Early methods of surveillance and control for contagious bovine pleuropneumonia

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Summary: From the many existing documents on the history of contagious bovine pleuropneumonia, it is possible to describe the practical measures adopted for disease surveillance and control from ancient times until the 19th century.

Surveillance was based on diagnosis, post-mortem examination, animal inoculation and also on knowledge of the conditions under which infection occurred: aetiology, pathogenesis, mode of infection, susceptible species, virulent material, incubation period, etc. The historical facts are assembled and compared, with comments on each of these points.

Control was based upon the application of health control measures or vaccination. A study of these two procedures makes it possible to compare their efficacy and to describe the principal steps in their implementation.

KEYWORDS: Bovines – Contagious bovine pleuropneumonia – Disease control – Disease surveillance – Mycoplasmoses – Veterinary history.

INTRODUCTION

According to Nocard and Leclainche (15), 'contagious bovine pleuropneumonia (CBPP) was not distinguished from other diseases of the chest until the end of the 18th century'. Consequently, it would be inadvisable to attempt to trace the history of this disease further back in time. However, it is possible to retrieve from ancient documents clinical, pathological or epidemiological accounts which indicate that the history of this disease goes back to more distant times.

The aim of this article, as with similar preceding articles (4, 5), is not to rewrite this history, but to examine certain technical and scientific aspects, and to discuss them in the light of current knowledge and attitudes.

The discussion will be confined to an examination of the methods used by different peoples during the course of time, on the one hand to recognise animals affected by CBPP, and on the other to prevent the disease and its spread. This examination will point to similarities and differences which existed in these fields at different periods of history.

Only the early methods of surveillance and control, up to the end of the 19th century, will be dealt with. That was the time when the prevention of animal diseases entered its modern phase, leading to almost universal harmonisation of procedures.

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SURVEILLANCE OF CONTAGIOUS BOVINE PLEUROPNEUMONIA

It is obvious that surveillance of an animal disease requires not only a way of diagnosing this disease, but also knowledge of the conditions under which infection occurs: aetiology, pathogenesis, ways of infection, susceptible species, virulent materials, incubation period, etc. Surveillance also requires a system for notification and for sounding the alert when an epidemic occurs. These various aspects were taken into account for CBPP, and will be discussed in turn.

Diagnosis

This is, of course, principally a matter of clinical diagnosis, but methods used for post-mortem diagnosis and experimental reproduction are included.

Clinical diagnosis

As mentioned in the introduction, clinical descriptions of CBPP before the 18th century must be treated with caution, because this disease could be confused with various other diseases of very different aetiology.

However, many ancient descriptions recall the clinical picture of this disease, according to Curasson, Leclainche, Moulé, Reynal and other authors.

Thus, Moulé believed that the disease described by Aristotle as κραυρος (‘craurus’) could have been CBPP, taking into account the symptoms of inappetence, dyspnoea and fever (15). Leclainche believed that the ignis sacer (‘sacred fire’) of cattle mentioned by the poet Virgil (70-19 BC) was CBPP (13). This was also the opinion of Paulet, who recognised in four lines of Virgil a description of ‘malignant pleuropneumonia in cattle, characterised by coughing of blood and chronic fever’ (18):

Ecce autem duro fumans sub vomere taurus
Concidit et mistum spumis vomit ore cruorem; then:
Solvuntur latera, atque oculos stupor urget inertes
Ad terramque fluent devesco pondere cervix.

(‘Behold, however, a bull spitting vapour under the rough ploughshare of the cart, he collapses and vomits blood mixed with froth... Lean-flanked, his dull eyes are stricken with paralysis and his head bows down towards the ground under its own weight’)

Similarly, Leclainche believed that a phrase by Columella (a Roman writer on agriculture, 1st century AD) describing a ‘phthisis’ of cattle referred to CBPP and not tuberculosis, as proposed by Moulé (13).

Est etiam illa gravis pernicies cum pulmo ulceratur,
Inde tussis et macies et ad ultimum phthisis inradit

(‘But when the lung is affected by this serious illness, then come the coughing, the weight loss and finally the phthisis’)

Paulet mentions an account by the poet Silius Italicus of an epidemic of malignant pleuropneumonia following the siege of Syracuse in 212 BC. This condition, which ended in fatal phthisis, commenced with shivering, dyspnoea and coughing. However,
according to the poet, the disease affected cattle, dogs, human beings and even birds, which raises doubt as to the actual nature of the illness (18).

Much later, in the *Hippiatrica* (4th century AD), there are descriptions of pulmonary diseases, provided notably by Apsyrte, which Leclainche recognised as CBPP. The 'morbus siccus' of cattle described by Vegece, characterised by anorexia followed by progressive wasting, might have been a chronic lung disease, according to Belitz (2).

In regard to other historical texts, it is fairly certain that CBPP was not mentioned again until a thousand years later.

In 1523, the Englishman John Fitzherbert described in his *Boke of Husbandrie* a pleuropneumonia of cattle (longsought), which took a chronic course, with frequent attacks of coughing (up to 20 attacks an hour), leading to wasting and death (13, 23).

In his *Treatise on Agriculture*, published in 1550, Agostino Gallo described the symptoms of a contagious pleuropneumonia of cattle in Italy called *polmonera* (or *male disperato* because of the fatal outcome) (13, 23). In the same period, a French doctor, Charles Estienne, reported this disease of cattle in his book *Praedium Rusticum* (1564), but does not seem to have seen it himself, because he attributed the clinical description to Vegece (13).

The disease was again described, two hundred years later, by Jacob Sch euchzer in Switzerland (1732), under the name of *gangrène volante des poumons* ('galloping gangrene of the lungs'), and then by Bourgelat (1765). It was the latter who finally set out the symptoms of the disease in an exhaustive and clear manner, followed by Alberti de Haller in 1773 and Paulet in 1775 (1, 13, 15, 18).

These accounts emphasise the most important symptoms: fever, anorexia and sluggish rumination, cough and foul odour of the breath. Some less common symptoms are also listed by Haller (diarrhoea, which may be bloody, and a rough coat) and by Paulet (dry tongue, mucopurulent nasal discharge, conjunctivitis). The differential diagnosis from rinderpest and tuberculosis remained difficult, however, and it was Bourgelet who deserves credit for clearly distinguishing CBPP from other ‘putrid fevers’ (15).

These clinical descriptions were completed by Chabert in 1792 in his *Instruction sur la péripneumonie*, and then by various other authors, notably Delafond in 1840 (8, 9; see below).

**Post-mortem diagnosis and lesions**

At necropsy there are certain lesions specific for CBPP: hepatisation of lung lobules (giving the lung a characteristic marbled appearance) and exudative pleurisy accompanied by abundant secretion of pleural fluid and formation of fibrin clots ('omelettes') within this fluid.

Such lesions seem to have been described on many occasions during history.

Of the two diseases of cattle described by Aristotle, Smith recognised foot and mouth disease in the ‘disease of the feet’ and CBPP in the ‘craurus’ disease. The latter was characterised by fever, fatal outcome and ‘when the carcass is opened the lungs are found to be disintegrated’ (23). Similarly, the pulmonary ulceration described by Virgil in cattle dead from phthisis (see above) would have denoted CBPP rather than tuberculosis, according to Leclainche (13). Moulé came to the same conclusion in his reading of two similar descriptions by Columella and Vegece (*Cum pulmones exculcerantur*) (15).
In 1765, Bourgelat emphasised the exudative nature of pleuropneumonia lesions, on which he based a differential diagnosis of pleuropneumonia and 'putrid pulmonary fever' (20). In 1773, Haller published a long list of lesions observed when opening carcasses, supporting the findings of Vitet and Bourgelat. In his introduction he wrote, *In omnibus infectis bobus vaccisque pulmo vitiatus pleurae adhaesit. Non raro pus inter eam membranam et adhaerentem pulmonem fuit*. These findings were repeated in his *Mémoire sur la contagion parmi le bétail*. In his observations, he found that, 'the lungs were always inflamed and adherent to the pleura... in many cows the lungs were gangrenous, while in others they are filled with abscesses, or contain vessels filled with water, sometimes mixed with pus', and, 'it was common to find a yellowish fluid within the thoracic cavity' (1). Later, the descriptions of lesions became more precise from the histological and microscopic aspects. Dieterichs was the first to observe, in 1821, that the marbled appearance of the lungs was due to the special anatomical structure of the bovine lungs, and to the development of interlobular lymphatic tissue (15).

Experimental diagnosis and reproduction of the disease

There are no reports of attempts to reproduce the disease in the ancient literature. The first attempts at reproduction became confused with preventive inoculation of virulent material (see 'Methods of medical prophylaxis', below). The oldest evidence is from the empirical practice of certain African tribes to control CBPP, practised 'from time immemorial' (3, 15). But systematic studies did not commence until the 1760s in Europe, during the epizootic wave which affected Alpine countries, the Netherlands and England. Concerning the last two countries, Haller wrote that, 'all hope has been abandoned of curing the disease, and it is to be mitigated solely by inoculation', but he gave no information on the nature of such inoculation (1). In 1845, experiments to infect cattle by forcible ingestion or inhalation of portions of infected lungs were unsuccessful (15). These experiments were repeated in 1852 by Willems, who developed the inoculation of pleuropneumonic fluid into a vaccination technique (see below).

No experimental diagnosis was undertaken before the isolation of the causal agent, and the only attempted test on live cattle, by intradermal inoculation of lung fluid, failed. This was tried by Siedamgrotzky and Noack in 1892 by analogy with the tuberculin test (15). The complement fixation test currently employed for serological diagnosis was proposed by Schochowsky and Poppe in 1913 (7).

Conditions under which infection occurs

Under this heading are placed all the indications mentioned in historical texts which were used at the time to establish and predict the conditions under which infection with the CBPP agent took place: aetiology, pathogenesis, modes of infection, susceptible species, virulent materials, incubation period, etc.

Aetiology and pathogenesis

The causal agent of CBPP was not isolated and cultured until 1898, by Nocard and his colleagues. Before that date, the disease was attributed to a variety of direct and
indirect causes. In the description of the pandemic which affected various species of animals (‘some generations before the reign of Augustus’, according to Paulet), Virgil described a pleuropneumonia of cattle which he attributed to the suffocating heat of autumn, and also to infection of water and pastures, recorded Paulet (18). Livy offered the same explanation for an epidemic in Syracuse (212 BC), namely, ‘the excessive heat which occurred in Sicily, which corrupted the water drunk by people and animals’ (18).

In his book, Praedium Rusticum, already cited, Charles Estienne believed the cause was a ‘grass poison’ (13), and also ‘pollution of cattle fodder by horse urine, a sanguineous plethora, and lack of air in overcrowded cattle houses’ (23). Haller (1773) expressed no opinion on the cause of the disease, but insisted that it was not just a fever degenerating into pneumonia, ‘as the men of science affirm’: nobis non dubium est, quin lues nostra bovilla a peripneumonia incipiat, inque pulmonis gangraenam aut in tabem progrediatur (‘there is no doubt of the fact that the cattle disease begins with pleuropneumonia and develops into gangrene or disintegration of the lung’) (10). This suggestion was not taken up by others: Chabert (1792) believed the disease was a ‘gangrenous fever’, Sanders and Veith (1810, 1818) referred to a ‘typhoid affection’, Tschelin to ‘pulmonary paralysis’ of external origin and Delafond (1840) to a contagious disease, the origin of which lay in the type of housing, too much feed and chilling (8, 15, 18).

The last-named author was not far from the truth, because he clearly emphasised the risk of infection by contact with virulent materials or infected animals, though without excluding the spontaneous generation of the disease. In 1852, Willems observed in infected lungs ‘small corpuscles’ exhibiting molecular movement, but he was uncertain whether they were primary or the result of disease (27). In 1853, Heusinger attributed an epizootic lung disease of European horned livestock to ‘foul vapours’ released by vegetative fermentation (11).

Towards the end of the 1870s, numerous microbiologists suspected the role of a ‘virus’ or bacterium, and attempted to identify the organism: Sussdorf (1875), Bruylants and Verriest (1880), Pütz (1889), Himmelstoss (1884), Lustig (1885), Poels and Nolen (1886) and Arloing (1885-1896). The last-named isolated a bacillus (Pneumo-bacillus liquefaciens bovis), but was unable to demonstrate involvement in the disease, or to produce a vaccine. It was left to Nocard and Roux (with the collaboration of Borrel, Salimbeni and Dujardin-Baumetz) to demonstrate in 1898 that the disease was caused by a microbe very different from typical bacteria. These workers, inspired by the technique of Metchnikoff for culturing cholera toxin, added pooled pleuropneumonic fluid to broth culture within collodion sacs inserted into the peritoneal cavity of rabbits. After 15 to 20 days the broth had become turbid through the multiplication of minute refractile, motile, proteiform bodies, difficult to stain. These bodies were not cultured in agar until 1900, by Dujardin and his colleagues, who produced the well-known colonies shaped like fried eggs. In the belief that they had cultured a filterable virus for the first time (and succeeded in staining this microorganism in 1910), the name Asterococcus mycoides was given, because of the pseudomycelial filaments and multiple polarities. Orskov described the morphology of this ‘pneumonia virus’ in 1927. Novak added to the general confusion by allocating the agent to the imperfect fungi, proposing the name Mycoplasma for the genus, which is still in use today (20).
Contagiousness and modes of contagion

The contagiousness of CBPP was not always clearly recognised. Authors of antiquity (Aristotle, Columella, Apsyrte, Vegece) made no comment on the transmissible nature of the 'phthises' which they described, but this nature was affirmed by John Fitzherbert (1523), Agostino Gallo (1550), Charles Estienne (1564) and Guiseppe Falcone (1619) (13, 15, 23).

Controversy commenced with the first epidemiological observations and detailed experimental studies. In 1773, Haller categorically stated that, 'we shall commence by discouraging the public from believing that Pulmonie is not a contagious disease, a notion conceived by certain learned persons' (1). However, two years later, Paulet was less definite: 'This annual and familiar disease of our climates... is not currently believed to be contagious, though prudence is needed in case it is' (18). Chabert supported contagiousness in 1792 (suspected in Italy by Brugnone and Toggia), but in Germany between 1819 and 1821 it was believed to be either not contagious (Havemann, Bosanus, Dieterich), or of limited contagiousness during the final stages of a disease which arose spontaneously in response to favourable conditions (Sander and Veith). Nocard and Leclainche stated that 'opinions were divided during a period of twenty years (1820-1840)', and that 'the doctrine of contagion was not adopted in France until about 1840, as a result of the studies of Delafond'. In 1841, the latter stated this either categorically ('the facts have convinced me that pleuropneumonia of cattle is contagious') or more discreetly ('it seems to me to be a contagious disease') (8). In 1852, Willems wrote again to the Belgian Minister of the Interior that he had 'doubts on the actual contagion of the disease' (28).

The modes of contagion (once admitted) were clear, particularly for Delafond. This author regarded cohabitation as the origin of most of the infections, but also 'direct contact with outside air at pasture, or between animals in contact across hedges or fences'. In exceptional cases, the contagion could be spread through the carriage of carcasses by dogs, or when healthy animals sniffed at the viscera of animals which had died of pleuropneumonia (9). Note that, in the 1830s, Clément, Dieterichs and Fodéré reported cases of infection of calves within the uterus. This was confirmed by the observations of Heusinger in 1853: 'a cow with contagious lung disease carried a foetus, the lungs of which were affected by the same disease' (11).

The question of modes of contagion was settled in 1898 by the isolation of the causal agent by Nocard (see above). It remained difficult to reproduce the disease with virulent materials or with the newly isolated mycoplasma, and this difficulty has persisted up to the present day. Looking back, this would explain the hesitation of earlier authors, particularly because they were probably working in an infected environment, in which animals possessed naturally acquired antibodies (20).

Susceptible species

It seems futile to place any value on the assertions of authors of antiquity, who frequently claimed that many species of animals were affected by the same 'pandemic', because they were incapable of imagining diverse causes. This applies to the epidemic of bovine pleuropneumonia reported by Virgil (see above), which was supposed to have 'affected wolves, red deer, fallow deer, snakes and even fish' (18). In 1775, Paulet stated that 'the epidemics of malignant pleuropneumonia' among
human beings, described by Guy de Chauliac (1348), Vierus (1565) and Fracastor, ‘are even more common among animals’ (18).

The observations of other authors were more precise. In 1773, Haller stated that, ‘the contagion which carries off horned stock does not attack pigs, sheep or horses’ (1). In 1852, experiments by Willems on preventive inoculation showed that inoculation of species other than cattle (sheep, goats, pigs, turkeys and fowl) had no effect. A report by Wedernikoff (1893) on the susceptibility of camels in Kirghizia was doubted by Nocard and Leclainche, who also denied the susceptibility of goats, which Galtier claimed to have infected by ingestion of portions of pleuropneumonic lung (15).

As far as human beings are concerned, Willems (28) wrote in 1852 that, ‘virulent fluid introduced beneath the human epidermis produced no adverse effect’, nor did cutting the hand of his assistant with a knife coated with virulent material!

In 1873, Reynal established the scientific basis for precautions against CBPP, stating that there ‘is no reason to forbid the eating of affected animals... because consumption of their meat has never been followed by any adverse effect’ and that ‘in the city of Lille alone, meat from over 18,000 affected cattle has been eaten without in the least upsetting the health of the population’. The same applied to milk drunk by workers at Lille abattoirs over a number of years ‘without the least inconvenience’ (21). This contradicts the assertion of doctors Dupré and Lécuyer that, some time previously, milk from pleuropneumonic cows had caused fatal pneumonia among infants (7). In 1898, Nocard and Leclainche were definite that, ‘small laboratory animals, pigs, horses and carnivores are refractory to pleuropneumonia. Man is exempt from infection’ (15).

Further details were provided subsequently by fortuitous observations (susceptibility of yak, bison and Asian buffalo during an epidemic at Brussels zoo) or experimentally (susceptibility of eland and reindeer). Observations on giraffes, certain antelopes and small domestic ruminants of Africa showed that they were not naturally receptive to the disease (20). Curasson found that zebus vaccinated by the inoculation of virulent lymph died at a higher rate (5%) than cattle which received the same treatment in Europe (1%). However, this was subsequently contradicted (20).

Virulent materials

Commenting on the epizootic described in the verses of Virgil, an epizootic ‘which killed so much livestock in the Julian Alps, along the banks of the Timave and in Bavaria’, Paulet drew attention to ‘the ease with which these diseased cattle infect pastures with their breath, their saliva or other bodily humours’ (18). Comparing ‘pulmonie’ with rinderpest, Haller (1773) believed that ‘the foul breath of the diseased animal adheres to the hair of animals that approached it’ (1).

Delafond conducted a two-year study in north-western France, and in 1841 concluded that ‘bovine pleuropneumonia seems to be a contagious disease’ and that ‘the virulent elements of this dread disease appear to reside in the nasal mucus, saliva, exhaled air and emanations from debris of carcasses’. These conclusions were based on detailed epidemiological facts presented in a report, entitled Recherche sur la contagion de la péripneumonie des bêtes bovines (9). He described cases of infection from portions of carcasses, skins and infected viscera.
Between 1845 and 1850, numerous attempts at experimental infection were made in Europe, with unsatisfactory results. Infection by cohabitation was difficult to reproduce, and incorporation of portions of affected lungs into the feed (or direct introduction into the nostrils), and swabbing the muzzle with pleural fluid had no effect (16). In 1852, following his experiments on vaccination by inoculation of pleuropneumonic fluid, Willems concluded that, ‘pleuropneumonia is not contagious by inoculation of blood or other materials obtained from sick animals’ (27). In 1873, Reynal reported, on the basis of experiments conducted between 1863 and 1869, that cattle ‘ingested portions of affected lungs and many litres of pleural fluid without any effect’ (21).

**Incubation**

There is no precise information on the incubation period of CBPP before clinical identification of the disease in the 18th century.

In 1773, Haller wrote: ‘it is only some days, or even weeks after the animal is infected, that fever develops’ and ‘we have very reliable information that an animal from an infected stable... did not become ill until a month after removal from the infected area’ (1).

These observations were confirmed by Delafond in 1841: ‘Animals are attacked and succumb 15 days after an initial case, or perhaps one month and even two months, seldom longer’. Nonetheless, to protect cattle from pleuropneumonia, he recommended ‘that newly purchased cows should be kept at a separate place for 4-5 months, the usual time required for the germ of the disease to develop’ (9).

In 1873, Reynal provided more precise information as a result of experimental inoculation of virulent materials, and he cited two extremes of incubation period, one of 67 days and the other of 97 days. These were comparable to earlier clinical and epidemiological observations: 60 days according to Verheyen, 90 days according to Gamgee (in the case of an animal transported to Australia from the Netherlands), and 112 days according to Roell (21).

Subsequent work led the Office International des Epizooties to recommend a maximum incubation period of 180 days in the *International Animal Health Code* (17).

**Systems for notification and alert**

In Switzerland, once the contagious nature of CBPP had been established by Haller (1773), it was recommended by that author that ‘any subject who has knowledge of the suspected presence of the disease in any cattle must declare to the nearest State official that which he has heard or seen, and which is dangerous to public safety, or else incur severe punishment.... the same applies to the owner or other inhabitants of the place... The officials must report to the Bailiff, who in turn notifies the Health Council.... Anyone who conceals a suspected case shall be punished according to the gravity of the case, either by imprisonment, or by some other serious penalty’ (1).

Such wise precautions were not adopted in other countries, and in 1840 Delafond warned farmers (of Bray) that ‘they should be wary of cattle sold by dealers... given that such cattle could come from areas where the disease occurs and where no administrative restrictions are in place to forbid the sale of cattle which have lived with, or have been otherwise in contact with, sick animals’ (8).
On 16 June 1857, Professor James Beart Simonds advised the British House of Commons to adopt a law to make the disease notifiable by any farmer who found 'even just one case' among his cattle. Simonds cited the example of veterinarians in Hanover, who were obliged to declare to the health authorities any case of CBPP found among their clients (12). However, it was not until the 1870s that the various legislatures of Europe compelled the owners of affected cattle to notify the health authorities, with a view to organising prophylactic measures (see below).

On 8 February 1872, the Federal Assembly of the Swiss Confederation added CBPP to four other diseases (rinderpest, foot and mouth disease, glanders and rabies) requiring immediate and compulsory notification by owners, veterinarians, police officers, etc.

**CONTROL OF CONTAGIOUS BOVINE PLEUROPNEUMONIA**

As the author has pointed out for other infectious diseases (4, 5), detailed research on their aetiology could not start until the advent of microbiology, but at no stage of history did this prevent the discovery and proposal of effective measures for protecting against epizootic diseases. It is therefore particularly interesting to examine the measures proposed for a disease as old as CBPP.

This account concerns the two classical aspects of prophylaxis (sanitary and medical) for contagious diseases, followed by treatment of the disease.

**Methods of sanitary prophylaxis**

These methods involve hygienic measures aimed at eliminating the causal factor.

*General hygiene*

Few general hygienic precautions were applied to CBPP, if one excludes bleeding, as recommended by Fitzherbert (1523), and the separation of cattle from horses (the urine of which was at one time believed to be infective) recommended by Estienne in 1564 (23).

In 1773, Haller considered as useless and preferred 'not to mention the so-called precautions' fashionable in his time: bleeding, feeding crab-apples, etc. (1). In 1775, Paulet recommended renewing the air of cattle houses, avoiding draughts, covering the animals and giving enemas (18). Delafond (1840) expanded these ideas: correct ventilation of cattle houses, winter rationing for cattle, protection against cold, periodic bleeding and purging (8).

*Restriction of movements of animals*

Such restriction was, of course, recommended by all those who recognised the contagious nature of the disease, notably Fitzherbert (1523) and Gallo (1550), who advised immediate segregation of sick animals (23, 26). In an agricultural treatise published in Venice in 1619, Giuseppe Falcone (a Carmelite friar) stated, 'Polmonera is a very serious disease. Sick animals should be separated quickly from those other animals still healthy, for the disease is as contagious as rinderpest' (13). Haller (1773)
strongly advocated the segregation of sick animals because, ‘above all, cattle from a country where “pulmonie” is prevalent must not be allowed to enter’. Every beast offered for sale or moved (identified by a mark on the horns specific for a given village) must not be admitted to a fair or market without a printed health certificate, signed by the local authority. Haller demanded that inspectors be allocated to fairs and markets in Switzerland, ‘in order to slaughter any beast without certification; and distribute their meat to the poor’ (1).

In 1849, a Belgian circular specified that owners of sick cattle must prevent contact with healthy animals at fairs and markets, a special place being reserved for such cattle (14). In 1857, Simonds recommended that a farmer who takes sick animals to a fair, market or communal pasture in Britain should be fined £20 (12). In the same period, or even earlier, owners of livestock in Dinka (Sudan) chased away from their encampments all owners of herds affected by ‘awuk’, coughing specific for CBPP (3). In 1870, a Dutch Royal Decree stipulated that dogs be segregated during an epidemic of CBPP. In 1872, a Swiss Federal law prohibited the importation of cattle from other countries which were infected.

Quarantine

The first text to recommend actual quarantine for cattle with pleuropneumonia seems to have been that of Haller in 1773. Among the numerous measures which he specified for limiting the spread of the disease, some were applicable to animals on infected farms. ‘The first precaution is prompt separation of any sick or suspect bovine... to be placed in a separate building, where it will be fed and watered within an enclosure.’ In the case of an affected village, ‘all cattle houses shall be carefully sealed and excluded from all communication when watering and grazing’. In the case of cattle from outside (or animals which had come into contact with such cattle), ‘the owner shall be ordered to feed it for 6 weeks on the lower slopes of mountains, isolated and fenced, without mixing with local cattle; such cattle shall be visited every 15 days, and not permitted to travel further until after this quarantine, if circumstances permit’ (1).

In 1830, in France, Grognier advised quarantine for cattle with pleuropneumonia (16). Yvart recognised the efficacy of this precaution in a report published in 1851, which outlines the steps taken in Auvergne: segregation of cattle within an enclosure or in mountain pastures (21). In 1857, this idea was considered by Simonds for cattle imported into England, but there were too many practical difficulties: he believed that there must be a quarantine period of unlimited duration, and this would result in futile mixing of healthy and infected animals (12). However, by the end of the 19th century, many European countries had adopted such precautions for infected cattle. In Switzerland (under the Federal Law of 8 February 1872), infected animals had to be segregated for four to twelve weeks (21). In France (under the Law of 1881 and Decrees of 1882), such cattle were to be placed under surveillance for three months after confirmation of the last case. This duration was four months in Sweden (1887). In Belgium (1883), every bovine coming from an infected region was to be isolated for 15 days before being introduced into fattening premises (15).

Slaughter of infected animals

A logical consequence of the recognition of the infectious nature of CBPP was precautionary slaughter of affected, suspect or in-contact cattle, as recommended by
Haller in 1773. ‘In a severe case, all the cattle in an infected cattle house shall be slaughtered for safety reasons, including those who appear to be healthy, and those which show signs of the disease... Such apparent cruelty is the only way of preventing the contagion from reaching other herds’ (1). The author added that, ‘at times, greater severity has been applied: pigs feeding with the cattle were slaughtered’. Such an energetic policy was adopted in only a few countries. This was the case in the Grand Duchy of Luxembourg, at that time annexed to France, where two French ‘veterinary artists’ (Petit and Peuchet) stated in a report, dated 5 August 1796, ‘should the disease continue to progress, it will be necessary to kill the affected animals’ (26).

However, the contagious nature of the disease was called into question in many countries until the start of the following century, and precautionary slaughter often gave way to simple segregation of affected animals (Delafond, 1840), or preventive inoculation of healthy animals (Willems, 1852). Not until the 1860s did slaughter become the general rule, after many delays, castigated by Nocard and Leclainche, who stated in 1898: ‘a first measure is self-evident: the slaughter of animals found to be infected... It has been demonstrated that slaughter is both economical and effective. Nevertheless, Germany and France continue to foster pleuropneumonia with hesitations both costly and incomprehensible’ (15).

These comments were no doubt inspired by the success achieved in the United States of America (USA), where affected and in-contact cattle were slaughtered (under the Federal Law of 3 March 1887). The USA became infected from European cattle in 1843, and again in 1886, and this was followed by the slaughter of 21,539 cattle. By 26 May 1892, the Secretary of State for Agriculture (a post created specially for the control of pleuropneumonia) was able to announce that the disease had been officially eradicated from American territory (15).

Eventually the slaughter of affected cattle, and very often of in-contact and suspect cattle as well, became compulsory in the Netherlands (1870), Switzerland (1872), France and Romania (1882), Norway (1886), Algeria, Belgium and Sweden (1887), the United Kingdom (1888), Austria (1892) and Denmark and Hungary (1893). All these countries provided compensation for the owners of slaughtered cattle, equivalent to at least half their market value. In the United Kingdom, 30,000 cattle (5,000 affected and 25,000 in-contact) were slaughtered between 1888 and 1890, and the disease was progressively eradicated from that country (15).

**Destruction of virulent materials**

For contagious pleuropneumonia, Gallo (1550) recommended the disinfection of feed for healthy cattle by washing in boiling water plus fragrant herbs (26). Estienne (1564), no doubt inspired by Gallo, stated: ‘the disease is deplorable in beef cattle and cows, that there is no remedy other than to wash out the feeding trough used by affected cattle with hot water and fragrant herbs, before tethering others, which should be kept in separate buildings’ (7). This recommendation was apparently copied by Falcone (1619): ‘Wash the mangers with hot water, clean them with care, and perfume the stable with fragrant herbs’ (13).

Two centuries later, Haller prepared a more extensive list of items to be disinfected: ‘the vessel from which the beasts drink... the manger, which shall be burned with all other wooden objects... soil of the stable to be removed to the depth of one foot, replaced, and covered with chalk... excreta to be buried in a ditch, well covered with soil and fenced off... while the cattle house should be aerated for at least 3 months’.
If the lungs of a slaughtered animal were free from lesions, the meat and hide could be used, but in other cases the hide ‘shall be cut into strips and buried with the animal in a trench six feet deep, filled with chalk and fenced off’ (1).

In 1796, in their official report to the Grand Duchy of Luxembourg, Petit and Peuchet recommended the burial of slaughtered, affected cattle, together with ‘their hides, seven feet deep, and in a trench situated half a league from the infected place’ (26). In 1840, Delafond issued very precise instructions for ‘disinfection and purification of cattle houses’: scrubbing of walls, mangers and paved floors, then washing with a suspension of wood ash and 1.25% calcium chloride, the burial of contaminated hay, and fumigation of the building with chlorine vapour (8). Scientific study of the efficacy of various treatments did not commence before the 1890s, when Arloing, Nocard and Leclainche demonstrated that the infectious agent was weakened by exposure to air, sunlight and heat. The agent was inactivated by temperatures above $55^\circ$C (16).

**Annulment of sale**

Legally, CBPP was never considered to be a defect which rendered a sale invalid, even though most authors regarded the disease as such because of the contagiousness of the infection (21). In France, a law of 20 May 1838 excluded CBPP from the list of just two diseases regarded as defects annulling a sale (sheep pox and glanders/farcy). By contrast, Article 11 of a Norwegian law of 27 February 1866 stated that ‘if someone sells an animal affected by a contagious disease (including “pulmonie of horned livestock”) the purchaser may cancel the sale, even if the vendor was ignorant of the state of the animal’ (21).

**Methods of medical prophylaxis**

In contrast to other epizootics, CBPP inspired few ancient authors to devise preventive remedies, with the exception of certain hygienic precautions mentioned above. Haller (1773) advised against any treatment, ‘because remedies were recognised as useless against rinderpest, pox and other contagious diseases’ (1). Apart from bleeding, few treatments were proposed, except for ‘the administration of beverages that temper and dilute: saltpetre and honey boiled in two pints of water’, recommended in Luxembourg in 1796 (26).

On the other hand, CBPP joined the poxviral infections as the first diseases against which vaccines were developed. CBPP and rinderpest were the diseases of cattle that received the most extensive research into the preventive inoculation of ‘attenuated’ virulent material, even before the causal agent was known.

Two types of medical prophylaxis were developed, apparently independently: that used empirically by cattle owners in Africa, and that studied and applied by physicians or veterinarians in Europe.

**Immunisation of African cattle**

‘There is published evidence that preventive inoculation against CBPP has been practised for time immemorial in Senegambia and southern Africa’, according to Nocard and Leclainche in 1898 (15). Among the sources cited, the best known is certainly that of de Rochebrune, a French physician who believed he had discovered
a new breed of three-horned cattle in Senegambia in 1880 (22). This new breed was illustrated in a plate published in 1891 in *Traité de zootechnie générale* by Ch. Cornevin (Fig. 1) (6). These animals were, in fact, zebus which had developed a nasal excrescence following subcutaneous insertion in the bridge of the nose of virulent pleuropneumonic material, a primitive method of vaccination practised at that time in Africa. If the owners of these ‘three-horned’ cattle had been questioned, de Rochebrune would have been spared the ridicule with which his report to the Academy of Sciences was later greeted: ‘Naturalists and travellers at all epochs have kept quiet, for reasons that are not clear, about a breed of domestic cattle in Senegambia which is of special interest... A feature which distinguishes it from other breeds is the presence in the nasal region of a genuine horn, identical to ordinary horns in its nature and mode of development. Carried by males and females, this horn is sometimes conical, but most often in the shape of a truncated quadrangular pyramid, reaching an average height of 0.06 m to 0.075 m, 0.055 m wide and 0.04 m thick. Its surfaces are grooved and bear perpendicular depressions, with stratified, horizontal growth bands from base to apex... This phenomenon, which might be judged to be a defect in a single individual, becomes obvious when a large number of animals is examined. In fact, in a typical herd of 100 head one would find 55-60 individuals bearing a well-defined nasal horn. It would be missing from 45-50 others, but these invariably showed a swelling of the nasal region, covered by a thin, rough cornified layer. Obviously, this is a hereditary feature transmitted through successive generations in a long-established

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**FIG. 1**

Illustration published in 1891 in *Traité de zootechnie générale*, by Ch. Cornevin, with the following caption:

‘Reproduction of the skeleton of the head of a Senegalese three-horned bovine

(*Bos tricerus*, A. de Rochebrune)’

(6)
De Rochebrune added: ‘The limitations of this brief account exclude consideration of differential characters; I cannot go into the causes which might have given origin to this remarkable breed. It is sufficient to state that it is scarcely exceptional among the order of ruminants, for the nasal horn of Senegambian zebus is strikingly similar to that of another African species, the giraffe.’

This detailed quotation is not intended to dwell on the confusion of that author, but to provide an interesting insight into post-vaccinal lesions, and the proportion of cattle which reacted.

In fact, this practice was not confined to Senegambia, for various authors have reported it from other African countries: South Africa, Mali, Mauritania, Sudan, Tanzania, Chad (7), Niger, Nigeria, Somalia (20), Ethiopia, Kenya and the Central African Republic (3).

In all these countries, the traditional vaccination procedure, in use before the publication of Willems in 1852 (see below), was to insert virulent material (treated or untreated) beneath the skin. However, there were numerous variants, as shown in Table I.

**TABLE I**

*Procedures used for vaccinating cattle against contagious bovine pleuropneumonia in Africa, before the discovery by Willems of preventive inoculation in 1852*

<table>
<thead>
<tr>
<th>Territory (present name)</th>
<th>Virulent material used</th>
<th>Site of inoculation</th>
<th>Treatment of virulent material</th>
<th>Mode of inoculations</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>South Africa</td>
<td>Pleuropneumonic lymph</td>
<td>Tip of tail</td>
<td>None</td>
<td>Thread saturated with lymph inserted under the skin</td>
<td>(7)</td>
</tr>
<tr>
<td>Gambia, Mali, Nigeria and Senegal</td>
<td>Lung fragment or lymph</td>
<td>Bridge of nose, thigh or tail</td>
<td>Maceration for 3-7 days in milk (bovine or human)</td>
<td>Subcutaneous insertion (incision)</td>
<td>(7)</td>
</tr>
<tr>
<td>Kenya and Mauritania</td>
<td>Lung fragment</td>
<td>Bridge of nose</td>
<td>None</td>
<td>Subcutaneous insertion or scarification of a skin fold</td>
<td>(3, 7)</td>
</tr>
<tr>
<td>Central African Republic and Chad</td>
<td>Lung fragment or ear</td>
<td>Bridge of nose or ear</td>
<td>Maceration in water (with a cube of liver), or in milk or urine</td>
<td>Subcutaneous insertion or oral administration</td>
<td>(7)</td>
</tr>
<tr>
<td>Tanzania</td>
<td>Pleuropneumonic lymph</td>
<td>Bridge of nose</td>
<td>None</td>
<td>Subcutaneous insertion</td>
<td>(7, 20)</td>
</tr>
</tbody>
</table>

Post-vaccinal reactions could be severe (local or general, sometimes resulting in considerable mortality) but the immunity was always solid. This procedure is still in use in Senegal and in some other countries (Fig. 2).
Pronounced local reaction of tissues of the bridge of the nose in a zebu vaccinated against contagious bovine pleuropneumonia by the traditional African method: subcutaneous insertion of a fragment of infected lung

(Photograph taken by Professors J. Chantai and J. Rozier in February 1974 on a ranch in Bambylor, Senegal)

It is certain that the technique used in Africa predates European attempts at inoculation, but it is impossible to determine from written records the precise date of introduction. The earliest travellers to observe the technique in southern and eastern Africa (Baines, Hayes, Mohr) or in western Africa (Monod) could only say that 'it is very ancient, and certainly older than vaccination as advocated by Willems', or that it was 'lost in the mists of time', or 'practised from time immemorial', etc. (7, 15).

Another question is who invented the African vaccination procedure? Curasson provides a good review of possible hypotheses: 'According to Monod, the Moors were guided in their first inoculations, after having tried various treatments, by their custom of inoculating human beings during smallpox epidemics. It is not known which procedure came first, for the Moors and Tuaregs used inoculation into the ear for sheep pox, pleuropneumonia and contagious ecthyma. It was probably first used against pox, for this was reported to have been practised in Senegal by physicians at the start of the 19th century. It seems that variolation was spread by one of the nomad populations (Moors, Tuaregs, Peulhs) during their extensive migrations in Africa, because it had
been known since time immemorial to the white populations from which these nomads derived. They may have extended the procedure to animals, teaching it to other African populations, because indigenous peoples throughout Africa had known of it for a long time’ (7).

These hypotheses are still valid today, and lead one to believe that African procedures for vaccination against CBPP were derived from similar procedures, already in use against caprine pleuropneumonia in Iran ‘for some centuries’, according to Tadjbakhsh (24, 25). In a personal communication, he stated that the method was used by Aryans in Iran eight to ten centuries BC.

Immuneinisation of European cattle

In Europe, the scientific study, development and promotion of preventive inoculation against CBPP is clearly the work of Louis Willems, Doctor of Medicine at Hasselt (Belgium). However, Willems was well aware of earlier attempts at preventive inoculation, which he cited with some condescension: ‘The question of inoculation has not been resolved so far, because the inoculations of Dieterichs to prove contagion were unsuccessful, and the inoculations of Vix produced pneumonia’ (28). In fact, Haller had reported attempts at inoculation made in the Netherlands and England 80 years previously (1), and according to Curasson the Germans also used this method (7).

Willems stated that his research was founded on two facts: the success of inoculation against contagious diseases in human medicine, and the existence of immunity following a previous attack of CBPP. He summarised the procedure which he developed, in the early 1850s, in Mémoire sur la pleuropneumonie épidiozootique du gros bétail addressed to the Belgian Minister for the Interior (28) as follows: ‘I took fluid expressed from the lung of an affected animal recently killed, or from an animal which had died from the disease. By using a type of large lancet dipped into this fluid, I made two or three incisions at the tip of the tail of the animal to be protected from the disease. A single drop of fluid was enough for inoculation.’ Willems used this method to protect 108 cattle on a farm, where CBPP killed 17 of 50 unvaccinated control cattle.

To verify these claims and the results of the first experiments conducted in Europe, many governments formed commissions of inquiry, including Belgium, France, the Netherlands, Italy and Prussia, etc. (7). All confirmed the value of the procedure, despite certain reservations. In 1854, the French Commission (Bouley) concluded, ‘the inoculation has a preservative value capable of protecting animals against infection for a period not yet determined’. In 1855, the Dutch Commission recognised ‘the preservative value of inoculation when performed with appropriate precautions’. The Belgian Commission (Thiernesse) was more reticent, and did not give a definite judgement until 1865, stating: ‘inoculation is an inoffensive operation and generally crowned with success when performed with suitable precautions’. This Commission provided statistics to support such conclusions: 35% mortality without vaccination; 86,149 successful vaccinations against 11,944 failures; 8% to 10% severe post-vaccinal reactions (requiring amputation of the tail) and 1.1% of deaths attributable to vaccination. But two camps had already become established in Belgium, one in favour of vaccination and one against, and the ‘anti-inoculators’ opposed compulsory vaccination in that country (14).

In view of a majority of favourable reports, vaccination extended to many other European territories, usually combined with other preventive measures (see above).
Vaccination then became prohibited during the 20th century, but was still performed in many African and Asian countries, using vaccines prepared from pure cultures of mycoplasma. Such vaccines were first prepared in 1899, reducing post-vaccinal mortality considerably. Numerous other live and inactivated vaccines, produced from cultures or in eggs, were developed until it was decided to use only live mycoplasmas of attenuated virulence derived from strains T₁ or KH₃J to immunise cattle (19).

**Treatment**

According to Paulet, Columella was the first to recommend treatment, at about year 40 AD: ‘place a seton, which has first been passed through a portion of hazel root, in the ear of cattle with “pulmonary phthisis”. At the same time, have it drink a pint of pear juice containing an equal quantity of olive oil and a pint of wine, once daily for several days’ (18).

It may have been this author who inspired Fitzherbert (1523) to recommend bleeding affected cattle, and the insertion, in the dewlap, of a seton soaked in hellebore suspension (23). In the same era, Gallo advised against treatment, because he believed the treatments to be ineffective and preferred to isolate sick animals (27).

In Switzerland, Haller (1773) was very cautious about treatment for pleuropneumonia, mentioning that, ‘in ancient times reliance was placed on setons and hellebore root inserted beneath the skin, but now there is no hope of curing the disease’. Nevertheless, he proceeded to review the ‘treatments recommended as cures for the disease: bleeding, emollients, febrifuges, cinchona, mercury, etc.’, and remarked that ‘people use some poorly chosen compositions and outdated recipes’. He concluded that ‘if the disease is actually “pulmonie”, it is preferable not to treat affected cattle but to slaughter them without delay’ (1).

In France, Paulet (1775) was more optimistic, and devoted three pages to a description of possible treatments in relation to the location and progression of the disease. If coughing was violent, a ‘very good effect’ could be achieved with infusions (flowers of red poppy and violet with honeyed barley) or the provision of ‘chewing blocks’ (treated with figs, honey and distilled rose water). Administration of boluses of liquorice, hound’s tongue, agaric, flowers of sulphur, Florentine iris and spermaceti, etc. was also recommended, but this was often inadequate. If the illness became worse, cinchona, camphor, gum ammoniac and even 1½ drams of powdered woodlice could be given! Enemas and purgatives completed the treatment (18).

In Luxembourg, at the end of the 18th century, pleuropneumonia was treated with setons of mercuric chloride and cantharides powder and perhaps with other additives (such as bugloss, viperine, cinchona, camphor, marshmallow and burdock) in which Thèves identified numerous active principles: expectorants, purifiers, diuretics, cough suppressants, anti-infective agents, etc. However, veterinarians in Luxembourg recognised that it was useless to pursue treatment of severe cases, ‘considering the total decomposition of the machine’! (26).

In Belgium, Willems (1852) tried various new treatments, and claimed to have cured 15 of 23 cases with 2 or 3 drams of ‘black sulphur of mercury’, coupled with ½ dram of calomel incorporated in gum. However, he concluded that, ‘although some cures are effective, they are incapable of obstructing the course of the disease, because those treated cattle which recover do so only with difficulty and slowly’ (27).
In 1873, Reynal also doubted the value of treating affected cattle because, 'after all, there are few specific treatments for pleuropneumonia', with the possible exception of revulsives (counter-irritants) (21). The same advice was given by Nocard and Leclainche in 1898 after concluding wisely that, 'the question of treating pleuropneumonia is of limited value, since the animal disease laws of every country stipulate the slaughter of sick cattle' (15). The coming of antibiotic therapy, which is effective against mycoplasmas, has modified the prognosis for individual cases, but such treatment has no place in the collective control of the disease.

CONCLUSION

The history of the surveillance and control of CBPP may be divided into three periods:

a) From classical times to the 18th century. The actual importance of the disease was practically unknown in Europe, Africa and Asia. Certain texts reflected the concern of cattle farmers in all these regions. However, the disease was poorly distinguished from other contagious diseases of cattle, particularly rinderpest and tuberculosis. It cannot be ruled out that a procedure for preventive inoculation of virulent materials already existed at this time in the Middle East, from where migrating populations brought it to Africa.

b) The 18th century. Epizootics of CBPP were confounded with successive waves of rinderpest which crossed Europe in 1715, 1740, 1756 and 1775, etc. The disease was identified clearly for the first time during the 1770s by physicians, and then by veterinarians trained in the first European schools. They proposed the early preventive measures of slaughter, while in Africa owners of cattle continued to control CBPP by preventive inoculation.

c) The 19th century. Trade in live cattle developed throughout Europe and spread pleuropneumonia. There are statistics for the damage caused in the Netherlands (100,000 cattle dead between 1833 and 1865), France (212,000 cattle dead between 1827 and 1846), the United Kingdom (1,187,000 cattle dead between 1841 and 1866), etc. (17, 20). In Europe, preventive inoculation, rediscovered and improved by Willems, was used for some decades, in addition to sanitary prophylaxis, which became the main choice by the end of the century. The situation was different in Africa, where traditional vaccination (succeeded by progressively modernised vaccination of the type employed by Willems) formed the basis of CBPP control. The disease entered Australia on two occasions (1858 and 1922) and was eradicated by a combination of slaughter and vaccination (7).

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* *
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