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A REPORT ON

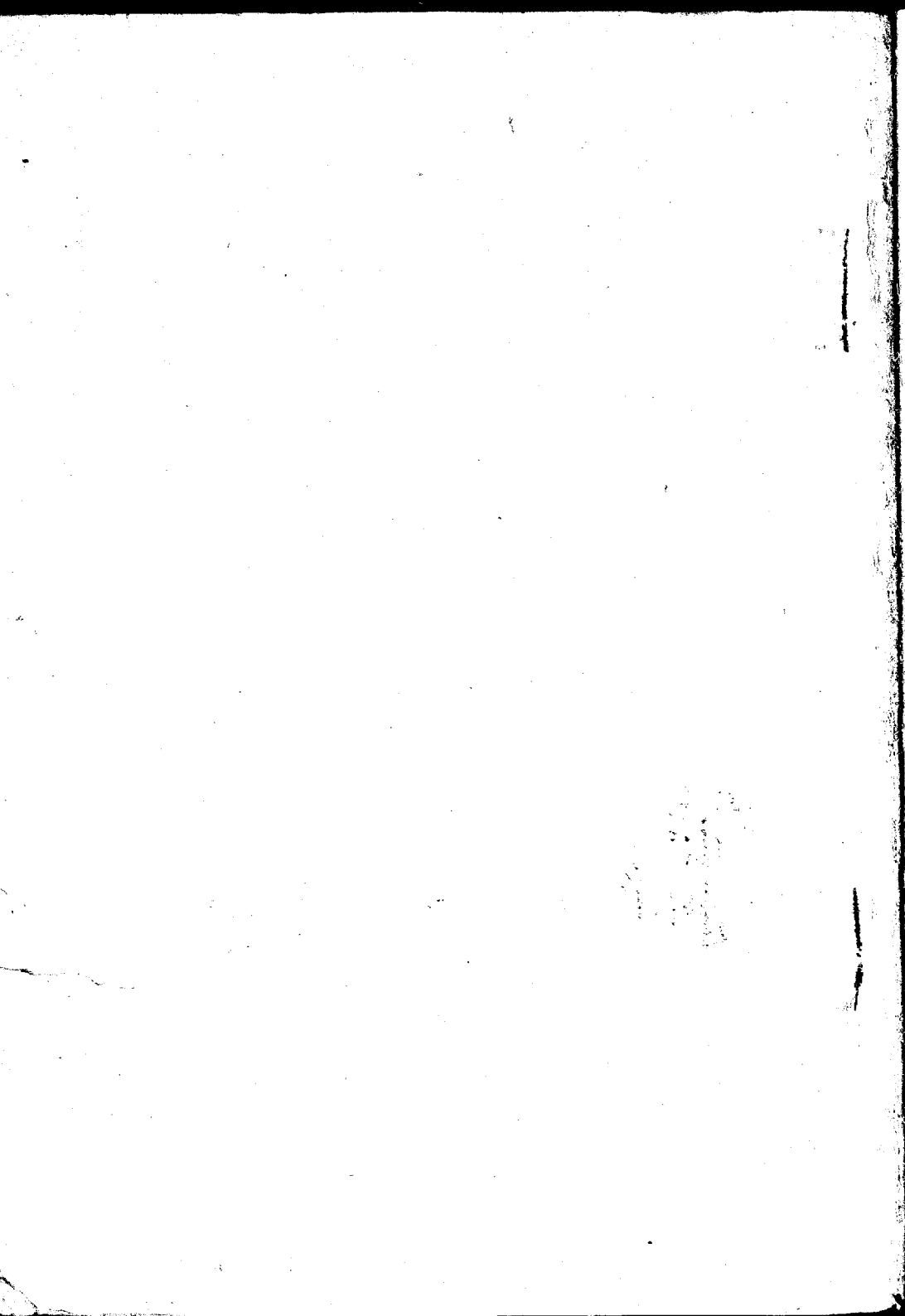
HEMORRHAGIC SEPTICÆMIA IN ANIMALS

IN THE PHILIPPINE ISLANDS.



By PAUL G. WOOLLEY, M. D., and J. W. JOBLING, M. D.

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LETTERS OF TRANSMITTAL.

DEPARTMENT OF THE INTERIOR,
BUREAU OF GOVERNMENT LABORATORIES,
OFFICE OF THE SUPERINTENDENT,
Manila, October 19, 1903.

SIR: I have the honor to submit for publication a report on "Hemorrhagic septicaemia of animals in the Philippine Islands," by Dr. Paul G. Woolley, Pathologist in the Biological Laboratory, and Dr. James W. Jobling, Director of the Serum Laboratory.

Very respectfully,

PAUL C. FREER,
Superintendent Government Laboratories.

HON. JAMES F. SMITH,
Acting Secretary of the Interior, Manila, P. I.

DEPARTMENT OF THE INTERIOR,
BUREAU OF GOVERNMENT LABORATORIES,
BIOLOGICAL LABORATORY,
Manila, October 14, 1903.

SIR: In reply to your communication regarding the occurrence of a recent epidemic among the Government carabao in and about Manila, I have the honor to transmit the inclosed report from this Laboratory on hemorrhagic septicaemia in the Philippine Islands, by Dr. Paul G. Woolley, Pathologist, Biological Laboratory, and Dr. James W. Jobling, Director of the Serum Laboratory.

Very respectfully,

RICHARD P. STRONG,
Director Biological Laboratory.

DR. PAUL C. FREER,
Superintendent Government Laboratories.

Manila, P. I.



A REPORT ON HEMORRHAGIC SEPTICÆMIA IN ANIMALS IN THE PHILIPPINE ISLANDS.

[By Paul G. Woolley, M. D., Pathologist, Biological Laboratory, and J. W. Jobling, M. D.,
Director of the Serum Laboratory.]

The appearance of hemorrhagic septicæmia in the Philippine Islands was a serious complication of the local cattle problem. Rinderpest, which had destroyed thousands of carabaos and which had brought agriculture nearly to a standstill, was just beginning to be controlled by vigorous work with protective sera, and cattle were being immunized in China for importation into these Islands. This work had been progressing very satisfactorily, when suddenly and without warning hemorrhagic septicæmia appeared in a herd of cattle arriving in Manila from Shanghai. At about the same time that the disease was recognized here reports were received from Hongkong giving accounts of a virulent disease prevalent there which very much resembled bubonic plague in human beings, and which was called "cattle plague."¹ Thus far these reports have been so meager that we are unable to state whether or not the disease is endemic at that port, as it seems to be in other parts of the world, and whether or not it corresponds with the disease studied here.

That hemorrhagic septicæmia existed in the Philippine Islands

¹Since the above notes were written, the report of the Government bacteriologist at Hongkong has been received. In this there is a brief summary of the morphologic and cultural characteristics of the organisms found in the animals that died in the epidemic there. This germ grew readily on the "ordinary culture media." It was a bacillus that stained more deeply at the poles, and which did not stain by Gram's method and which was nonmotile. Its appearance on culture media was similar to that of *B. coli*. All inoculated animals died after twenty-four to forty-eight hours with symptoms of septicæmia. From his facts Hunter concluded that he is dealing with a form of hemorrhagic septicæmia. But whatever the disease may be, it is not the same one that we are studying, judging from the description of the organism.

previous to the present epidemic can not be stated with any degree of definiteness. Since our preliminary report¹ of the present outbreak some Philippine physicians have stated that they had previously seen animals dying in the same manner as the ones lately affected, but whether or not these cases were identical with ours can not be determined. Carabaos are sometimes affected with a condition similar to heat stroke, and if one of these animals should die in such an attack the clinical features presented by it might remarkably resemble those of the acute form of hemorrhagic septicæmia. One of us had recently seen such a case, in which a carabao lately landed from a vessel had been driven through the town and was suffering for lack of water. It suddenly staggered and fell by the roadside, frothing slightly at the mouth, and was unable to rise for at least an hour, and not until it had been drenched frequently with water. If the animal had died in this attack acute hemorrhagic septicæmia might have been suspected and a correct diagnosis could have been made only after an autopsy. But the above-mentioned statements of the previous occurrence of hemorrhagic septicæmia must be taken *cum grano salis*, for there is a proneness among many to recognize any symptom complex as the disease under discussion at the time. This tendency is well shown by the fact that pseudo-farcy is taken for surra by men well acquainted with horses. It is, however, possible that hemorrhagic septicæmia has been seen here before and that the disease has thus far evaded the detection of laboratory men, even though these have a wide experience with cattle diseases.

The disease is a widespread one and has been noted in almost every quarter of the globe. The first cases were in Germany and were cited by Bollinger. Later reports of epidemics in Germany have come from Kitt, Jakoby, Buch, and others; from France, Nocard and Leclainche have reported cases, as have also Galtier and others; Bosso, Oreste, and Armanni, and others have detailed descriptions of epidemics in Italy; Poels reports cases from Holland; Jensen from Denmark; Piot from Egypt; Van Eecke Fischer and Hubenet from Java; Reischig, Sequens and v. Ratz from Hungary; Janson from Japan; Sanfelice, Loi, and Malato from Sardinia; Pease from British India; Carrougeau and Blin and Carré from Indo-China; Lignières from South America, and Smith, Wilson and Brimhall, Reynolds, Fennimore, and Nocard

¹Read at a meeting of the Manila Medical Society July, 1903.

from the United States. It is easy to see how the spread may have included the Philippine Islands if the disease is a contagious one and not endemic in that Archipelago. There is a possibility that China is not to blame for the cases recently discovered here. It may be that the disease is endemic in the Philippine Islands and that by food or water the organisms gained access to these animals through wounds of the gastro-intestinal tract (or even without such wounds), or through wounds on the surface of the body, or possibly by way of the respiratory tract.

It is now settled beyond doubt that organisms resembling those of hemorrhagic septicemia are found in waters and soils. It has also been shown that similar ones are present on the mucous membranes of healthy animals. Moore, of the Department of Animal Industry at Washington, for instance, has demonstrated that organisms resembling those of hemorrhagic septicemia and capable of producing the disease occurred in 80 per cent of the cattle, 48 per cent of the hogs, 50 per cent of the sheep, 16 per cent of the horses, 30 per cent of the dogs, and 90 per cent of the cats examined by him. Davaine, Pasteur, Gaffky, Gamaleia, S. Mayr, and Kitt have found organisms in waters, soils, sputum, and bronchial secretions which, injected into the circulation of animals, caused septicemia which agreed with the usual disease in all its details.

Since this disease so closely resembles bubonic plague, it might be supposed that it corresponds with it also in the sources and means of infection, but these points are awaiting explanation. Since the organisms occur so commonly in healthy animals, it is probable that a lowering of resistance to disease, whether brought about by trauma or by abnormal conditions of environment, relatively increases the virulence of the organisms and brings about conditions from which infection and disease result. This is certainly true in the sporadic pneumonias of cattle, described by Theobald Smith, who believes that the organisms, present in such conditions, are secondary invaders.

The history of the present epidemic of hemorrhagic septicemia in the Philippine Islands is as follows:

A shipment of cattle arrived in Manila Bay from Shanghai on May 28, 1903, and was kept on board until June 1, when the animals were sent to the Perez estate, in Paco. On the following day two of the animals were noticeably ill, and were sent to the serum laboratory for observation.

Case 1.—The first animal to die was very weak when first observed, but in fair physical condition otherwise. The conjunctivæ were somewhat congested, respiration was rapid, and the feces normal. Temperature, 39.2° C. When taken off the truck at the Laboratory it staggered a few steps and fell on its side. There were numerous bruises on the body, probably the result of a rough voyage across the China Sea. It ate food when placed near it and also drank, although it did not, apparently, suffer from thirst. It had no cough. During the next few days it became a little brighter and somewhat stronger. On June 6 it was again weak and could not stand up, the hind legs seeming to be especially feeble. It gradually became weaker and diarrhœa developed, but with no traces of blood or mucus. Death occurred on the ninth day after landing.

The post-mortem examination showed a few patches of subcutaneous edema on the sides. There were a few small pericardial hemorrhages about the base of the heart. The lungs showed a number of subpleural-nodules, which on section exposed granular areas similar to those seen in bronchopneumonia, in the stages of red and gray hepatization and suppuration. The suppurating areas were filled with a thick, granular, greenish-yellow, sticky material.

Cultures were made from the lung abscesses on agar and blood-serum. After twenty-four hours at 37° C. the agar tubes showed a growth of small, transparent, grayish, round colonies. The blood-serum showed a very scanty growth of small colonies. Transfers were made from these tubes to various other media, and plates were also made. After a careful study of its morphologic and cultural characteristics, it appeared that the organism under consideration was a short bacillus with rounded ends, and nonmotile. Its measurements varied between 1 and 2 microns in length and 0.3 and 0.5 of a micron in thickness. The largest forms were seen in glucose media, the smallest on potato. From the animal body it showed well-marked polar staining, although this was not so distinct in organisms grown on artificial media. It was stained easily with the usual watery aniline stains, but was not stained by Gram's or Weigert's methods. The rods, as a rule, occurred singly, often in pairs, occasionally in chains of five or six individuals. The appearance of the growths on the usual culture media was in no way characteristic. The colonies on agar were small, grayish, transparent, and well circumscribed, with little or no tendency to spread. On all the solid media approximately the same appearance was noticed. In gelatin no liquefaction was caused. In bouillon a granular deposit was formed on the sides and bottom of the tube; during the first few hours of the growth the whole medium was faintly clouded, but as the sediment was deposited the liquid became clear. After a few days the sediment became viscid, as could be shown by shaking the tube, when the precipitate rose, not in floccules but in threads. In Dunham's peptone solution the same general characteristics were observed as in broth, but the growth was not so abundant. Indol (cholera-red) was produced rapidly so that at the end of twenty-four to thirty-six hours the addition of pure sulphuric acid (free from nitrites) produced a well-marked pink color. No phenol could be appreciated. No gas was produced in solid media

containing sugars nor was the reaction changed. Milk remained unaffected even after ten days; no acid was produced, no coagulation occurred, and there was no reduction of litmus. Stab cultures in solid media showed nothing remarkable. The growth followed the line of inoculation closely with no tendency to spread, and extended to the bottom of the tube as a finely granular growth composed of small colonies. The surface growth was small, just surrounding the point of entrance of the needle.

Case II.—The second carabao was also taken to the Serum Laboratory on a truck, and on arriving there was unable to walk more than thirty feet. Its eyes were clear, the respiration quickened, and the body covered with numerous bruises. On the 4th of June it was able to walk and graze, and seemed to be in good condition. On June 14 it was again dull and weak, but still able to walk, the greatest weakness being in the hind quarters. Nevertheless, in spite of a good appetite and in the absence of other symptoms, the animal gradually became greatly emaciated. On June 21 a swelling was noticed on the inner and outer sides of the left ankle. At this time the animal was hardly able to stand, although the appetite was still fair. The swelling on the ankle was incised by Dr. Slee and the contents evacuated. The abscess cavity was a loculated one and the incised tissue was quite edematous. On June 25 diarrhea developed and two days later, when just about to die, the animal was killed and an autopsy done immediately.

The findings were as follows: Besides the incision over the left ankle there were dried scars on both sides of the body, and a small opening, discharging a greenish-yellow pus, in the left flank over the quarter. Dissection of the left ankle showed that the abscess cavity extended around the joint without involving it, but reaching far up the leg. An opening into the abscess of the flank led into a mass of suppurating glands, which looked much like the broken-down ones of bubonic plague, save that the contents were more granular. The tissues about these suppurating areas were infiltrated with a gelatinous exudate of a distinct yellow color. In the intermuscular and surrounding tissues there were more or less well-marked hemorrhages. On skinning the animal a widespread subcutaneous gelatinous edema was discovered, with large and small hemorrhages. In the stomach walls and beneath the pericardium and the pleura hemorrhages were present, some of which were well circumscribed and others irregular in outline. There was a very large extravasation of blood into the mediastinal portion of the parietal pleura, and another similar one along the descending aorta.

The lymph glands were generally enlarged, pale, and edematous, but some were smaller and hemorrhagic. There were petechiae in some of the edematous glands. The lungs were for the most part crepitant, although there were areas in the left one which resembled the stage of red hepatization of broncho-pneumonia. The spleen was of about normal size, with no hemorrhages into its substance but with many subcapsular ones. The liver showed nothing remarkable. The kidneys were of fair size and the perirenal tissues were edematous; their pelvises were filled with a gelatinous material. There were no hemorrhages into mesentery.

In the abscesses from the left ankle and from the hind quarters small

polar-staining bacilli were found in almost pure culture. Smears from the lymph glands showed similar organisms, but the heart's blood was apparently free from them.

Cultures showed an organism that corresponded with that from Case I.

Case III.—This animal, a full-grown carabao, was apparently perfectly well until one morning, when it looked stupid, its gait was clumsier than usual, and its eyes appeared to be sunken. It did not care to use the mud bath, but wandered about aimlessly. (The native overseer said that it was insane.) On the third day weakness across the loins and in the forelegs was evident. Its appetite was still fair. On the morning of the fourth day it fell over and was unable to rise again, and died in the afternoon. Smears showed a few organisms that were polar-staining. Culture on placenta fluid showed organisms that resembled diplococci. Culture lost in moving.

Case IV.—This animal was stupid and weak when first noticed, and as nearly as could be determined had been sick for about three hours. It kept on its feet and moved from place to place for several hours, eating occasionally. Death took place suddenly. Smears from organs showed a few polar-staining organisms. Cultures not made.

Case V.—This carabao lived about three days after the appearance of the first symptoms, the principal one of which was weakness, especially in the forelegs. At autopsy the base of the heart was edematous and there were scattered areas of subcutaneous edema with a few hemorrhages.

Case VI.—Death was very sudden, occurring twenty-four hours after the beginning of the illness. Autopsy revealed edema and blood-stained infiltrations about the inguinal and prescapular glands. The latter were much swollen and showed areas of necrosis and a few small hemorrhages in their substance. Hemorrhages, varying in size, were present in the inner and outer surfaces of the intestines, under the visceral and parietal pleura, under the parietal and visceral pericardium, and under the endocardium. Petechiae were present on the surface of the liver and were especially well marked on the inner surfaces of the branches of the portal vein. A few small hemorrhages were found under the capsule of the spleen and under the surface of the diaphragm. Smears from lymph glands, liver, spleen, and lungs showed polar-staining bacilli.

Case VII.—The duration of the illness was two and one-half days, during which the animal could scarcely stand, although the appetite remained good. The almost characteristic edemas were well marked in this case and polar-staining bacilli were found in smears.

Case VIII.—This animal, one of the finest in the herd, was alive and well on the morning of June 7; the following morning it was found dead. The post-mortem examination showed hemorrhagic edema under the skin along the spine, about the prescapular glands, and around the base of the heart. There were ecchymoses on the surface of the heart, under the capsule of the spleen, in the inguinal and prescapular glands, in the pancreas, in the gall bladder, and under the serous and mucous surfaces of the intestines. Smears from heart and spleen showed polar-staining bacilli, some of which showed evidences of having been a capsule or pseudocapsule. Blood serum

from the case had no agglutinative reaction on the bacilli previously obtained from other cases.

Cases IX, X.—These were calves. Their clinical histories and the details of the autopsies are wanting. All that is known is that in neither case was the spleen enlarged, that there were subcutaneous gelatinous edemas, and that the usual edema was about the base of the heart in each.

Case XI.—In this case also the clinical history is wanting, but the same pathological changes that were observed in the two previous cases were present. Smears from the blood showed a number of polar-staining bacilli occurring singly or in chains of three or four individuals. Cultures were made from the heart's blood on agar and an organism was obtained in pure culture which corresponded to that isolated from Case I.

Case XII.—This animal died within twenty-four hours after the appearance of the first symptoms. It was large and well nourished, and when examined was lying on the ground, the legs stiff and the head drawn back. The agonal stool was bloody. There were hemorrhages along the spine into the subcutaneous tissues, as well as on the surfaces (inner and outer) of the small intestine. The prescapular glands showed areas of hemorrhage and necrosis.

Case XIII.—This was a fairly well-nourished carabao which had arrived from Shanghai three days previous to the appearance of illness. It was well on the evening of July 22. On the following morning it was found dead. In this case the lesions were confined to the heart and lungs; the subcutaneous gelatinous edema seen in the other cases was absent. The lungs were not collapsed and contained air in only a few patches. The costal pleura was intensely inflamed and showed numerous ecchymoses and was covered with a thick stratum of fibrino-purulent exudate. The visceral pleura was in a similar condition. The mediastinum was filled with a mass of yellow gelatinous material composed of serum and fibrin. On section the lung tissue was red, in places very dark, and divided into large and small lobules by white fibrous and fibrinous bands intersecting in all directions. These bands varied from $\frac{1}{4}$ to $\frac{1}{2}$ an inch in width and were studded with loculi containing a sero-fibrinous and fibrino-purulent material. In the pericardial cavity was a large amount of serous fluid, containing floccules; both layers of the pericardium were covered with fibrinous shreds. The serous membrane under this exudate was inflamed and dotted with ecchymoses. The auricular appendages were thickly mottled with hemorrhages. Smears from the heart's blood, lungs, liver, and lymph glands showed a considerable number of polar-staining bacilli, and cultures showed a similar organism in uncontaminated growths. This case followed closely the description of sporadic pneumonia as given by Theobald Smith.

Besides the cases of hemorrhagic septicemia seen in carabaos, a native horse was found to be suffering from a malady which was caused by the same organism. In this case the chief lesions were found in the lungs, and these corresponded perfectly with those of

Case I. There was also a well-marked gelatinous edema about the base of the heart. The organism was recognized in smears and cultures and identified with that from Case I.

In summarizing the chief clinical features of these cases we can say that there have been three fairly well differentiated types—one, intensely acute, in which the animals died within a few hours after the onset of symptoms; one less acute, in which the animals suffered chiefly from weakness in the limbs, and more especially in the hind ones; during the course of this form of the disease the appetite was little or not at all affected. Lastly, one in which the course was prolonged, accompanied by great weakness and emaciation and occasionally by suppuration. These are not distinctly defined types, for rarely does the disease affect the animals in the same way, except in the acute form, in which the course is so rapid that there is little time to observe symptoms.

The pathological types are as indefinitely defined as the clinical types. In a general way, however, there is a pulmonary type in which the lesions resemble those of broncho-pneumonia, sometimes with abscess formation, and with or without pleuritis. Combined with these changes there may be marked changes in the interlobular tissues, with the formation of fibrous bands and with emphysema. Another type is the rapidly fatal septicemic form, in which there are few macroscopic changes in the organs beyond incipient parenchymatous degenerations. The third type is a glandular and suppurative one which terminates in a general infection. In all these forms there have been more or less wide-spread hemorrhagic lesions combined with gelatinous edemas. This last-named condition was especially frequent about the base of the heart, and in some cases it was the only macroscopic lesion.

In but few cases have complete temperature charts been kept—this because most of the animals have come from herds regarded as healthy and have died too suddenly to allow records to be prepared. The temperature charts of Cases I and II will, however, be appended. These curves show very little except a primary rise of temperature, after which there is a fall, with the subsequent curve, running either near the base line or very irregularly.

The clinical cases given above agree in many points with those described in the literature, but in many details, too, they do not. Wide variation clinically and pathologically seems to be one of the most common features of the disease caused by *B. plurisepticus*.

In Bollinger's report the cases are divided into an exanthematous and pectoral type. The former is characterized by fever, swellings of the face and neck, inflammation of the mouth and tongue, with ecchymoses under the mucous membranes, and bloody diarrhea. Such cases we have not seen, nor have we observed any cases resembling them. This may be because the hide of the animals that we meet with here is thick enough to prevent the edematous swellings from becoming apparent, for certainly in the majority of cases there have been subcutaneous edemas, some of quite considerable size. The latter type showed, principally, pulmonic lesions. We have studied three cases that followed this type—two in carabaos and one in a horse. In one carabao and in the horse the chief lesion was a broncho-pneumonia without pleuritis; in the other carabao it was a pleuro-pneumonia.

Lignières divides the cases he has seen into three classes—first, a diarrheic one, the acute form, in which death occurs in from one to two days after the onset of the diarrhea. In this, pectoral lesions are common (i. e., pneumonia, pleurisy, etc.). The second class he calls *Éntèque*. The course of this form is more chronic, and the animal may live for three to four weeks with more or less fever and fetid diarrhea, culminating in anemia and death. The third or cachectic form is marked by wasting, articular localizations, and profound anemia, followed by death.

Speaking generally the commonest types in the literature are the exanthematous and pulmonary. Oreste and Armanni described cases in which edemas and hemorrhages were the chief lesions. Many of Kitt's animals showed pneumonia and pleuritis, Jensen's showed phlegmonous edemas, fibrinous pleuritis and pericarditis, and gastro-enteritis; Galtier described hepatization of the lungs, subendocardial hemorrhages, and peritonitis; Guillebeau and Hess found fibrinous pleuritis and pericarditis, hemorrhagic infarcts in the lungs, and small hemorrhages into the mesentery and on the surface of the kidney; Bosso described hemorrhages into the serous membranes and into the mucus lining of the intestine; Pease observed cases in which the chief lesions were ecchymoses, widely disseminated in the bodies; Fennimore remarked inflammation of the mucus membranes of stomach and intestines, pleuritis, pericarditis, and edema of the lungs; and Smith carefully studied and minutely described peculiar lung lesions in which interlobular changes were an important feature.

From a summary of the facts given in the literature we may conclude that the animals attacked are of all ages, the onset is usually sudden, the course is rapid and death occurs in from 85 to 98 per cent of the cases. In the less rapid forms of the disease the symptoms may be refusal of food, cessation of rumination, and lactation. The initial rise of temperature may be followed by a drop to normal or subnormal. The respirations may be rapid or labored, bloody material may be discharged from the nostrils, bowels, or bladder, and noncrepitant swellings may occur on face, neck, or about the back and ankles.

From all these varying types of disease similar organisms have been isolated. In the outbreak which we have studied the characters of the microbe have been as follows: Short bacilli with rounded ends, polar staining, occasionally encapsulated, and nonmotile; nonliquefying, non-Gram staining, growing invisibly on potato, not producing gas, not coagulating milk or reducing litmus, producing indol and nitrites, and not forming spores. It is an organism which corresponds closely with the bacilli of hemorrhagic septicæmia of Hueppe, and especially with that member of the group called *B. bovisepiticus* (Kruse), *B. bovisepiticum* (Kruse-Migula), *B. plurisepticus* (Kitt), *B. bipolare multocidum* (Kitt), *B. der buffel-seuche* (Oreste-Armanni), etc. The chief differences between the present bacillus and the ones described in the literature are its indol production, which is invariable, and its invisible growth on potato, which also appears constant.

ANIMAL EXPERIMENTS.¹

The organism has been injected into guinea pigs, rabbits, monkeys, small birds, a dog, a chicken, and a calf. In all but the last three animals death has followed in from nine to twenty-four hours after intrapleural injection, in five to eighteen hours after intraperitoneal inoculation, and in five to fourteen hours after intravenous injection of virulent material. Subcutaneous injection with a culture kept at 60° C. for ten minutes killed a rabbit in thirty-six hours; intravenous injection with the same material killed in eighteen hours. In both these two last cases the organism was recovered from the heart's blood.

The lesions in animals killed with virulent cultures varied with

¹In these experiments Mr. Clegg, Assistant Bacteriologist in the Biological Laboratory, has given us much valuable assistance.

the length of the disease and with the animal used. In all there was edema and some hemorrhagic infiltration or ecchymosis at the site of inoculation. In addition to this two or three cases were noticed in which there were large collections of pus surrounded by tissues in a state of coagulation necrosis in the immediate neighborhood of the wound of inoculation. The more resistant the animal and the more attenuated the organism (within certain limits) the greater was the tendency to suppuration.

The protocols of the animal cases are as follows:

Rabbit 321.—Inoculated subcutaneously with 0.5 c. c. of an emulsion of a twenty-four-hour-old agar growth of the organism from Case I. Dead in eighteen hours. Large hemorrhage at site of inoculation, and a few small ones in axillary and inguinal glands. Lungs congested, vessels of the heart distended with blood. Liver soft and showed diffused pale areas of fatty or necrotic tissue. Adrenals congested. No changes in gastro-intestinal tract. Small amount of fluid in peritoneal cavity. Smears from the liver, spleen, heart's blood, and site of inoculation showed the typical organisms which were recovered in pure culture from the blood.

Rabbit 322.—Inoculated subcutaneously with 1 c. c. of a suspension of a twenty-four-hour-old agar growth of the organism obtained from Case XIII. Dead in five hours. No well-marked lesions. Some edema about the wound of entrance. Liver and kidneys congested. No hemorrhages or necroses. Smears from heart's blood, spleen, kidney, and liver showed the typical organisms, and these were obtained in pure culture from the blood.

Rabbit 335.—Inoculated intravenously with 1 c. c. of a bouillon culture of the organism from Case I, which had been kept at 58° C. for ten minutes. Dead twenty-four hours later. Autopsy showed focal necroses in the lower lobes of both lungs, in liver, and spleen. Smears from spleen, kidney, and blood showed a few small polar-stained bacilli, which, however, were recovered in pure culture.

Monkey 256.—On June 16 1 c. c. of a bouillon culture was injected under the skin of the right side. On June 18 1 c. c. of a culture in placenta fluid was injected into the peritoneal cavity. Death occurred six hours after the second inoculation. About the points of inoculation were areas of edema. Surrounding the point of inoculation on the right side was also a large intermuscular abscess filled with a pale, greenish-yellow, gelatinous pus. Practically the whole right side of the animal was the seat of a subcutaneous gelatinous edema. The peritoneal cavity contained a large amount of sanguinous fluid, and the luster of the peritoneum was dulled and it was injected. The other lesions were cloudy swelling of the parenchymatous organs. Smears from the spleen, liver, and peritoneum, and from the pus at site of inoculation showed polar-stained organisms: none seen in blood. In several leucocytes from the peritoneal cavity polar-staining bacilli were seen. The heart, peritoneum, and liver showed pure cultures of this organism.

Monkey 297.—Inoculated intraperitoneally, with 1 c. c. of a broth culture of the organism from the horse mentioned above. Death in thirty-six hours. The abdominal wall about the wound made by the needle was edematous. The serotum was distended and edematous. There was well-marked subcutaneous ecchymosis and gelatinous edema with a certain amount of purulent infiltration. Coils of intestines were adherent, and there was a quantity of sanguino-purulent exudate in the peritoneal cavity. The pus extended down along the spermatic cords into the serotum. The left testicle was much enlarged and contained some purulent and ecchymotic areas. The right testicle was small and surrounded by a purulent exudate. The intestines showed a few small hemorrhages under the serous layer. The other organs showed no marked change. Smears from the abdominal cavity, liver, spleen, subcutaneous abscesses, and testicular abscesses showed polar-stained bacilli. These were recovered in pure culture.

These case reports of animals dying spontaneously and those dying after inoculation will give some idea of the ability of the organisms to cause widely varying types of disease. It is as Reynolds has said: "For the present, at least, we must consider the term hemorrhagic septicæmia as quite inclusive—a sort of generic name, which must cover a multitude of varying types of disease."

When we encountered the first cases of the present epidemic we feared that the outbreak might take on as dangerous proportions as it had in some of the epizootics first reported, and as time has gone on this fear has been partially realized, but we still hold to the idea that the cases have been the direct result of trauma, in many of these cases, and the indirect, of infection with an organism present in the mouths of healthy animals.

It does seem very probable now that a pasture may become infected and that new animals placed on such infected ground may die of the disease in one of its forms, especially if the animal is in any way predisposed to disease. For this reason it seems most unwise, in the present state of our knowledge of the disease, to move animals from place to place, and so risk infecting new ground to be used by well animals.

In one of the corrals in Manila a few animals in one herd had died. Following these deaths came heavy rains, so that the corral became a great mud hole. Into this place a new herd of carabaos were driven after they had crossed the China Sea from Shanghai. Following the advent of these animals deaths occurred rapidly, and in all but a few of the dead the lesions of hemorrhagic septicæmia were found.

It is our opinion that the disease was the result of the rough sea

voyage, change of diet, the preliminary inoculation against rinderpest, and finally the filthy surroundings in Manila. Under these circumstances disease might be expected.

The question concerning the source of the infection by which death resulted remains to be answered. Were the organisms in the animals themselves? Were they in the mud of the corral? Neither of these questions can be answered positively as yet. We have not been able to find the bacilli in the mud. But just as long as there is any doubt the animals must be treated as though they could scatter the germs of the disease with their excreta.

Up to the present time there has been no evidence that the disease here is directly contagious, but Carrougeau and Blin, Bollinger, Friedberger and Frohner, and others considered the disease which they studied was. On the other hand Nocard and Leclainche think that the cases are sporadic and that the disease is enzootic, and Wilson and Brimhall, and Reynolds believe that it is not contagious.

For the purpose of eliminating all or most chances of contagion we suggest that each animal in a suspected herd should be isolated or staked out separate from the rest of the herd for at least ten days after the occurrence of the last case in the herd to which it belongs and until any abrasions on the surface of the body have healed. (Brimhall and Wilson have suggested that skin lesions may play a part in the spread of the disease.) While the herd is under suspicion none of the animals should be allowed to use the mud baths. After an animal has died the ground upon which it has stood should be cleansed by fire or antiseptics, and if it has had access to the mud bath this should be disinfected in the most thorough way with lime or crude carbolic acid and then flushed out with water. There seems to be some difference of opinion as to whether the organism is able to live any length of time in contaminated water, but since similar ones have been found in rivers and in soils it is wiser to be on the safe side and use all precautions. Theobald Smith states that the organism is very vulnerable, and Nocard and Leclainche say that it is a facultative parasite. The result is that for the present it must be dealt with as though its resistance were great. Kitt, in Kolle and Wassermann's *Handbuch*, recommends that it be treated as though it were the bacillus of anthrax.

Up to the present time we have been unsuccessful in obtaining any serum or protective substance that will modify or ameliorate the course of the disease in experimental animals. Work along these lines is, however, being done and will be made the subject of a later report. Wilson and Brimhall report that they were able to produce a fairly high degree of immunity. In the book on "Les Maladies Microbiennes des Animaux," by Nocard et Leclainche, it is stated that the belief of Lignières—that the organism of bovine pasteurellosis can be attenuated and used as a vaccine—is possible. The details of his method are not given, and thus far we have been unable to obtain them.

NOTE.—We have been interested to hear, since the notes for this report have been prepared, that there have been epidemics of an unknown nature among chickens and swine in the provinces about Manila. The reports make it seem probable that these diseases are related to the hemorrhagic septicæmias of fowls and hogs. We have received from a gentleman in Manila a chicken that had died suddenly. From this we have isolated an organism that resembles the bacillus of chicken cholera, which, when inoculated into small birds in minimal quantities, gives rise to a rapidly fatal septicæmia that corresponds exactly with the disease chicken cholera. In inoculating with this organism a needle whose tip was covered with a culture of the organism was plunged through the skin and into the subjacent muscles. The disease prevalent among swine remains to be determined. However, hog cholera has been previously reported here.

These observations make it seem probable that chicken cholera is endemic here, and, if there is any value in analogy, that other forms of hemorrhagic septicæmia may be.

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