Manifestation and epidemiology of contagious bovine pleuropneumonia in Africa

W.N. MASIGA*, J. DOMENECH** and R.S. WINDSOR***

Summary: Contagious bovine pleuropneumonia (CBPP) is one of the major threats to cattle health and production in Africa. This article reviews the clinical manifestations, lesions and epidemiology of the disease. The clinical manifestations and lesions are typical and are no different in Africa from those seen in other countries. CBPP is a respiratory disease characterised by pneumonia and serofibrinous pleurisy. The usual form of this disease is acute but chronic forms are frequent, particularly in endemic regions. Hyperacute forms, with a high mortality rate, can be seen at the beginning of outbreaks in newly infected regions.

The epidemiology of the disease in Africa is dominated by four factors, namely: cattle are the only species affected, there is no reservoir in wild animals, clinical cases or chronic carriers are the usual sources of infection, through direct contact, and cattle movements play a very important role in the maintenance and extension of the disease.

CBPP is widespread in Africa and, according to the Office International des Epizooties and to various reports in 1995, the disease is present in 24 countries of tropical Africa. In western Africa, CBPP is mainly enzootic or sporadic but in some countries the incidence is increasing. The situation in Central Africa is not very alarming. However, in eastern and south-eastern Africa, CBPP has become a major issue, placing southern Africa under direct threat.

An evaluation of economic losses due to the disease and the cost-benefit ratio of control programmes is indispensable, since such economic assessments are needed before policy-makers decide on programmes of control or eradication. This is an area which needs to be addressed immediately, as the launching of new campaigns, particularly in eastern and southern Africa, is urgently needed.

INTRODUCTION

Contagious bovine pleuropneumonia (CBPP), which is caused by *Mycoplasma mycoides* subsp. *mycoides* SC (small colony, bovine biotype), is one of the major constraints to cattle-raising and trade in Africa and in other parts of the world. Until the 16th century, CBPP was restricted to the alpine region of Europe, from where it spread to the west and south of the continent, as a result of cattle movements caused by wars and importations. The disease spread to the United States of America (USA) in the second half of the 19th century. CBPP spread to Australia through cattle imported from England in 1858, and from Australia the infection was taken to Asia at the beginning of the 20th century. By the end of the 19th century, CBPP had been eradicated from most of Europe, but was still present in Germany and Austria in the 1910s and 1920s. CBPP has never been totally eliminated from the Iberian peninsula. Australia eradicated the disease in 1973 after an intensive campaign of more than 10 years.

The disease was introduced into South Africa in 1854 through importations of cattle from the Netherlands, and from there it spread to other countries in the region. However, Zimbabwe eradicated the disease in 1904, South Africa did so in 1924 and Botswana completed eradication in 1939. Namibia and Angola have remained infected to this day, and the infection was reintroduced into Botswana in 1994. North African countries have been infected only on a sporadic basis, the most recent being Egypt in 1972. However, Egypt rapidly eradicated the disease. The origin of the disease in Central, West and East Africa is obscure. It has been suggested that the infection was introduced by zebu cattle when they first migrated to the African continent (A. Provost, personal communication). There is a strong possibility that CBPP was introduced into East Africa from India by the army of Field Marshal Napier when he invaded Ethiopia in 1867-1868.

Today, CBPP is widespread in Africa and also occurs in other parts of the world, particularly southern Europe (Spain and Portugal), parts of Asia (India and Bangladesh) and the Middle East (Kuwait).

Thanks to the Joint Project 16 Campaign for the control of CBPP in the 1960s and 1970s, CBPP almost disappeared from the African continent. As with rinderpest, so with CBPP: the mass vaccination campaigns did not completely succeed and the diseases were not eradicated, once more becoming endemic throughout Africa in the 1980s. On this occasion, mass vaccination campaigns have not managed to control the epidemic of CBPP.

The epidemiology of CBPP in Africa has several facets which are not seen elsewhere, e.g. transhumance, nomadism and the trekking of trade cattle. Other problems include the difficulty of implementing control measures because of economic weakness, political disturbances and governmental inability or lack of will to enforce movement controls. These difficulties are aggravated by the lack of diagnostic tools and an efficient epidemiological surveillance network. The true situation in regard to this disease is therefore often not appreciated, making it very difficult to take decisions on control measures.

CLINICAL SIGNS AND LESIONS

Incubation period

There is considerable dispute about the incubation period of CBPP. Animals which have been experimentally infected can show signs of the disease after three weeks.
However, the normal incubation period in naturally infected animals is six weeks, rarely shorter but often longer. Moreover, with these long periods of incubation, it is impossible to ascertain when infection occurred. The long incubation periods recorded in the literature, occasionally up to six months, may reflect the spread of disease from inapparently infected animals.

**Affected species**

*Bos taurus* and *Bos indicus* are both equally susceptible to CBPP (25). Most other animals are resistant. Australian water buffalo can be infected by artificial means but the African buffalo (*Syncerus caffer*) is refractory.

**Clinical signs**

CBPP was probably known to the ancient world but an accurate description of the disease was not made until 1765 by Bourgelat. Since then, the clinical manifestations and lesions have been described in great detail in many publications, with no new information on the subject being published in the recent past (1, 3, 17, 24, 41). This section will therefore simply summarise the principal characteristics of the signs and lesions of CBPP.

CBPP is a respiratory disease, characterised by pneumonia and serofibrinous pleurisy. In an affected herd, CBPP can be seen in hyperacute, acute, subacute or chronic forms. Symptomless cases and cases in animals with a history of non-specific pneumonia also occur. Diagnosis of CBPP in a single animal may be difficult, and it is often advisable to examine many sick animals in an outbreak. In endemic regions, 13% of cases are of the hyperacute form, 20% of the acute form and 46% of the subacute form (3). Approximately 21% of animals are resistant to the disease (3).

**Hyperacute form**

CBPP may be rapidly fatal with no clinical signs observed. This form occurs during the onset of an outbreak and death may be all that is seen. In some cases the animal may die after one to three days with no signs of pneumonia. Death may result from asphyxia, toxaemia or heart failure.

**Acute form**

This is the form that is usually observed, particularly in the early stages of an outbreak. The first abnormality noted may be an increase in body temperature and moderate respiratory signs, such as polypnoea and a dry, painful, irregular cough. The posture of the animal may be stiff.

After a few days, the temperature rises to 40°C or higher, accompanied by a fall in milk yield (in cows), anorexia and cessation of ruminations. At this stage, chest pain is evident. Affected animals are reluctant to move, and stand with the elbows abducted and the back arched, the head extended and the nostrils dilated. Breathing becomes short and rapid and a moist cough may be present. In the severe form of the disease, the mouth remains wide open and may contain foam. Mucoid discharge from the nostrils may occur. Exercise will aggravate the respiratory distress. In rare cases, diarrhoea occurs.
Auscultation reveals pleuritic friction sounds during the acute inflammatory phase and then, during the later stages, fluid sounds or moist gurgling rales are observed. Some areas of silence may be found, caused by consolidation of the lung, or the formation of pleural liquid can be detected. Complications such as arthritis, pericarditis, peritonitis and abortion may occur.

**Subacute and symptomless forms**

Subacute and symptomless forms of CBPP are very frequent and are characterised by mild signs, or no clinical signs at all. It is known that animals with these forms are able to transmit the infection and so it is possible that they are the most dangerous of all.

**Chronic form**

The chronic form is very common and can evolve from the acute form. The affected animals might show unspectacular signs, with mild respiratory distress on exercise, but they can also exhibit a violent and prolonged cough. The animal may remain in poor condition for a long period, depending on the size of the chronic lung lesion. Fever is intermittent and the temperature is never high. An animal with a sequestrum may be normal in appearance. The evolution into chronic forms is more common towards the end of an epidemic.

**Courses of the various forms of the disease**

Animals with hyperacute disease will, in a great majority of cases, die. Those that survive will develop acute lesions which may in turn become sequestra. The mortality rate in the acute form can reach 50% but varies, according to the severity of the disease. Many cases will develop into the chronic form but complete recovery is not unknown. Chronic lung lesions may develop and become encapsulated: these are the so-called sequestra and their sizes vary from pea size to 30 cm in diameter or more. Such animals will continue to show clinical signs of disease for some time. Both acute and chronic cases can recover completely, and scar or even normal tissue may result. The convalescence may be protracted and the lung and pleural lesions may remain for a long time.

It is worth repeating that approximately 21% of animals are resistant to the disease and that almost 50% of animals develop subclinical disease. It is believed, although there is no published evidence, that antibiotic treatment of acute cases will increase the number of animals that develop subacute or chronic forms of the disease.

**Willems’s reaction**

The Willems’s reaction is named after the Belgian veterinarian who first described it in the mid-19th century. This is a reaction of the subcutaneous connective tissue caused by the local inoculation of virulent strains of *M. mycoides* or of certain vaccine strains with residual pathogenicity, such as the T₂ or V₃ strains, or even the T₁/44 strain. Willems’s reactions do not give rise to the natural or typical form of CBPP. The oedema which develops seven to ten days after inoculation is often very painful and extensive, but most of these lesions will regress spontaneously.
If the T₁/44 strain is inoculated into the tail, as in East Africa during earlier vaccination campaigns, about 1% of zebu cattle develop reactions. After subcutaneous inoculation behind the shoulder, 3% to 4% of zebu develop a Willems’s reaction. These reactions occur more frequently and are more severe in *Bos taurus* than in *Bos indicus* and up to 25% of the animals may develop a reaction after caudal or prescapular inoculations. During vaccination campaigns, tail inoculation can lead to necrosis of the tail with loss of the tip. Following prescapular inoculation, extensive necrosis may occur at the site of inoculation. These reactions are unpredictable when animals are vaccinated for the first time. Fatal cases are very rare. Antibiotic treatments approximately two weeks after the vaccination will cure the lesions.

**PATHOGENESIS**

The pathogenesis of CBPP is not, as yet, completely understood and research work is currently in progress. Infection with *M. mycoides* causes bronchiolitis and pneumonia. The organism becomes attached to the surface of the cells and is not susceptible to removal by mucosal secretions. The induction of auto-immune and hypersensitivity reactions seems essential for the development of lesions (23). CBPP is an acute lobar pneumonia, with pleurisy developing after initial septicaemia. An essential part of the pathogenesis of the disease is thrombosis in the pulmonary vessels, prior to the development of the pneumonic lesions. Thrombosis, perivascular organisation and then necrosis are pathognomonic vascular lesions of CBPP. Death results from anoxia and/or toxaemia.

**LESIONS**

The characteristic lesion of the acute form of CBPP is a severe fibrinous pneumonia, with pleural exudate. The volume of pleural liquid varies but can sometimes be several litres.

The lung is adherent to the costal wall, due to the presence of yellow fibrin up to 2-3 cm thick, with an ‘omelette’ look. The pleura overlying affected areas may be thickened and grey to red in colour. Affected areas of the lung have distended interlobular septa due to accumulation of exudate. The lung parenchyma appears pink to dark red in colour, turning to yellow or grey with time. Every lobule seems to be within a frame. This ‘marbled’ appearance of the lung is typical of CBPP. In the early acute phase, necrosis of the lung parenchyma is often seen, surrounded by a forming fibrous capsule. For several weeks, these lesions can become a source of viable mycoplasmas in animals which have clinically recovered from the disease.

In severe cases of CBPP, there may be infarcts in the kidneys. Other lesions which can be seen are hypertrophy of the bronchial lymph nodes, exudative pericarditis and peritonitis. In calves, lesions may also be found in the joints and synovial membranes. Carpal and tarsal joints are more frequently affected. The lesion is a synovitis, with an accumulation of liquid containing coagulated fibrin.

In chronic cases, the necrotic lesion or lesions become encapsulated and one or more sequestra are formed. These can vary in size from 1 to 30 cm in diameter. In most cases, the sequestrum retains the architecture of the acute lesion. Later, the
necrotic material may liquefy or become caseous, enclosed within a fibrous matrix. Some sequestra are located deeply, and can only be detected by careful palpation of the lung. If a sequestrum opens into a bronchiole, this will result in discharge of the causal agent. There is no published evidence that this occurs, but many workers believe that such is the case. Small sequestra may eventually be replaced by fibrotic scars. In this case, all that will be seen is a small fibrous attachment to the thoracic wall and an area of fibrosis in the lung. Large sequestra may persist for many years (49). Adhesions between visceral and parietal pleura are common. When this type of lesion is frequently observed at abattoirs, it may indicate the existence of CBPP in an area where clinical cases are not apparent.

**EPIDEMIOLOGY**

**Aetiology**

Strains of *M. mycoides* vary in virulence from the hypervirulent and velogenic strains, which cause severe epidemics, to the almost non-pathogenic, hypovirulent or lentogenic strains. The virulence seems to decrease as the epidemic progresses. The pathogenicity of the strains declines after passage through embryonated eggs or broth. The resistance of *M. mycoides* to African environmental factors is low and the organism will survive for only two to three days in these tropical regions. Heat sensitivity depends on the medium in which the organism is suspended. In lung fluid, *M. mycoides* is inactivated within 240 min at 40°C, 60 min at 50°C, 5 min at 55°C, and in under 2 min at 60°C. Inactivation is accelerated in normal saline. However, hypertonic solutions have a thermostabilising effect (41).

These characteristics are important in Africa, particularly for the preservation of vaccines. Resistance to other agents, such as cold, radiation, ultrasound, antiseptics or osmotic shock, is low, while cold and lyophilisation are excellent preservatives.

**Hosts**

In Africa, under natural conditions, CBPP affects only cattle, both *Bos taurus* (European) and *Bos indicus* (zebu) types. The domestic buffalo (*Bubalus bubalis*) is susceptible but the African wild buffalo (*Syncerus caffer*) is not. Some serological response may occur in other animal species, such as the gnu (*Gorgon taurinus*), and a case has been described of a Willems’s phenomenon in a roan antelope (*Hippotragus equinus*). It is considered that CBPP has no wild reservoir (40). Camels are resistant to the infection (41).

*M. mycoides* can be isolated from cases of pneumonia in goats and, very rarely, in sheep (2, 21, 22), but the role of small ruminants as a reservoir for CBPP has not been demonstrated. Very rare infections in bison (*Bison bonasus*) and yak (*Bos grunniens*) have been detected in zoos (41).

**Virulent material**

*M. mycoides* is present in the lung and the pleural liquid. If, at the beginning of the infection, the mycoplasmas enter the bloodstream, other organs can be infected, such as the brain, liver, kidney, lymph nodes, uterus, foetal membranes and foetus. Larger numbers of organisms may be found in exhaled air, especially in the acute form of the
disease. If there are kidney lesions, the urine may also be infected and if the foetus is infected, there may be a large number of organisms in the foetal fluids.

Source of contagion

Clinical cases are responsible for the spread of the infection, although some authorities believe that 'lungers', that is, chronic carriers, may break down and shed organisms into the bronchus and then into the environment (41). The main source of infection under natural conditions is the excretion of Flugge-type droplets by the coughing animal. Urine, foetal fluids and even nasal discharge of sick animals can present sources of contagion.

Newly infected cattle can harbour the mycoplasma in the pharyngeal region and act as a source of infection (41).

Methods of transmission

In the majority of cases, infection requires inhalation of infected droplets emitted by coughing animals which are suffering from acute or subacute forms of the disease.

In general, the contagion needs immediate direct contact or contact over short distances. But transmission of the Flugge-type droplets over distances of 50 m to 200 m, transported by an air current, have also been recorded, as well as contamination through urine micro-droplets. Infection of cattle through fomites and contaminated fodder has also been observed (26, 47, 48).

Susceptibility

The first susceptibility factor to be considered is species. As has already been stated, cattle and buffalo are the only species which are affected under natural conditions. In general, it seems that taurine cattle are more susceptible than Bos indicus, at least in Africa. The small Côte d’Ivoire zebu breed and the Masai breed of Tanzania are resistant. N’Dama cattle of Guinea and imported cattle from Europe are more susceptible than the zebu (41).

There are also variations between breeds in regard to susceptibility to the vaccines. For example, N’Dama cattle from Guinea or Côte d’Ivoire usually develop a higher percentage of post-vaccinal Willems’s reactions with the T1/44 strain than zebu or other cattle breeds (41). Hudson demonstrated that dairy breeds of European cattle were more susceptible than beef breeds (17).

Age plays a significant role in susceptibility to the disease (27). Adult cows are more susceptible than calves, which tend to develop joint lesions rather than pulmonary lesions.

During the course of an epizootic in a newly infected area, 10% to 30% of the population will be fully resistant and 25% will have an inapparent infection. Not only are there individual, group and breed variations in susceptibility to the disease, but vaccine reactions show a comparable severity, and so any new vaccine must be tested on the group of animals on which it will be used.

Intercurrent diseases can also increase the susceptibility of animals to CBPP. Those animals which recover from the disease are resistant to further challenge (49).
Cattle movements are responsible for the transmission of CBPP from one herd, region or country to another. Hence, the type of husbandry employed plays an important role in the epidemiology of the disease (8, 18, 28).

In parts of Sahelian and Sudano-Sahelian Africa (although not in Central or southern Africa), due to the arid conditions, nomadism and transhumance are a common way of life (Fig. 1) and controlling cattle movements is very difficult. Some countries, such as Guinea, have attempted to set up strong disease control barriers between endemic and CBPP-free regions, but with poor results. In countries such as Zambia and Botswana, where there are no nomadic peoples, movement of cattle for trade purposes is controlled by the erection of fences.

The movement of trade cattle by trekking is also one of the most important risk factors. These movements are widespread in western and Central Africa (Fig. 2), and have recently been implicated in the spread of disease within Tanzania. When animals are grouped together, e.g. herds often mix at watering points, in market places or when they are confined at night, then these conditions are favourable for transmission.

When animals are moved, or new stock are introduced, CBPP, which may be at a low intensity of infection, can become evident. An example is when a farmer or breeder contracts a pastoralist to look after cattle. If the disease is present among the nomadic herd, the newly introduced animals could become infected and reactivate disease within this nomadic herd.

On return to their own herd, such cattle may well bring the newly acquired infection with them. This can make interpretation of the epidemiology very difficult, as virtually no disease may be evident among pastoralist cattle while, in the nearby settled areas, severe outbreaks occur. The political situation in affected countries is another important risk factor. As a result of civil wars, or unrest and insecurity, unexpected movements of cattle occur, with the possibility of transmitting the disease from one country to another. The case of Rwanda is the most recent and dramatic example of this situation (see below), and illustrates the difficulty of controlling cattle movements in some areas of Africa. Namibia was unable to eradicate CBPP because of the regular incursion of infected cattle from Angola, as a result of the war in that country.

Since infected droplets can be inactivated by ultraviolet light, climate may play a part. The fact that the disease is less prevalent in dry climates than in humid ones confirms this suggestion (41). In fact, climate is not directly important to the disease itself but has a crucial effect on the type of husbandry practised, which in turn affects the epidemiology of the disease. In conclusion, it can be said that the epidemiology of CBPP in Africa is dominated by four factors, as follows:

- cattle and buffalo are the only species to be affected naturally
- there is no reservoir of infection in wild animals
- clinical cases are the major source of contagion, requiring direct contact between infected and clean cattle
- cattle movements are responsible for the maintenance and spread of the disease.
FIG. 1

Routes of cattle transhumance and nomadism in West Africa
(18)
Movements of trade cattle in West Africa
GEOGRAPHICAL DISTRIBUTION IN AFRICA

According to information published by the Office International des Epizooties (OIE) (34) and Food and Agriculture Organisation (FAO), and to various country reports and communications, CBPP was present in 24 countries in Africa in 1995, namely: Angola, Benin, Botswana, Burkina Faso, Cameroon, Chad, Côte d'Ivoire, Eritrea, Ethiopia, Ghana, Guinea, Kenya, Mali, Mauritania, Namibia, Niger, Nigeria, Rwanda, Somalia, Sudan, Tanzania, Togo, Uganda and Zaire.

The situation in Burundi is unclear. Senegal and Sierra Leone declared themselves free from the disease in 1992.

This list shows that CBPP is absent from North Africa and most of southern Africa, but that it is of great importance in tropical African countries (3, 9). The incidence and economic importance of the disease vary from country to country.

West Africa

Here the disease is mainly enzootic or sporadic, but in some countries, such as Mali or Niger, CBPP is on the increase. Mauritania experienced new outbreaks in late 1995. In Côte d'Ivoire, the disease almost disappeared after three years of massive vaccination campaigns (1990 to 1994). However, there was a decrease in vaccination during the following years, and this, together with a steady introduction of infected cattle from Mali and Burkina Faso, resulted in an increased number of outbreaks in 1995. CBPP has been endemic in the eastern part of Guinea for many years and several incomplete vaccination campaigns have failed to eliminate the disease (5, 6). A disease control barrier was set up between the eastern and western parts of the country and cattle movements were prohibited. The only movement allowed was the transportation of trade cattle to the abattoirs by lorry. Unfortunately, illegal movements of cattle resulted in CBPP crossing the disease control barrier and in 1995 three outbreaks appeared in the CBPP-free area (Fig. 3) (38). It is thought that, as a result of wars in Liberia and Sierra Leone, cattle movement has been redirected from the eastern to the western part of Guinea. Cattle from the eastern part of Guinea, which were previously exported to Liberia and Sierra Leone, are now exported from the west instead of from the east or south (Fig. 3) (38).

CBPP has occurred in northern Nigeria for many years and the OIE Annual Reports show a fluctuating number of outbreaks from year to year. By 1965, CBPP was under control but, following relaxation of controls, the disease recurred (32). As part of the international campaign for the eradication of CBPP in West and Central Africa (Joint Project 28 or JP28), Nigeria launched a vaccination campaign in 1974 and the number of outbreaks subsequently decreased. By the end of the 1980s, CBPP had again become endemic in Nigeria and remains so in the 1990s with campaigns conducted annually in various parts of the country (32). The outbreaks now tend to be concentrated in specific areas, e.g. along the Benue river, in the north-east part of the country bordering Cameroon, along the Sokoto and Hadejia rivers in the northern region and along the river Niger in the north-west region (Fig. 4) (30). These regions happen to be grazing areas during the dry season and receive large numbers of nomadic cattle. As the control policies for CBPP at the national borders are inadequate, the disease may spread to neighbouring countries or be imported. Movements of cattle between Niger and Nigeria in the east and north, and between Cameroon and Nigeria...
Cattle movements in Guinea

in the west and north-west, have been responsible for the spread of CBPP (Fig. 1) (41). Nigeria is the biggest importer of cattle in the region: because of the size of the human population, Nigeria is the largest consumer market (Fig. 2). For this reason, the movements of trade cattle have been well mapped. The arrival of large numbers of these trade cattle is a particularly important factor in understanding the spread of CBPP in Nigeria.

Central Africa

The disease is not widespread in Central Africa. CBPP is rare if not absent in Chad, which has not reported an outbreak for several years. The Central African Republic has been free from CBPP, at least in the western part of the country, since 1975. An outbreak of the disease occurred in 1991 and was promptly eradicated. As a result of repeated vaccination campaigns, CBPP occurs only sporadically in Cameroon.
Contagious bovine pleuropneumonia endemic areas

Cattle movements

East Africa and southern Africa

The spread of CBPP within Tanzania and from Namibia to Botswana has put all of southern Africa at risk. Civil war and strife in the horn of Africa, particularly in Sudan and Ethiopia, have led to a cessation of control programmes and an increase in cattle movements. During the last decade, CBPP has reappeared in Uganda, Kenya and Tanzania and is gradually moving towards countries in the south (Fig. 5) and west (Ituri, north-eastern Zaire) (12, 13).

In Uganda, the disease broke out in Karamoja (in the north-east) in 1962 and remained sporadic until 1979. Vaccination and rigid controls on cattle movement in the 1970s confined the disease to the north-east of the district but in 1979, because of the civil war, the infection spread all over the country, reaching the south-western districts bordering Rwanda and Tanzania (14, 36, 43).
CBPP has been endemic in the north and north-east of Kenya for many years. In late 1989 and early 1990, the infection spread to the southern districts bordering Tanzania. Southern Kenya and northern Tanzania are inhabited mainly by Masai and Kuria pastoralists, who move in and out of the two countries seasonally and purchase livestock from both sides of the border. As a result of such movements, CBPP was reintroduced into the Ngorongoro district of Tanzania, after an absence of 30 years. The disease soon spread because of cattle rustling in the Mara region. In 1992, other outbreaks appeared in the Kagera region, north-west Tanzania, as a result of disease spread from southern Uganda. From 1990 to 1994, the disease was confined to the north of Tanzania but, in 1994, CBPP was introduced into the south of the country.
This was a direct consequence of the failure of controls over livestock movement and delays in the detection and reporting of disease outbreaks (46). Inadequate vaccination coverage, possibly associated with an ineffective vaccine, may also have played a part. Tarime cattle from the Mara region are reported to be resistant to tick-borne diseases and are therefore highly marketable in the southern regions: this trade was undoubtedly one of the major factors in the spread of the disease to the south. The status of CBPP in Tanzania in 1995 is shown in Figure 6, which demonstrates how the spread of the disease correlates with cattle movements (15, 20, 35). This spread to the south of Tanzania (for the first time in the history of that country) puts Malawi, Mozambique, Zambia and Zimbabwe at high risk.

After the civil disturbances and political changes which occurred in Rwanda in 1994, thousands of Watutsi exiles who had settled mostly in the northern part of

![Contagious bovine pleuropneumonia outbreaks in Tanzania in 1995](image)

**FIG. 6**

Contagious bovine pleuropneumonia outbreaks in Tanzania in 1995
Uganda (the Mutara region) returned home (42). These exiles brought back their cattle and, with them, *M. mycoides*: thus, CBPP was reintroduced into Rwanda from southern Uganda (37, 39, 45). A large number of outbreaks were reported, together with high morbidity rates (sometimes as high as 50% to 70%) and mortality rates (between 20% and 50%) (4). As a result of the immigration of herds towards the south and west of Rwanda (Fig. 7), almost all regions of the country are now infected and it is very probable that the disease has spread to northern Burundi (29).

As a result of the rinderpest vaccination campaigns, in which the combined rinderpest/CBPP vaccine was used, and which covered almost the whole of Ethiopia, outbreaks of CBPP became very rare in the highlands between 1990 and 1992. However, the rinderpest control strategy changed, and vaccination campaigns were stopped in regions of central Ethiopia which were free of the disease. After a few years, CBPP was reintroduced through trade in cattle from the lowlands, where the infection had never been fully controlled. Today, CBPP is a major problem for Ethiopian livestock.
South-west Africa

CBPP has been endemic in Angola for many years and has been a constant threat to neighbouring countries. The disease was introduced from South Africa at the end of the 19th century and since then CBPP has remained endemic, particularly south of the 14th parallel. In 1994, after the end of the civil war, cattle movements from the south spread the disease northwards (7).

CBPP was brought to Namibia in 1856 and had spread throughout the whole country by the 1870s. In 1919, the disease was eradicated from the commercial farming area but has remained endemic in the north until the present day. The distribution of the disease is shown in Figure 8. CBPP is endemic in Ovambo district, where infection rates can reach 12% or even higher in the areas bordering Angola. From there, CBPP is occasionally introduced into the Kaokoland. Okavango district is regularly reinfected from Angola or Ovambo. The Caprivi strip has been free of disease since 1938. To maintain this position and to protect the 150,000 cattle in the Eastern Caprivi, the Namibian Government slaughtered all cattle (700 head) in the Western Caprivi (R.S. Windsor, unpublished findings). The current disease status is mainly due to the illegal introduction of cattle from Angola, the return of refugees with their cattle, cattle rustling, and trade movements between Ovambo and adjacent areas (44). In order to prevent any reinfection of the areas south of these northern communal areas, the veterinary cordon fences, which were built primarily for the control of foot and mouth disease, are regularly inspected and controlled by veterinary guards. This ensures that no southward movement of cattle or their products takes place.

Well-maintained fences on the Botswana-Namibia border and military surveillance during the Namibian war of independence prevented cattle movements into Botswana. However, with the end of the war and reduced border controls, cattle smuggling and uncontrolled movements through the border fence resulted in outbreaks of CBPP in the north-west of Botswana in February 1995. Despite the prompt reaction from veterinary authorities with attempted control of cattle movements, the building of fences and the slaughter of infected herds, the disease spread towards the south (Fig. 8) (16). In 1996, the Government of Botswana decided that all 300,000 cattle in Ngamiland should be slaughtered. This has been done and appears to have stopped the outbreak (M.V. Rabhorokwe, personal communication). There is strong evidence that the T1SR vaccine which was used in the infected and adjacent areas did not work, and CBPP has continued to spread among herds, despite a well-managed vaccination campaign and well-handled vaccines. This worrying situation is under investigation and, in the meantime, use of T1/44 strain vaccine has been recommended by the FAO.

The region will always remain under threat of infection from Angola and/or north-east Namibia while CBPP remains endemic in those two countries. It can be concluded that the situation in eastern and southern Africa today is grave (Fig. 9) and that the region needs to develop emergency control programmes.

**EPIDEMIOLOGICAL INDICATORS**

Infection rates within herds vary greatly. Surveys conducted using the complement fixation test (CFT) show infection rates ranging from 1% (in parts of west Africa) to 70% (in northern Rwanda in 1995). The same applies to infection rates between herds.
In northern Rwanda, almost all herds were infected by 1995. The mortality rates are also highly variable. In a new epidemic, mortality can be as high as 50%, whereas in endemic zones mortality is low (< 10%). In fact, cattle owners often slaughter animals suspected to have the disease. The prevalence rate shows the same variation. In newly infected regions, the prevalence can be very high (up to 70%), while in enzootic regions, the prevalence is much lower. The incidence rate, which is the number of new cases in a defined time unit, varies according to the epidemiological position. It will, of course, be higher in epidemic areas.
After stamping out the entire bovine population in the infected zone, the Delegate of Botswana to the OIE declared Botswana 'provisionally free' from contagious bovine pleuropneumonia on 7 January 1997

FIG. 9

Distribution of contagious bovine pleuropneumonia in East and southern Africa

EVOLUTION OF AN EPIDEMIC

In countries which have been infected for some years, CBPP is endemic and only sporadic cases are seen. But when the infection is introduced into a clean area, numerous foci will appear. Many animals will be infected and will develop the acute clinical form of the disease. The mortality rate can be as high as 50%. After some time, the disease will have a less explosive character, the severity of the symptoms will
The epidemic which occurred in Rwanda in 1994 is a perfect example of how the disease can spread like an oil patch in water when it affects a non-infected area. One year later the incidence declined dramatically. The characteristics of each epidemic also vary, according to the pathogenicity of the strain involved.

The epidemiology of CBPP also depends upon the control measures taken by the Veterinary Services. Prompt diagnosis, isolation and stamping out of the outbreaks, followed by strict cattle movement control, will eradicate the disease. Incomplete and irregular vaccinations will lead to the disease becoming endemic. Mass blanket vaccinations over a period of several years, coupled with intensive surveillance, lung examinations at abattoirs and control of cattle movements between areas, will also eradicate the disease. However, this will take more time, and may well involve greater cost. After several years of blanket vaccination alone, the number of outbreaks will be few, but the regular reintroduction of cattle from neighbouring countries, where the disease remains endemic, will maintain the infection and clinical disease will develop again as soon as the vaccination campaigns cease.

EVALUATION OF ECONOMIC LOSSES AND DECISION-MAKING FOR CONTROL STRATEGIES

The economic importance of CBPP is high where the disease is epidemic, but is more difficult to calculate in endemic areas. Losses are caused by mortality, loss of weight, reduced working ability and reduced fertility and growth rate. Other economic constraints are caused by restrictions on cattle trading, quarantine and vaccination campaigns.

The control strategies cannot be properly defined if the epidemiological situation is not entirely understood. The prevalence and incidence of the disease must be known and the different areas classified according to their disease status (19). A good knowledge of cattle movements is also required. Several factors hamper full knowledge of the disease position, such as insufficient investigation and surveillance capacities because of economic constraints at the field level, in the laboratories, and in abattoirs. Other technical problems can also complicate investigations, such as lack of ability or equipment to conduct simple diagnostic tests. Therefore, the crucial prerequisite for designing a control strategy, which includes assessment of the status of the disease in neighbouring countries, remains difficult to achieve. Another problem is the difficulty of evaluating the economic losses caused by the disease and of performing cost-benefit analyses of various control programmes. The difficulties of such economic analyses lie in the problems already mentioned above, but are also due to the lack of precise knowledge about the productivity of livestock, and to the scarcity of specialists in animal health economics. This is an area that requires improvement, because such economic assessments are necessary before policy-makers can make decisions about programmes of CBPP control or eradication (10).

CONCLUSION

CBPP is one of the most important diseases in Africa and can have extremely severe consequences for cattle production. The clinical manifestations and lesions of CBPP in Africa are no different from those seen in the rest of the world. The epidemiology,
however, is quite specific to this continent, where it is highly influenced by economic, political, socio-cultural and agricultural factors. These factors make control measures very difficult to implement. The main features of the epidemiology are the absence of a wild reservoir, the importance of chronic carriers, and the necessity of close direct contact for the transmission of the disease. However, the most important factor of the epidemiology of CBPP in Africa is the type of husbandry employed, that is, the type of cattle movements, particularly transhumance and nomadism in arid and semi-arid areas and cattle trade by trekking.

CBPP is widely distributed in tropical Africa but the importance of the disease varies. CBPP is mostly sporadic or endemic in Central and western Africa, where vaccination campaigns have reduced the number of outbreaks. However, in some countries, such as Guinea, Mali, Niger and Côte d’Ivoire, the disease is still important. CBPP has become of major significance in eastern and southern Africa. The economic impact is high in those countries where the disease is endemic and where, consequently, control programmes are necessary.

In countries where CBPP is sporadic, it is necessary to evaluate the economic losses and calculate cost-benefit analyses of possible control plans, according to the economic priorities of the country, before the decision-makers decide to support such programmes. Without elaborating on the various control methods which have been discussed elsewhere (11), as well as in this special issue of the Scientific and Technical Review, and which have also been reviewed in recent meetings (9, 31, 33), it is worth noting that all means should be used, including vaccination, control of cattle movements and slaughter policies, as well as strong surveillance. Private veterinarians should be involved as often as possible in vaccination, and the use of community-based animal workers could be a help in difficult areas. All these strategies will benefit from strong communication programmes.

* * *


Résumé : La péripneumonie contagieuse bovine (PPCB) représente une des menaces majeures pour la santé et la production bovines en Afrique. Les auteurs passent en revue les manifestations cliniques, les lésions et l’épidémiologie de la maladie. Les signes cliniques et les lésions sont caractéristiques et ne diffèrent pas, en Afrique, de ceux observés dans d’autres régions du monde. La PPCB est une maladie respiratoire qui se caractérise par une pneumonie et une pleurésie sérofibrineuse. L’évolution est le plus souvent aiguë, mais des formes chroniques sont fréquentes, notamment dans les régions d’endémie. Des formes hyperaiguës entraînant un taux de mortalité élevé sont observées en début d’épidémie dans les régions récemment infectées.

L’épidémiologie de la maladie en Afrique est dominée par quatre caractéristiques majeures : seuls les bovins sont affectés ; il n’existe pas de réservoir dans la faune sauvage ; la transmission se fait par contact direct avec un animal cliniquement atteint ou avec un porteur chronique ; les
déplacements des bovins jouent un rôle très important dans la persistance et
la propagation de la maladie.

La PPCB est largement répandue en Afrique ; d’après l’Office international
des épizooties et divers rapports parus en 1995, elle est présente dans 24 pays
d’Afrique tropicale. En Afrique de l’Ouest, elle est essentiellement enzootique
ou sporadique, mais son incidence est en progression dans certains pays. La
situation en Afrique centrale n’est pas trop préoccupante. En revanche, la
PPCB constitue un problème majeur en Afrique de l’Est et du Sud-Est et
menace de s’étendre en Afrique australe.

Avant de mettre en œuvre des programmes de lutte ou d’éradication, il est
indispensable d’évaluer les pertes économiques dues à la PPCB et de
déterminer le rapport coûts/bénéfices des mesures de prophylaxie. Cette
question est d’autant plus cruciale qu’il devient urgent de lancer de nouvelles
campagnes, notamment en Afrique orientale et australe.

Péripneumonie contagieuse bovine – Prophylaxie – Lésions – Mycoplasma
mycoides subsp. mycoides SC – Symptômes.

* * *


Resumen: La perineumonía contagiosa bovina (PCB) constituye una de las
mayores amenazas para la sanidad y la producción de ganado vacuno en
África. Los autores examinan de forma sugunta las manifestaciones clínicas,
lesiones y epidemiología de la enfermedad. Los signos y las lesiones son
característicos y no difieren en África de lo que se observa en otras regiones del
mundo. La PCB es una enfermedad respiratoria que se caracteriza por una
neumonía y una pleuresía serofibrinosa. Su forma más habitual es la aguda,
aunque las formas crónicas son también frecuentes, sobre todo en las regiones
donde la infección es endémica. En zonas infectadas recientemente es posible
observar, en las fases iniciales de los brotes, formas hiperagudas con una tasa
de mortalidad muy elevada.

La epidemiología de la enfermedad en África viene determinada
esencialmente por cuatro factores, a saber: el ganado vacuno es la única
especie afectada; no existe reservorio alguno de la enfermedad entre la fauna
salvaje; los casos clínicos o los portadores crónicos constituyen las fuentes
habituales de infección, que se propagan a través del contacto directo; y los
movimientos del ganado desempeñan un importante papel en el mantenimiento
y dispersión de la enfermedad.

La PCB está muy extendida en África. A tenor de los datos suministrados
por la Oficina Internacional de Epizootias y de diversos informes hechos
públicos en 1995, la enfermedad está presente en 24 países del África tropical.
En África del oeste, la PCB es sobre todo enzootica o esporádica, aunque en
algunos países su incidencia está creciendo. La situación en África central,
por otra parte, no resulta demasiado preocupante. Sin embargo, en África
oriental y suoriental, la PCB se ha convertido en un problema de primer
orden, que amenaza directamente el sur de África.
Resulta indispensable una evaluación de las pérdidas económicas ligadas a la enfermedad, así como una estimación de la relación coste/beneficio de los programas de control, pues las autoridades competentes precisan de este tipo de indicadores económicos para tomar decisiones en materia de control o erradicación. Se trata de una cuestión cuya resolución no admite más demora, en particular en el este y el sur de África, donde la necesidad de iniciar nuevas campañas es apremiante.


* * *

REFERENCES


