

RECOGNIZING CONTAGIOUS BOVINE PLEUROPNEUMONIA

(REVISED EDITION)

FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS
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FOREWORD

This booklet is one of a series prepared by FAO's Emergency System for Transboundary Animal and Plant Pests and Diseases (EMPRES) (Livestock) Unit as an aid to emergency preparedness for major transboundary diseases of livestock.

Contagious bovine pleuropneumonia (CBPP), caused by *Mycoplasma mycoides* subsp. *mycoides* Small Colony variant (*MmmSC*), is a major obstacle to cattle production in Africa, and indeed considered to be one of the great cattle plagues, following closely on the heels of rinderpest. The disease appeared to be under control in the 1970s following intensive vaccination coupled with strict movement controls. However, it made a spectacular return in the 1990s, affecting areas previously known to be free from the disease. Increased outbreaks were likewise observed in known enzootic areas. The ability to recognize the disease in the field and the capability to confirm the diagnosis of the disease in the laboratory accurately are very important components of epidemiological surveillance for CBPP. Important decisions on control options are based on information obtained from such surveillance. This manual has been prepared with these factors in view and it is expected that it will assist all stakeholders in cattle production on the African continent and elsewhere with familiarization with key epidemiological features of the disease, allowing early recognition and diagnosis.

Remember: *Early warning is the key to early reaction for containment, control and rapid elimination*

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CONTENTS

List of abbreviations used in the text	vi
Acknowledgements	vi
INTRODUCTION	1
THE DISEASE	3
The cause	3
Animals affected	3
Geographical distribution	3
Transmission and spread	4
CLINICAL SIGNS	5
INVESTIGATION	7
Epidemiological investigation	7
Clinical examination	9
POST MORTEM FINDINGS	11
DIFFERENTIAL DIAGNOSIS	13
DIAGNOSIS	15
Laboratory confirmation	15
Samples required for laboratory testing	15
Interpretation of laboratory results	15
CONTROL	17
SOURCES OF ASSISTANCE	18
COLOUR PLATES	19

LIST OF ABBREVIATIONS USED IN THE TEXT

CBPP	Contagious bovine pleuropneumonia
CFT	Complement fixation test
DAHP	Department of Animal Health and Production (Botswana)
ELISA	Enzyme-linked immunosorbent assay
EMPRES	Emergency Prevention System for Transboundary Animal and Plant Pests and Diseases
FMD	Foot-and-mouth disease
IZST	<i>Istituto Zooprofilatico Sperimentale dell'Abruzzo e del Molise "G. Caporale"</i> (Teramo, Italy)
<i>MmmSC</i>	<i>Mycoplasma mycoides</i> subsp. <i>mycoides</i> Small Colony variant
PAP	Peroxidase-antiperoxidase test
PCR	Polymerase chain reaction
TADInfo	[EMPRES] Transboundary Animal Disease Information System

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Their contributions are gratefully acknowledged.

INTRODUCTION

Contagious bovine pleuropneumonia (CBPP) is one of the great plagues that continue to devastate the cattle herds on which so many people are dependent in Africa. In recent years, the disease has emerged from areas where it has been persisting in endemic form to re-invade other areas from which it had previously been eradicated. In addition to these newly infected areas, the endemic areas are experiencing an upsurge in the incidence of CBPP.

Recent dramatic events confirm that early recognition of the disease after its introduction or re-introduction to a country, or previously free zone of an infected country, is essential if control and elimination are to be achieved rapidly. Only if the introduction is detected rapidly can stamping out by slaughter of infected herds – undoubtedly the most cost-effective option in the long term – be considered an affordable strategy option for many countries. Because of the nature of the disease, any delay can result in widespread dispersal of infection, complicating and greatly increasing the costs of any control measures adopted. Vigilance is therefore required, whether at the national or district level, to ensure that the disease does not escape detection.

This manual presents the most important features of CBPP to enable the disease to be recognized, both by clinical and post-mortem examinations. It is intended for use by all veterinary and paraveterinary staff in the front line of defence against the disease, and also to assist in informing farmers of the risk and key features of CBPP so as to enable early recognition of the disease.

CBPP is a serious threat to livestock production in sub-Saharan Africa and some Asian countries. It is a serious obstacle to livestock development. Once introduced to a new area, initial losses can be very high and its eradication is difficult, requiring major expenditure for control.

THE DISEASE

Contagious bovine pleuropneumonia (CBPP) is an infectious and highly contagious disease of cattle and water buffaloes, and considered to be amongst the most important infectious diseases. Affected animals have difficulty in breathing due to damage to the lungs, lose condition and a proportion die. All ages of cattle are susceptible, but young cattle develop joint swellings rather than lung infections. Many cattle show no disease signs, despite being infected. Others recover quickly after a transient mild disease, yet they can carry the infection for as long as two years, and may be responsible at a later stage for passing on infection to susceptible cattle.

The cause

The disease is caused by a bacterium called *Mycoplasma mycoides* subsp. *mycoides* Small Colony variant (*MmmSC*), which is difficult to see even with a light microscope. However, growth of the organism can be seen when infectious material is cultured on suitable media in the laboratory.

Animals affected

Cattle of all types (both *Bos taurus* and *Bos indicus*) are susceptible; domestic buffaloes are generally more resistant. CBPP has been reported in Asian yaks and in American bison, but never in African buffaloes (*Syncerus caffer*). Sheep and goats are resistant to the disease.

Geographical distribution

CBPP is widespread in Africa and is recognized to be present in some countries of Asia and Europe.

In Africa, it is found in an area south of the Sahara, from the Tropic of Cancer to the Tropic of Capricorn and from the Atlantic to the Indian Ocean. Endemic infection extends throughout the pastoral herds of much of western, central and eastern Africa, with Angola and northern Namibia in southern Africa. Newly infected areas in the 1990s include much of Uganda, parts of Kenya, the Ituri Region of the Democratic Republic of the Congo, and most of the United Republic of Tanzania, where recently the disease has spread alarmingly. Rwanda (1994), Botswana (1995, now free), Burundi (1997) and Zambia (1997) were recently re-invaded, but Lesotho, Malawi, Mozambique, South Africa, Swaziland and Zimbabwe are currently (2002) free.

In Asia, CBPP has been reported in recent times from Assam in India, Bangladesh and Myanmar. Sporadic outbreaks have been recognized in the Middle East, probably derived from importation of cattle from Africa.

CBPP was eradicated from the United States of America in 1892, Zimbabwe in 1904, South Africa in 1924, Australia in 1972 and China in the 1980s.

After virtual elimination from Europe in the nineteenth century, the disease reappeared in Portugal and Spain in 1951 and 1957, respectively. Outbreaks have been reported in southern France on a few occasions, the latest being in 1984. In Italy, the disease reappeared in 1990 but was eliminated by 1993.

Transmission and spread

CBPP is invariably introduced into a herd by contact with an infected animal; transmission occurs from direct, close, repeated contacts between diseased and healthy animals in shared night accommodation or at water holes, dip tanks, markets, common grazing or gathering places. Indirect transmission from pastures and water or by carriage, for example, on people and feed sacks, is thought not to be important in the transmission of the disease.

The causative agent is present in liquid droplets in breath and urine. Although the CBPP organisms are killed rapidly in hot dry environments, airborne transmission appears possible over distances of up to 200 metres.

Transmission is favoured by close crowding of cattle, and outbreaks are more common and extensive when cattle are housed or have been transported by train or truck or trekked on foot in groups.

Chronically infected and symptomless animals play an important role in the persistence and spread of the disease. In this context, pastoral herds are especially significant since they may contain many chronically infected animals. Fleeing with apparently healthy animals away from a focus of active disease has been known to spread the disease widely.

CLINICAL SIGNS

Typically, when first introduced into a herd, CBPP is severe and mortality relatively high. A small proportion of cattle may die rapidly without showing any signs other than fever. It may be possible to link the onset of disease to previous contact with other cattle three to six weeks earlier, but this is not always the case as the incubation period can be as long as six months. Clinical signs may become apparent only several months after the contact. The disease can therefore become established in a herd before it is noticed, and tracing back to the origin can be difficult. This is particularly so where routine vaccination has been practised with long intervals between campaigns, and where antibiotics have been used to treat clinical cases. Both reduce the incidence of clinical disease, making its recognition more difficult.

After some time, the disease in the herd becomes chronic, clinical signs become less severe and the mortality rate falls. However, losses continue to occur. Not all the animals are affected in the same way and often the disease has a chronic course from its onset.

The hyperacute form, involving up to 10 percent of infected animals, may be observed at the onset of an outbreak. Death is sudden and is often without any other signs. The acute form is observed in approximately 20 percent of diseased animals. The course is usually from five to seven days. The earliest signs are a sudden onset of fever to 40°C or more and, in milking cows, a drop in milk yield. Sick cattle tend to isolate themselves from the herd and may stop eating.

A typical respiratory disease develops; breathing is laboured and obviously painful. Abdominal breathing with a respiratory rate of 50 to 55 breaths/minute may be seen and cattle may “grunt” when breathing out. Some animals develop a shallow, dry and painful cough, particularly noticeable on exercise. Application of pressure between the ribs is painful and resented by affected cattle, which sometimes react violently. On percussion, the ventral part of the chest sounds dull owing to the presence of fluid in the chest cavity. Acutely affected cattle stand with head and neck extended and forelegs spread apart (Plate 1), with dilated nostrils and mouth open panting for air. There may be nasal discharge, sometimes streaked with blood, and frothy saliva accumulates around the mouth. Some animals develop swellings of the throat and dewlap. Pregnant cows and heifers may abort, and diarrhoea has been recorded.

The subacute form occurs most frequently in about 40 to 50 percent of the animals affected. The symptoms resemble those of the acute form, but are less severe and fever is intermittent. This form usually becomes chronic.

The chronic form is a natural evolution of both acute and subacute forms but in some animals it may develop directly. The clinical signs regress but cattle can still have intermittent fever, together with loss of both appetite and weight.

Calves in the first six months of life more often show lameness from swollen, hot, painful limb joints.

The mortality rate is variable, rarely exceeding 50 percent, and depends on a range of factors, such as age, breed, nutrition, presence of other infections or infestations, and the type of management.

Many affected cattle appear to recover fully, yet the lesions in the lungs take a long time to heal completely. The causative agent can survive for as long as two years within the lesions. Up to 25 percent of affected cattle can become chronic carriers of infection. They are often referred to as “lungers” and are believed to play a role in initiating new outbreaks when they are introduced into susceptible herds.

In summary, look for one or more animals with:

- fast, difficult or noisy breathing;
- discharge from the nose; or
- coughing, especially after exercise.

Any chronic (persistent) mild cough in cattle otherwise appearing normal or losing weight should be a reason to suspect CBPP.

INVESTIGATION

Investigations leading to a conclusive decision will rely on a combination of the following activities:

1. Clinical examination.
2. Epidemiological investigation to obtain a general picture of disease pattern in the herd.
3. Post-mortem examination to observe the characteristic lesions in organs of dead and/or slaughtered animals.
4. Laboratory examination to confirm infection.

EPIDEMIOLOGICAL INVESTIGATION

When CBPP is suspected, the questions asked should include the following:

1. What species of animals (e.g. cattle, sheep, goats, pigs, and wild animals) are present on the livestock holding facility (or village). How many of each is present and which species are affected?

If domestic or wild animals other than cattle or water buffaloes are affected, a condition other than CBPP should be considered.

2. What ages of cattle/domestic buffaloes are affected?

Record the various age groups of the animals (e.g. under 6 months; 7 to 18 months; over 18 months). In CBPP, the more severe respiratory forms are observed in adult animals.

3. Have the cattle been vaccinated against CBPP or other epidemic diseases, and, if so, when did the last vaccination take place? Which vaccine was used? How many animals were vaccinated? Who conducted the vaccination?

If all the cattle have been vaccinated with a quality-assured CBPP vaccine at the appropriate time intervals, they should theoretically not develop the disease. However, CBPP can still occur in non-vaccinated cattle in partially vaccinated herds, and even in vaccinated cattle that have not been re-vaccinated as scheduled.

4. When did the first signs of disease appear? Is this the first time that this disease has occurred? If not, what are the approximate dates of previous episodes?

This can help to indicate whether the disease is endemic or newly introduced, and can help to calculate when infection entered the herd.

5. Have other cattle been bought or introduced for any reason during the six months before the disease was first noticed? If so, from where? Did any become sick?

The answer can provide a clue as to how the disease entered the herd.

6. Was the herd exposed to another herd, during the six months before the disease was first noticed? Do nomadic herds pass through the area? If so, when and from where?

Nomadic herds can be a CBPP reservoir. The answers can also provide an explanation of how the disease might have entered the farm or herd.

7. Are grazing lands, water holes, and drinking troughs or dipping tanks shared with other nomadic or sedentary herds?

This is to indicate possible contacts with animals from other herds, allowing tracing of the origin of outbreak, and therefore help to provide an early warning signal of disease spread in the locality.

8. Were replacement animals vaccinated against CBPP and other diseases before or after introduction to the herd?

This provides information why sickness might be limited to a particular group of animals.

9. Does the community know the disease and does it have a local name?

Pastoralists are often able to provide a useful guide to disease conditions they have encountered in the past.

10. Have the infected animals been treated with antibiotic(s)? If so, which type(s)?

Antibiotics may mask the clinical appearance of CBPP and alter the progression of disease in a herd. They may also alter the appearance of typical pathological lesions and thereby complicate diagnosis of the disease.

11. What are the signs observed in diseased animals?

Respiratory signs are more evident in adult cattle, whereas enlargement of joints may be present in calves under six months of age.

12. How many animals are clinically sick out of the total?

13. How many animals have died since the outbreak occurred ?

14. What is the health status in neighbouring herds?

To decide if CBPP is present in the area, the neighbouring herds should be inspected for evidence of disease.

15. Have any animals been sold, transferred or given on loan in the last six months, e.g. for ploughing or as gift (dowry)?

The answer to this question might give important information on spread of the disease and assist in tracing the source of the outbreak.

CLINICAL EXAMINATION

As the clinical appearance of the disease can differ between individuals in a herd depending on the different stages of disease development, it is important to examine as large a number of animals as possible in order to obtain a full clinical picture. A notebook is essential to systematically record all the findings for later reference. The use of pieces of paper is not recommended, as these often get lost, and the vital information with them.

1. Record the farmer's or animal attendant's observations

Ask for the farmer's or animal attendant's description of the disease observed.

Has any treatment been given? Antibiotics such as tylosin and the tetracyclines can be effective in modulating clinical symptoms and progress of disease.

[Conventional understanding is that antibiotic therapy is contra-indicated in outbreaks of CBPP because it is believed that its use leads to the generation of a high proportion of "lungers" (chronic carriers with sequestra in the lungs) in the herd and that these can later spread infection to susceptible cattle. This may be true, but in most countries in which CBPP occurs, antibiotic therapy is a fact of life. Disagreement over its use should not be allowed to create a barrier between the animal health worker and livestock owner.]

Have any cows aborted?

2. Observe the animals at rest

Before attempting to handle the animals, check if they are alert or depressed, if lameness is present, and if body condition is satisfactory for the time of year and type of management system.

Do any stand with the neck and head extended, forelegs spread apart, mouth open and panting for air? It is worth remembering that this also happens not only to:

- animals severely affected by CBPP – acute cases – but also to
- animals with respiratory diseases other than CBPP.

Is breathing difficult, rapid and painful? If breathing is difficult, the nostrils are generally dilated and clear or bloodstained discharge may be seen from the nostrils.

Check the character and rate of respiration. Is it fast (more than 20 per minute)?

Do any animals cough?

Is there discharge from the eyes and nose? A clear discharge may be present.

3. Physical examination

Take the rectal temperature: in acute cases it can rise above 40°C.

Check the surface lymph nodes: enlargement is not a feature.

Check the mouth, including the lining of the lips, tongue, cheek papillae and the hard palate – lesions are not found, unlike in rinderpest and foot-and-mouth disease (FMD), although saliva may dribble from the mouth.

4. Force the animals to run for a few minutes and examine them again

CBPP symptoms can be more clearly seen after a few minutes exercise – coughing and signs of lameness.

POST MORTEM FINDINGS

The carcass

Abnormalities (lesions) are generally confined to the chest cavity (Plate 2) except in young calves, where inflammation of the limb joints (usually the carpal and tarsal joints), with increased fluid, is sometimes seen (Plate 3).

A most striking feature of the acute disease is the very large volume of yellow fluid (up to 30 litres) containing clots, which can accumulate in the chest (Plate 4).

The lungs (almost always one) and pleura are affected. In most cases, only the diaphragmatic lobe is involved (Plate 5); it is firm and fleshy, resembling liver rather than healthy pink lung. It does not collapse when the chest is opened.

In acute forms, the yellowish fluid in the chest cavity may solidify and cover the lining of the chest and surface of the lung (the pleura) with a yellow or yellowish-grey coating resembling an omelette (fibrin) (Plate 6). Under this, the pleura is thickened and opaque. Accumulation of fibrin on the pleura causes the lung and chest wall to stick together (adhesion). The cut surface of the lung often shows a marbled appearance, with areas of different colour (dark red, red and pale pink) separated by a network of pale bands (Plates 7 and 8); this is typical of CBPP.

In the chronic form, fluid is rarely seen in the pleural cavity, but adhesions between lung lobes and between lungs and the chest wall are commonly found. A capsule of fibrous connective tissue surrounds areas of dead lung tissue. This structure is called a sequestrum [plural: sequestra] (Plate 9). Various intermediate stages between the acute lesion and a fully formed sequestrum can be found, depending on the stage of the disease. The diameter of a sequestrum can vary from 2 to 25 cm and the capsule can be as much as 1 cm thick. Sequestra of different diameter can be detected in the same lung. When they are small and deep they can be felt only by careful palpation.

In the pink or white necrotic – *odourless* – mass that is found in the sequestrum, the lobular structure of the lung may still be recognizable. This is typical of the disease and differs from lung lesions due to tuberculosis or abscesses. The contents of sequestra shrink, and become dry, although they may later become liquefied.

Lymph nodes in the chest may be enlarged and wet (oedematous), with small necrotic foci and pinpoint haemorrhages. The difference between cortex and medulla may be indistinguishable. In the kidney cortex, white spots of dead tissue of variable size, called infarcts, can sometimes be seen (Plate 10).

In summary, look for:

- yellow fluid in the chest cavity;
- lungs covered with yellowish material;
- lungs adhering to the chest wall;
- lungs which do not collapse and are solid or marbled; and
- sequestra in the lungs of chronic cases.

Slaughterhouse monitoring is a powerful tool to use in detecting introduction and spread of the disease because the lesions of CBPP are so characteristic.

DIFFERENTIAL DIAGNOSIS

In carrying out CBPP diagnosis it is necessary to differentiate this disease from other diseases that may present similar clinical signs or lesions. Remember that the disease pattern in a herd is as important as the findings in a single animal when carrying out an investigation.

Some sources of confusion are:

Rinderpest: The confusion with rinderpest results from the fever and discharges observed from the eyes, nose and mouth. However, the characteristic lesions of rinderpest, which are essentially erosions in the mouth and throughout the digestive tract, together with the profuse, often bloody diarrhoea in advanced cases, should enable easy differentiation from CBPP in which these are not seen. Lung lesions are seen in more chronic cases of rinderpest, consisting of red areas of collapse together with emphysema of lung lobules and the septa separating them. At this stage, the erosive lesions of rinderpest may have healed.

Foot-and-mouth disease (FMD): Salivation, lameness and fever are the cause of confusion.

Haemorrhagic septicaemia (HS): This is an acute disease and most affected animals die within 6 to 72 hours after the onset of clinical signs. Buffaloes are particularly susceptible. Oedema of the throat and neck to the brisket is often very pronounced. The lung lesions seen in animals that survive the longest can appear very similar to the marbling lesion of CBPP. There may be yellow fluid in the chest cavity and the affected lung may adhere to the inside of the rib cage. Thus, in the individual case distinguishing between HS and CBPP can be difficult.

Bacterial or viral broncho-pneumonia: Clinical signs may resemble closely those of acute CBPP. Post mortem examination shows usually both lungs to be affected, fibrinous exudate may be present but not to the same extent as in CBPP. While dark, solid areas of lung may be seen, these are usually restricted to the anterior lobes (not the diaphragmatic lobe as in CBPP) and marbled lungs are not often seen.

Theileriosis (East Coast Fever): Coughing, nasal and ocular discharge and diarrhoea are observed. Affected cattle show general enlargement of superficial lymph nodes and especially those of the head. The lungs contain much clear liquid, which is also present in the chest cavity; the airways in the lung may be filled with white froth. "Cigarette burn-like" ulcers are seen in the abomasal folds. Neither pneumonia nor inflammation of the pleura is present.

Ephemeral fever: In most cases this is a self-limiting disease of short duration; most affected cattle recover quickly, even those which are severely affected. The fever fluctuates with two or more peaks. Pneumonia is not a main feature of the

disease but a secondary pneumonia can occur with lung oedema and emphysema in a small proportion of cases. Confusion with CBPP arises from the presence of fever, discharges from the eyes and dripping of saliva from the mouth, lameness and swollen joints (but in animals of all ages, unlike CBPP).

Abscesses: They can be mistaken for sequestra. When cut open the content of abscesses is often offensive smelling, consisting of liquid purulent material. In abscesses a total destruction of the lung tissue occurs. Old, thickly encapsulated hydatid cysts can also cause some confusion.

Tuberculosis: Tubercular nodules can superficially resemble sequestra but they are degenerative cheese-like lesions, sometimes calcified. The lung tissue is destroyed and the same lesions are also seen in lymph nodes in the chest. The capsule of the tubercular nodules is not well defined when compared to that of sequestra.

Farcy: The lung lesions of farcy differ from sequestra as they are filled with foul smelling purulent material, as described for abscesses. Similar lymph node lesions are always present.

Actinobacillosis: The pulmonary lesions, when found, could be mistaken for sequestra. Lesions are generalized and seldom present in lungs alone.

Echinococcal (hydatid) cysts: These cysts have a double wall and contain a clear liquid, often calcified when old.

Foreign body reticulum pericarditis: Clinically similar to CBPP because of the dyspnoea associated with the disease. Only one animal is usually affected.

DIAGNOSIS

Laboratory confirmation

The presence of the disease can be detected in two ways: detection of the causal organism in affected tissue, and detection of serum antibodies to the organism.

The causal organism, *MmmSC*, can be demonstrated in the fluid present in the chest and in diseased lung by culture, by antigen detection tests (interface precipitin test or agar gel immunodiffusion test) and by a polymerase chain reaction (PCR) test.

The rapid slide agglutination test using whole blood or serum for antibody detection can be a useful test to detect infected herds; it can be used in the field to give rapid results. It is performed by mixing a drop of a suspension of killed and stained *MmmSC* organisms with a drop of serum or blood on a glass slide. In a positive result, aggregates form within one minute.

At present, the laboratory test of choice for detecting serum antibodies is the complement fixation test (CFT). Great care is needed in collecting and storing sera to be used for this test. The competitive ELISA test is equally sensitive and useful as a herd test. Histopathology of affected lung fixed in 10% formalin can also help in confirming the diagnosis. The peroxidase-antiperoxidase (PAP) test is an excellent test.

Samples required for laboratory testing

Samples required for successful confirmation of CBPP are chest fluid, diseased lung, and regional lymph nodes, kept on ice during transportation to the laboratory. Additional samples may be fixed in 10% formalin solution for histopathology.

Serum used for antibody tests is obtained by allowing blood to clot at room temperature and collecting the clear liquid when the clot contracts. This usually takes a few hours and during this time the blood samples should be kept at ambient temperature. Separated sera should be kept on ice, and transported quickly to the laboratory.

Interpretation of laboratory results

Culture and antigen detection tests provide conclusive confirmation of CBPP diagnosis.

It is important to note that *Pasteurella* spp. bacteria can frequently be cultured from any pneumonic lung (and even from normal lung). Thus, their isolation does not indicate a diagnosis of pasteurellosis nor does it rule out the diagnosis of CBPP.

The slide agglutination test can give false-positive results in uninfected animals, and also antibodies become undetectable by this test as the disease progresses.

Therefore it can not be used reliably for individual animals.. It is useful in detecting infected herds early in the course of the disease. The CFT is the most reliable test currently available, but it should be noted that false-negative results could be found early and late in the course of the disease.

CONTROL

CBPP control is achieved by eliminating the whole cattle herd population, i.e. stamping out, wherever the disease is detected. However, this may not prove realistic, and quarantine coupled with vaccination is the most frequently used CBPP control measure. To be effective, vaccination must target 100 percent of cattle within an epidemiological and geographically defined area. Vaccination must be repeated, initially at short intervals and thereafter annually over several years, i.e. not less than three to five years. Such vaccination must be maintained until evidence of CBPP eradication is demonstrated by structured surveillance. Live attenuated vaccines (T₁ strain) are widely used in Africa, and only those with quality assurance certification should be used. Vaccine quality control is, therefore, an essential component of CBPP control programmes.

SOURCES OF ASSISTANCE

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COLOUR PLATES



Plate 1. Clinical case of CBPP

This animal is showing difficulty in breathing. It stands with its head and neck extended and legs placed widely apart. Often the elbows are turned out.

A decrease in healthy lung mass and inflammation of the membranes surrounding the lungs causes pain in the chest, resulting in pronounced abdominal breathing movements. Poor general condition.

(Photograph courtesy of DAHP, Botswana)

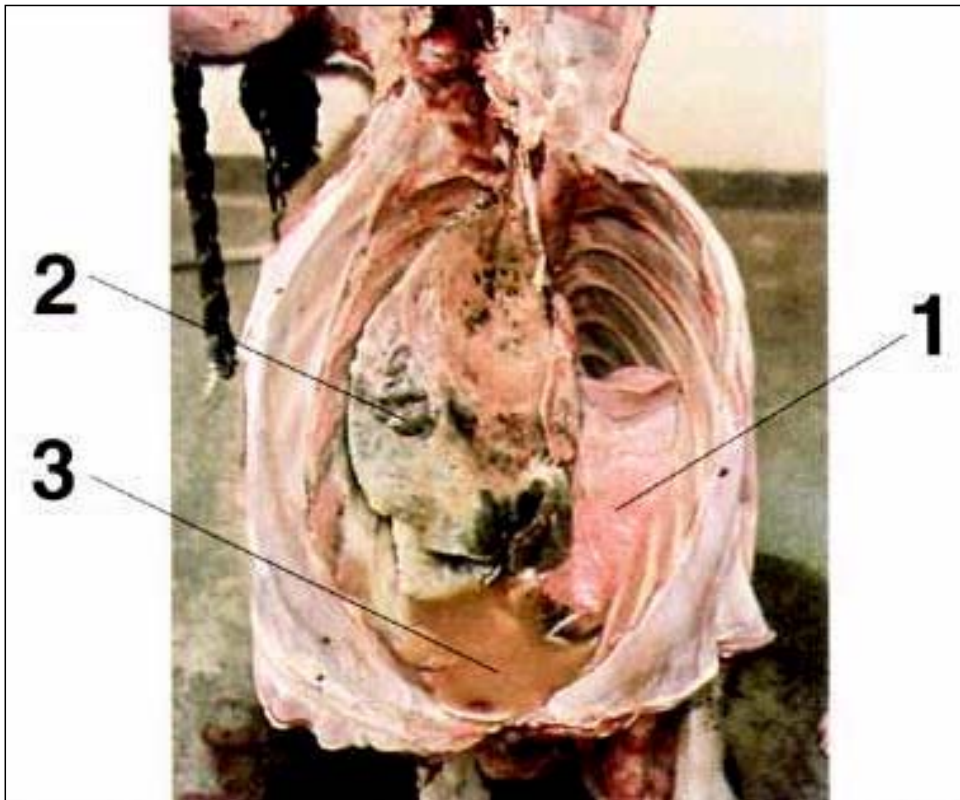


Plate 2. Early stages of CBPP abnormalities in the chest

The diaphragm has been removed to allow a better view.

The right lung (1) has a normal pink colour and has collapsed as the chest was opened. The left lung (2) has not collapsed; it is firm, discoloured and fleshy. It is coated with a yellow fibrin deposits, which are also present on the inside of the ribs. Often parts of the lungs adhere to the chest wall.

Also visible are the remains of the large amount of yellowish fluid (3), which was present in the chest. Here, it is coloured pink by blood from the carcass.

(Photograph courtesy of R. Windsor)



Plate 3. Swollen joints of a calf with acute CBPP

Swollen joints can occur with CBPP infection and are usually found in younger stock, that can be lame and in pain. If a post mortem were to be performed, the joint fluid might be watery, slightly cloudy or even have flecks of yellow-white material floating in it.

(Photograph courtesy of R. Windsor)



Plate 4. Early stage of CBPP, with fluid in the chest

The chest cavity has been opened to show the large volume of yellowish fluid due to CBPP infection. If the volume is great, it can be sufficient to interfere with breathing and be the cause of death.

(Photograph courtesy of DAHP, Botswana)



Plate 5. The lungs in early CBPP

The lung lobe at the bottom of the photograph is normal, and collapsed when the chest was opened. The affected lung at the top is firm and fleshy, liver-like, and did not collapse. Clotted fibrin is visible on its surface.

(Photograph courtesy of IZST, Italy)



Plate 6. Characteristic post mortem appearance

Part of diaphragm cut away to show heavy fibrin deposits (“omelette-like”) on lungs and yellowish pleural fluid in chest cavity. There are adhesions of the lungs to the chest wall.

(Photograph courtesy of the University of Pretoria, Republic of South Africa)



Plate 7. CBPP marbled lung

Lung from an animal that died of CBPP. The hardened lung has been cut open to reveal the marbled appearance of the fleshy and diseased areas. A network of pale bands separates areas of dark red lung. This is very typical of CBPP. *(Photograph courtesy of DAHP, Botswana)*



Plate 8. Early CBPP: Marbled lung

Early stages of marbling.

Close-up view of a section of lung tissue of an animal that died from CBPP. The lung would feel very firm. Note the different colorations separated by white bands, known as interlobular septae. The overall mottled appearance is referred to as “marbling”.

(Photograph taken from CBPP CD-ROM, courtesy of University of Pretoria, Republic of South Africa)

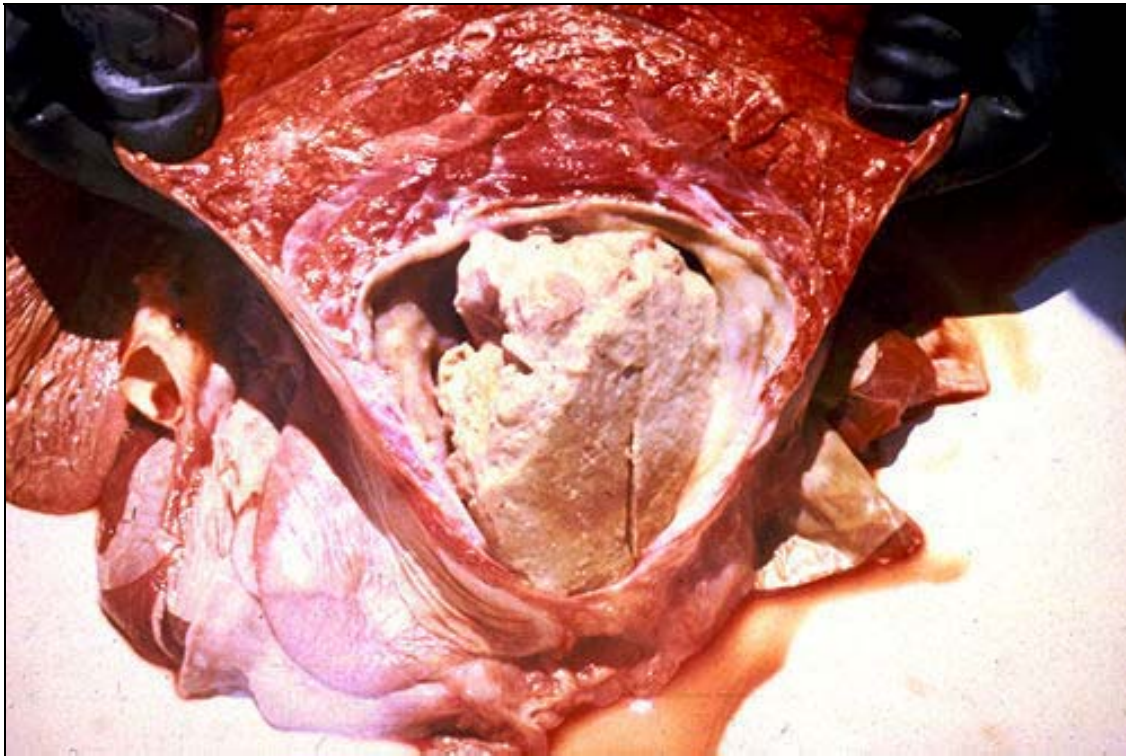


Plate 9. Chronic CBPP with sequestra

The dead lung tissue has changed into a solid cheese-like material, which is encapsulated. Many recovered cattle have one or more of these *sequestra* in their lungs. Chronic cases of CBPP often have such lesions.

This is a typical lesion of CBPP to be looked for during meat inspection.

(Photograph courtesy of R. Windsor)



Plate 10. Early case of CBPP: kidney infarcts

Areas of dead tissue on the kidney surface (called ‘infarcts’) clearly stand out as white spots against the background of the red, normal kidney tissue.

(Photograph courtesy of IZST, Italy)