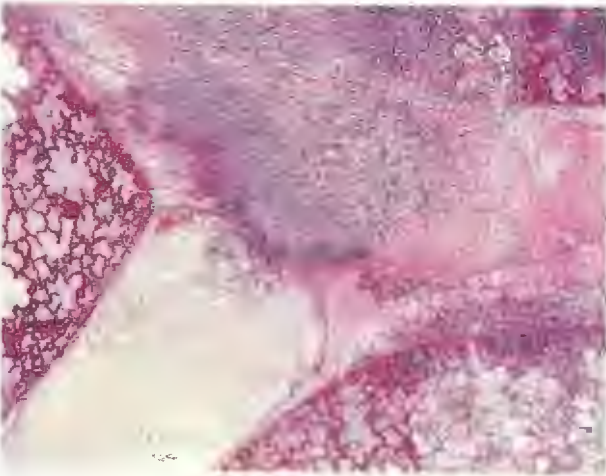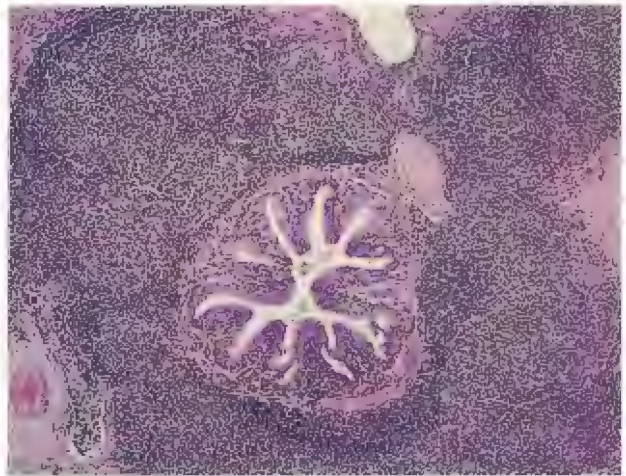
FIG. 10 Acute case of CBPP. Distension of interlobular septum (S) and lymphatics (L) by a serofibrinous exudate. Some of the lymphatics contain thrombi (arrow). Necrotic neutrophils form inflammatory zones between the septum and lobule, and along the walls of the lymphatics. HE × 200

FIG. 11 Acute case of CBPP. The pleura and lymphatics (P) are distended by a serofibrinous exudate admixed with moderate numbers of neutrophils which form distinct inflammatory zones (arrow). A fibrinonecrotic exudate (E) covers the pleura. HE × 80

FIG. 12 Subacute case of CBPP. Different stages of pneumonia in adjacent lobules and fibrosis of the interlobular septum. HE × 80

FIG. 13 Subacute case of CBPP. Thrombosis of an interlobular artery (T) resulting in necrosis of the septum and adjacent lobule. A distinct inflammatory zone is present at the border of the necrotic area. HE × 60

FIG. 14 Subacute case of CBPP. Interlobular septum: the lower portion is distended by a serofibrinous exudate, whilst the upper portion shows a more advanced stage with mononuclear cell infiltration and organization by fibrous tissue. HE × 200

FIG. 15 Chronic case of CBPP. Extensive peribronchial infiltration of lymphocytes forming several follicle-like aggregates. HE × 100

bronchiolar mucosae and submucosae were oedematous and infiltrated by neutrophils. The lymphatics, and, to a lesser extent, the peribronchial and perivascular areas were prominently distended by a serofibrinous exudate admixed with mononuclear cells, and neutrophils. Lymphangitis, accompanied by thrombosis, was a prominent feature in these areas.

Inflammation of intra- and interlobular arteries and veins occurred commonly in affected portions of the lung and was characterized by hypertrophy of endothelial cells and the accummulation of a few plasma cells and lymphocytes subendothelially; necrosis and vacuolation of mycocytes in the media; and an infiltration of mild to moderate numbers of mononuclear cells within the media and adventitia of the vessels, as well as in the perivascular spaces (Fig. 9). Vascular thrombosis was occasionally noticed.

The microscopical lesions in Cases 5 & 6 were similar to those in Cases 1–4, but were limited in extent and interpreted as early lesions. A fibrinonecrotic exudate covered the visceral pleura in Cases 1–4. The other changes in the pleura resembled those described in the septa (Fig. 11).

Subacute lesions: The interlobular septa became organized by fibrous connective tissue, arising from the periphery of blood vessels and from septal walls (Fig. 12 & 14). The fibrosis was accompanied by a widespread infiltration of lymphocytes, sometimes forming discrete foci, and lesser numbers of plasma cells and macrophages. Fibrosis often spread into adjacent lobules and resulted in partial or complete obliteration of the alveoli, while in other lobules, moderate numbers of lymphocytes and plasma cells infiltrated the alveolar walls.

A lobule, or part of a lobule, or multiple lobules, and their intervening septa became necrotic. Vasculitis and thrombosis of intralobular and interlobular arteries was noticed in some of the necrotic areas (Fig. 13). Initially, a zone of necrotic inflammatory cells separated the infarcted area from the surrounding tissue, but eventually fibrous connective tissue at the edges of the lesions and thickened interlobular septa sequestrated the necrotic tissue from the neighbouring parenchyma.

The bronchial mucosa and submucosa, and peribronchial and perivascular areas were infiltrated by variable numbers of round cells, accompanied by fibrosis. Polypoid projections of fibroblastic tissue partially or completely obliterated the bronchiolar lumina in some areas.

Chronic lesions: Sequestra were surrounded by fibrous capsules of variable thicknesses composed of an inner zone of capillaries, fibroblasts, macrophages, lymphocytes and occasionally giant cells, and an outer zone of fibrous connective tissue. The sequestral content usually consisted of a mass of eosinophilic necrotic tissue. Some of the sequestra contained massive numbers of necrotic neutrophils, cellular debris and mineralized material. Multifocal infiltrations of lymphocytes, often forming follicle-like aggregates, occurred in the fibrous capsules of some of the sequestra and in thickened fibrotic interlobular septa, as well as in the peribronchial and perivascular areas (Fig. 15).

Serology

The results of the CF tests are summarized in Table 2. Antibodies were detected in the sera of 16 cattle, and the titres varied between 10 and 320. Antibodies were absent in animals with early lung lesions (Cases 5 & 6) and in 2 of the subacute cases, as well as in some cattle which were chronically affected and harboured pulmonary sequestra.

DISCUSSION

Immunofluorescence is a rapid and very specific technique for the diagnosis of CBPP (Masiga & Stone, 1968; Provost, Perreau, Breard, Le Goff, Martel & Cottew, 1987). In this study, there was positive immunofluorescence on impression smears from 53 out of 55 animals with lesions of CBPP, which confirmed the accuracy of this technique. Thus, impression smears which are made in the field, air-dried and brought to a well-equipped laboratory could be used most effectively in making a rapid diagnosis of CBPP.

The specific antigen galactan occurs on the surface of *M. mycoides* and probably forms an integral part of this structure. It may give rise to difficulties in fluorescent antibody work, as this polysaccharide is normally present in lung tissue. After 30 bovine lung specimens from CBPP negative animals were processed, and no fluorescing material was recorded, this factor was regarded to be negligible. The major disadvantage of this technique is that the test can only be accurately interpreted on animals which are already dead. Furthermore, the operator must be experienced in performing the immunofluorescent technique.

From the results summarized in Table 1, we concluded that the transit times and temperature of the samples on arrival at the VRI, Onderstepoort, influenced the isolation percentages. Specimens should be taken as sterile as possible and packed in isothermic containers in order to maintain the temperature at 4 °C–8 °C. They should reach the laboratory within 24 h and be processed immediately.

Though no single serological test is capable of detecting all infected animals during the various stages of the disease (Gourlay, 1965), the CFT is still regarded as the single most specific and sensitive serological test for the diagnosis of CBPP since its inception by Campbell & Turner in 1936 (Ladds, 1965; Gourlay, 1983). Discrepancies include serologically negative animals in the very early stages of the disease (Cases 5 & 6; Table 2). False negatives may also occur after the acute stage has passed, especially in those animals having small sequestra encapsulated by thick fibrous walls (Cases 21 & 22; Table 2). The latter may be explained by the fact that a positive CFT is dependent on the presence of sufficient circulating antigens and a thick fibrous capsule does not allow adequate diffusion of organisms into the circulation (Hudson, 1971). Case 23 was serologically negative, but *M. mycoides* was isolated from the thoracic fluid. This phenomenon is occasionally encountered and may be due to the use of insensitive or inappropriate serological tests, lengthy incubation periods or other unkown factors (Windsor, 1977; Onoviran & Taylor-Robinson, 1979). This fact must be borne in mind when isolation techniques are used to evaluate serological tests.

Queval, Provost & Villemot (1964) developed a more sensitive modification of the CFT, but the extra sensitivity of the latter test has the disadvantage of producing more false positives. This is particularly true in the case of the diagnosis of CBPP (Shifrine & Gourlay, 1967). Chima & Pam (1985), comparing the CFT with the more sensitive passive haemagglutination test (PHA), concluded that this highly sensitive test had a lot of advantages over the CFT and

could be a valuable additional tool in the effort to control CBPP.

The spread of CBPP can be dramatic, although the incubation period is ill-defined and can vary from 5 to 207 days (Martel, Perrin, Belli & Froget, 1983). With only a communal fence and communial watering troughs serving as contact points, 69 out of 70 calves aged 2–9 months, which 42 days previously had tested serologically (CFT) negative, all showed typical lung lesions at slaughter. The above circumstances provided sufficient close contact to allow the rapid transmission of the disease and illustrated once again that young animals are highly susceptible to CBPP (Masiga & Windsor, 1978). At slaughter, a smaller number of positive animals were recorded in the Sanga type oxen than in the home herd, stressing the point that CBPP is a disease of moving cattle.

The lesions in the lungs of the 24 cattle in this study correspond with those described previously for CBPP (Meyer, 1909; Henning, 1956; Bygrave et al., 1968; Hudson, 1971). The nature of the primary lesion in CBPP is accepted by most workers to be primary respiratory bronchiolitis with a secondary involvement of the lymphatics and blood vessels (Bygrave et al., 1968; Hudson, 1971). In this study, respiratory bronchiolitis, thrombosis of intra- and interlobular arteries and lymphatics were common, particularly in the subacute cases. According to Bygrave et al. (1968), thrombosis of the former vessels give rise to necrosis of a lobule or part of a lobule, while involvement of the interlobular arteries culminates in infarction of multiple lobules and intervening septa.

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