In conclusion it may be stated that black-quarter is the only disease of this group—(gas oedemas would be a more correct name than gas gangrenes)—which is of economic interest. Even here the position can be regarded as satisfactory in so far that the farmer can protect himself against losses at very little expense and that to a large extent he actually avails himself of this opportunity.

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Paper No. 24.

BOTULISM IN THE DOMESTICATED ANIMALS IN SOUTH AFRICA.

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THE study of botulism in the domesticated animals is a comparatively recent one and has only received much attention during the past thirteen years. In considering the occurrence of the disease in South Africa, it will be necessary to do so under different headings, dealing with the condition as it occurs in the different species of domesticated animals. In each case the incidence in other countries will be considered first and then the occurrence in South Africa from the comparative standpoint.

Botulism may be defined as a disease caused under natural conditions by the ingestion of the powerful exotoxins produced by the bacilli of the botulinus group. The symptoms are, in general, those of a bulbar paralysis and are fairly characteristic. The disease has long been known in man as the cause of a very fatal type of food poisoning, usually associated with the eating of canned foods or preserved meats such as ham. The usual symptoms in man are paralysis of the tongue and throat, disturbance of vision, severe headache, ptosis of the eyelids accompanied by a general paralysis of the skeletal muscles. Marked constipation is usually present. In animals the symptoms are essentially the same, except that ptosis of the eyelids is unusual. The intensity of the symptoms and the course of the disease depend on the amount of toxin ingested, large amounts causing death in some cases in less than twenty-four hours. Recovery sometimes takes place, but the convalescence is very protracted.

(1) Botulism in Equines.

Attention was first directed to the subject of equine botulism by the investigations of Graham and co-workers on a disease called " forage poisoning " in the United States of America. This condition was seen chiefly in horses and mules, and had previously been attributed to a variety of causes, including even fungi in the food. The possibility of " forage poisoning " being botulism had previously been considered by Mohler and Pearson in the United States, but the proof was lacking.

Graham, Brückner, and Pontius (1917) investigated several outbreaks of " forage poisoning " in equines. The food could definitely be shown to be the cause and from it, and in one case from the caecal contents of an affected horse, a toxic anaerobe of the botulinus type was cultivated. It is of interest to mention that in one outbreak the toxic material was found to be chicken faeces contaminating some oat straw, and that the oat straw itself was non-toxic. Other outbreaks were found to be due to silage contaminated with botulinus toxin.

Rusk and Grindley (1918) described similar outbreaks in Illinois, U.S.A., and claimed good results for the use of botulinus antitoxin as a preventive for " forage poisoning."

Mitchell (1922) in Canada was able to trace an outbreak of the disease caused by hay to an organism of the botulinus B type.

Buckley and Shippen (1917) and Graham, Brückner, and Pontius (1917) were able to produce typical cases of " forage poisoning " in horses and donkeys by giving them cultures of what is known as the B type of botulinus organism. In his original work on botulism Von Ermengem (1912) described the causal organism simply as *Bacillus botulinus*, but about 1918 it was realized, owing to the work of Burke (1919) and others, that at least two distinct types existed, in nature the toxins of which were distinctly different as judged by toxin-antitoxin tests. It was, therefore, recognized that the *Bacillus botulinus* could be of an A or a B type. From extensive experiments carried out in different countries, it would appear that the B type has the wider distribution.

It was definitely shown by Hart and Hayes (1920) that experimentally both the A and B types could produce symptoms of " forage poisoning " in equines, although under natural conditions it appears to be usually the B type that is responsible.

Graham and Schwarze (1921) actually describe an outbreak of the disease due to the A type of toxin. Cameron (1907) in Australia described the disease in equines, but was not able to suggest any definite cause. It occurred in the State of Victoria from time to time, and the symptoms described are undoubtedly those of botulism. Recently, Jones, Pillers, and Matthews (1928), in England, have described symptoms in horses employed in the work of the Liverpool Municipality, which they attributed to botulism. The etiology in these cases was not definitely determined. There has been much controversy in Scotland as to whether the so-called " grass disease " is botulism or not. The evidence is conflicting, and it would seem more likely that the condition is related to Borna's disease (infectious cerebro-spinal meningitis of equines). Coming now to botulism in equines in South Africa, this has been discussed at considerable length by Theiler and Robinson (1928).

In this article a record is given of a number of outbreaks of what was apparently botulism which had occurred in South Africa between the years 1913 and 1924, and which had been brought to the notice of the Veterinary Department. Descriptions were given of the symptoms shown by the animals in several outbreaks of an obscure disease which had occurred from time to time amongst equines especially in stabled animals. The condition was afebrile and the symptoms are those of a sudden paralysis, though in some cases the animals showed muscular weakness only, with slow recovery. All attempts at artificial transmission of the disease were unsuccessful. At the time it was called spinal paralysis or cerebro-spinal meningitis. These outbreaks occurred in various parts of the country, such as Johannesburg, Bloemfontein, and Grahamstown. Their etiology was never cleared up, but in the light of our more recently acquired knowledge, there can be but little doubt as to their having been genuine botulism.

In December, 1924, two mules died suddenly in the transport stables at the Veterinary Research Laboratory, Onderstepoort, within two days of each other. Both animals went down without any premonitory symptoms, and when down lay stretched out as if completely paralysed, death occurring after a few hours. Bearing in mind the work which had been carried out on " lamsiekte " in cattle with such definite results, it was thought that this equine condition might be similar to the acute form of that disease. On searching the hay-rack from which the two mules had been eating ,the carcass of a partially decomposed rat was found and the hay surrounding it was soiled by the decomposition products. From portions of the rat, cultures were made in anaerobic media, and part of it was ground up in normal saline and dosed to rabbits by the mouth, producing typical symptoms of botulism in these animals. The cultures proved highly toxic as well. A few experiments were then carried out on various animal species, using filtrates from these impure cultures. The symptoms produced in equines were typical of those seen in the natural disease, and varied in intensity according to the amount of toxic material given, and the route of administration. Per os the cultures were highly toxic, but as is usual with botulinus toxins, much larger doses were necessary by the mouth than subcutaneously to produce symptoms. In small laboratory animals typical symptoms of botulism were produced.

After prolonged and very tedious cultural work on the isolation of the toxicogenic organism in pure culture, this was finally accomplished. The organism was found to correspond very closely with the bacteria of the *Cl. botulinum* C type, to be mentioned later in connection with botulism in cattle and birds.

The toxin from pure cultures was found to be much more virulent for equines than for cattle, and it was different from those of the A and B types as judged by cross toxin-antitoxin tests. On the evidence it was considered that the organism producing equine botulism was a new variety of botulinus organism, and provisionally the name *Clostridium botulinum equi* was given to it. At the end of this article a brief discussion of the relationships of the various botulinus types to each other will be attempted in order to try and relegate them to their true positions.

In these two cases in mules the chain of circumstantial evidence is fairly complete. Since their occurrence, other outbreaks of a similar type have occurred and are discussed at length in an article by the writer (1929). In this article, three outbreaks of botulism are discussed, in all of which the symptoms were very typical. In these unfortunately the toxic material could not definitely be traced, but the circumstantial evidence was fairly strong. In one outbreak, a botulinus type of organism was obtained from the caecal contents of one horse, and its toxin produced typical symptoms of botulism in laboratory animals. The strain was unfortunately lost before it could be typed. In another outbreak caecal contents of a horse gave similar toxic cultures. In this case it may be of interest to mention that the deaths, two in number, were the sequel to a rat-poisoning campaign carried out by the owner. In a third outbreak the only material available was the decomposed carcass of a kitten from the forage loft. This produced highly toxic cultures of a botulinus type.

It should be understood that in these three cases actual toxin of a botulinus type was not demonstrated in the material used for cultural work, but only spores of the bacteria. In any case it would be difficult to demonstrate botulinus toxin in the intestinal contents of a horse on account of the bulk of the ingesta.

In toxin-antitoxin tests in the latter two cases the toxin was shown to be of a C type and closely related to, if not identical with, the lamsiekte toxin of cattle to be mentioned later.

Since the publication of the article on equine botulism by Theiler and the writer (1928), letters have been received from Government veterinary officers in different parts of South Africa referring to mysterious outbreaks of disease in equines in their past experience and which they are now convinced were botulism in these animals.

(2) Botulism in Cattle.

References to botulism in cattle are not very frequent in the literature, but those which exist show that it has a very wide distribution. During their experiments on forage poisoning in the United States of America, Graham and co-workers (1917) showed that cattle fed on material which produced botulism in horses did not develop symptoms. Graham and Schwarze (1921) describe what they considered to be an outbreak of botulism in cattle, but the symptoms were not typical. Hart and Hayes (1920), as previously mentioned, produced cases in cattle experimentally. In the drier parts of the United States, such as Texas, a disease called " lumbar paralysis " has been described by Kinsley. This condition seems to be definitely allied to the South African " lamsiekte " of cattle. Sir Arnold Theiler, during his American trip in 1924, was shown cases of this lumbar paralysis and is definitely of the opinion that it is " lamsiekte."

In Europe the evidence for the occurrence of botulism in cattle is rather uncertain. It would appear that endemic paralysis of the pharynx as, for instance, is described by Wyssman (1914), in Switzerland, is possibly a type of botulism, but the proof is lacking. Many cases of diseases in cattle with symptoms resembling those of botulism have been described from Europe, but in the absence of definite information as to their etiology, one has to be guarded in expressing an opinion as to their real nature.

In the Dutch East Indies a disease of cattle with the symptoms of bulbar paralysis has long been known, and was described by Hofstra (1918).

In Australia botulism in cattle has been known for many years under various names. Cameron (1906) refers to a disease called " cripples " in cattle which may possibly, however, have been some other condition. Under such names as " bushsickness," " midland disease " of Tasmania, and other local descriptions it has existed for many years. Seddon (1922), whilst investigating the condition known as " midland disease," was struck by its similarity to the South African " lamsiekte " of cattle. His attention had been attracted to the latter condition by the article of Theiler (1920) on " The Cause and Prevention of Lamsiekte," and in which the obtaining of the causal organism in mixed cultures was described. From a bone from a farm where " midland disease " occurred, Seddon was able to isolate an anaerobic organism producing a powerful exotoxin. On account of its many points of similarity to the A and B types of the *Clostridium botulinum*, but marked dissimilarity when compared by toxin-antitoxin tests, Seddon called his organism *Bacillus parabotulinus*. This study of Seddon's was a very fine piece of work, and in conjunction with that of Bengtson (1922) and Graham and Boughton (1924), enabled a third type of botulinus organism to be recognized, the *Clostridium botulinum* C.

Seddon (1925) refers to the isolation of his *B. parabotulinus* in impure culture from bones of cattle, sheep, and from forage in Australia. Later in the same article he refers to the occurrence of *Cl. botulinum* B in maize silage which had produced botulism in cattle and horses.

Having briefly mentioned the distribution of botulism in cattle in other parts of the world, one may now consider the disease as it occurs in South Africa, where it is known to stock owners in most parts as " lamsiekte." Its distribution in the country is very wide, and it is probable that individual cases occur everywhere. It is, however, chiefly in the areas where, owing to phosphorous deficiency in the vegetation, cattle develop a depraved appetite and chew bones that the disease is a serious menace. At present, however, owing to the systematic feeding of bone-meal to cattle, the incidence of the disease has been greatly lessened, and it is only seen now to any extent where bone-meal feeding is not carried out.

In a special report, Theiler and his co-workers (1927), give in full detail the experimental work carried out by them on " lamsiekte." The work opened up a vast field in the study of mineral deficiency which is still being exploited successfully.

The history of the experimental work carried out on " lamsiekte " in South Africa, and the various theories elaborated in connection with its etiology, make an interesting chapter in the study of diseases in general. From the earlier bacteriological work of Bowhill, Spreull, and Robertson, in the Cape Province, who isolated bacteria, later shown not to be etiologically associated with the condition, to the work of Theiler from about 1912 to 1919, one theory after another was followed until discarded as unsatisfactory. It was not until 1919 that the final proof of the disease being a form of toxaemia related