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**EQUINE MALARIA AND ITS SEQUELÆ.**

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THE disease generally known in our sub-continent by the name of "biliary fever" of horses I have called "equine malaria." In thus indicating the nature of the disease we are at the same time able to allocate it to its proper class, for biliary fever is a disease of the blood, or, to be more accurate, a disease of the red corpuscles, these being infected by the specific parasite. In previous publications I opined that this parasite belonged to the genus *plasmodium* or *hæmamoeba*, the genus to which the parasite of human malaria belongs. I came to this conclusion on account of the multiform appearances of the endoglobular parasite in stained preparations. Since then, however, better staining methods have enabled me to come to the conclusion that the parasite in question belongs not to the genus *hæmamoeba*, but to the genus *pyroplasma*, the parasite being closely related to the *pyroplasma bigeminum* (*pyrosoma* or *apiosoma bigeminum*) and *pyroplasma canis*.

These parasites are both known in South Africa; the former causes the redwater of cattle, the latter malarial biliary fever of dogs. Laveran, to whom I sent specimens of blood taken from horses, gave this parasite the name *pyroplasma equi*, and this is the name by which I shall in future designate it.

By using Laveran's method of staining the pyroplasmodic nature of the parasite is easily seen, whereas by staining with the ordinary basic aniline dyes the parasite shows a variety of forms, such as are

commonly found in the æstivo-autumnal fever in man. They differ, however, from the parasite of human malaria (1) in not producing any pigment, and (2) in the method of reproduction.

It is convenient to retain the term malarial fever, inasmuch as the similar diseases of other domesticated animals are termed malaria by the different authors. For instance, the Italians Celli and Santori, Lignières of Argentina, and Nicolle of Constantinople, call redwater "bovine malaria," and Hutcheon of Cape Town gives the name of canine malaria to the similar malady of dogs.

Before describing the pyroplasma equi I wish to describe briefly the well-known pyroplasma bigeminum (*alias* pyrosoma bigeminum) of redwater.

We know at the present time two different forms of pyroplasma in South Africa—the small rod-shaped or bacillary form, which produces the atypical virulent redwater; and the large oval and round form, which is found in the common typical redwater. Between these two extremes every variety of intermediate form, both in shape and size, is found.

It is especially the large form I am here discussing, as commonly found in the typical forms of Texas fever all over the world.

The individual hæmatozoon is usually pear-shaped, and it was called the pyroplasma bigeminum from the parasite so frequently occurring in pairs.

A full-grown pyroplasma is often as long as the diameter of a red disc, but usually about half to two-thirds that length. The large forms are not always pyriform, but very often spherical, oval, or spindle-shaped. When stained by Laveran's method a distinct karyosome is found, which takes a deep red colour, surrounded by a clear zone which lies in a slight bluish mass of protoplasm. The karyosome is always found at the edge of the pyroplasma. The spherical forms are generally found in the internal organs, especially kidneys and heart muscle.

The pyroplasma equi is also represented by a similar variety of forms to that seen in redwater. I found stave-like, leaf-like, club-shaped, oval, pyriform, and round-shaped parasites.

Round and ring forms are very common, and the parasites are found equally distributed in the different organs. When the blood of horses suffering from the disease in its early stage is taken and defibrinated, and kept for some time before cover-glass preparations are made, nearly all the parasites appear spherical. A similar phenomenon can also be observed with pyrosoma bigeminum.

The diameter of pyrosoma equi is from  $1.5\ \mu$  to  $2.5\ \mu$ , and the largest form fills about one-fourth to one-third of the red disc.

The smallest forms are motile, inasmuch as by careful observation they can be seen to change their place within the corpuscle. The larger forms are non-motile.

In unstained fresh preparations the parasite can easily be detected by its different refraction, thus standing out in clear outline.

I repeatedly found that fresh unstained parasites appear commonly as the round form; this is, in my opinion, an indication that its real form is the sphere, and that the non-spherical forms above described are probably dependent upon the fixation and staining of the blood film or other influences.

In the non-stained hæmatozoon no structure whatever can be detected. When stained according to Laveran's method, however, a deeply red stained body, the karyosome, can be observed in the slightly blue tinged protoplasm. This karyosome also shows different shapes; it is never in the centre, but always close to the edge. Very often around this nucleus a clear unstained or only slightly stained zone can be detected.

Reproductive forms may be found in unstained preparations, but they are more frequently seen in the stained preparations. When ordinary aniline dyes are used, a form somewhat resembling a rosette, with four leaves, can be seen connected to a centre by very fine threads. When stained by Laveran's method these forms show a karyosome in every leaf. It must be stated here that the rosette form is better shown by using the ordinary single dye than when the double stain is used, where all forms of parasites are more universally spherical. Leaf-like bodies into which the parasite splits are preceded by forms where, in a quadrangular or round mass of generally bluish-stained protoplasm, four distinct karyosomes can be observed lying crossways, thus indicating the subsequent fission with the four leaves. Besides the fission into four parts, a division into two can also be observed, but in my experience the rosette form is the most commonly found. Reproduction takes place in the circulating blood as well as in the organs, but is very frequently met with in the spleen, where the parasites may also be seen outside the red blood corpuscles. In the method of reproduction the pyroplasma equi differs somewhat from the pyroplasma bigeminum; in the latter parasite the division into two is the common way of reproduction, whereas the division into four is the rarer method.

Lignières states in a paper upon "Tristezza" that he succeeded in cultivating the pyroplasma bovis outside the animal body in cattle serum containing hæmoglobin. For this purpose he takes the blood of cattle suffering from the disease which shows abundant parasites, and distributes the blood into test tubes; he finds that in some of them a veritable proliferation takes place, which continues when fresh serum containing hæmoglobin is inoculated with the serum showing the proliferating forms.

I have on several occasions kept blood from a horse suffering from equine malaria, and which contained the pyroplasma, in test tubes at the temperature of the ice box, of the room, and of the incubator. I have found that the parasites disappear from the blood kept in the incubator as soon as the corpuscles lose their hæmoglobin; that they keep their colorability longer at the temperature of the room than at that of the incubator; and that even after twelve days specimens kept in the ice box showed the pyroplasmata as distinctly as fresh ones.

It was seen, however, that the variety of shape of the parasites had disappeared, or was only exceptionally seen, and that nearly all appeared spherical. It was noticeable that the parasites were commonly seen at the edge of the red discs, but never or very exceptionally outside the blood disc. Some of the red discs which contained the pyroplasma did not take the stain in the immediate neighbourhood of the parasite, so that it seemed to be embedded in a white zone. It is possible that this phenomenon indicates a

destruction of the hæmoglobin by some excretions of the parasite. As already mentioned, pyroplasmata were seen exceptionally in the surrounding serum. In the red corpuscles kept in the ice box, however, the reproductive form could be observed as long as four days after the removal of the blood from the horse, and the interpretation of this observation would be, that the reproduction continued even in corpuscles out of contact with the animal from which they are derived. The possibility of staining by Laveran's method both karyosoma and plasma distinctly after twelve days in the ice box, and not so after the same length of time in the incubator, indicates that the pyroplasma in the former case must have been still alive, the colorability of a nucleus being an indication of its being alive. Although transplantations into fresh serum were made, a proliferation, as mentioned by Lignières, could not be seen.

Pyroplasma equi is always present in equine malaria, and can be constantly found when the blood is examined early in the disease. It is, however, not constantly present during the whole course of the disease, and it is quite possible that one may diagnose a case as one of typical biliary fever and a subsequent microscopical investigation may fail to reveal the presence of any intra-corpuscular parasite; yet notwithstanding the total absence of the parasite the horse may die. I shall refer to this when I deal with the sequelæ.

The administration of quinine and chloride of ammonium results in a rapid disappearance of the pyroplasma, but I have on the other hand also seen a similar rapid disappearance in horses which were not treated at all, because, from the general condition of the horse no treatment was necessary. In all these instances the clinical symptoms of malaria were present for some time after the parasite had apparently disappeared.

Upon one occasion I have seen what appeared to be a karyosoma persist for a considerable period, and I have little doubt that careful observation will show that the phenomenon will not be altogether unusual. The following is the account of the case:—

5th March 1902. Brown English hackney mare, belonging to Dr T., shows symptoms of biliary fever; temperature  $105^{\circ}$  F., seedy, yellow conjunctival mucous membranes of the eye, urine yellowish, and dung of the same colour. Treated with quinine. Blood microscopically examined shows the presence of numerous parasites; all the previously described forms are present, especially the multiplication forms.

6th March 1902. Same symptoms as the day before, no improvement. Quinine is given three times a day, and chloride of ammonium in a half ounce dose.

7th March 1902. Same symptoms, but the animal shows marked improvement: treatment continued. Pyroplasmata appear to be less numerous in the blood.

8th March 1902. Symptoms of jaundice are still very marked, but the mare has improved, feeds very well, and is very lively. She stales frequently and abundantly, the urine being of a yellow colour. Parasites are very rarely met with in the blood; at the commencement from five to twelve parasites could be detected in each microscopic field, now as many fields must be searched in order to discover one parasite.

The mare recovered, but the blood was examined daily up to 24th March 1902 (*i.e.* for nineteen days). During the whole of this period there was constantly present a round body in a red corpuscle, which, when stained by Laveran's method, was of a deep red colour. This reaction to the stain shows that the body is of the nature of a karyosome. No protoplasmic tissue could be traced, not even after using different stains. This chromatic body was of the size of the karyosoma in the full developed parasite previously described; a further peculiarity was that at the beginning it could only be found in the so-called macrocytes, or corpuscles of about double the size of an ordinary erythrocyte: and it was not until towards the end of the observation that normal-size blood discs containing these bodies could be observed. It may be stated here that in this mare's blood both megaloblasts (*viz.*, large corpuscles with a nucleus) and macrocytes were repeatedly found, indicating the acute anaemia which was caused by the invasion of pyroplasma. But the macrocytes disappeared rapidly after recovery. More than one body in one corpuscle was never seen: this fact would exclude the possibility of its being simply a basic alteration of the hæmoglobin.

I consider the chromatic body to be one of the forms in the development of the parasite, and have no hesitation in thinking that it represents a latent or dormant form. For this reason I have eschewed the term spore.

*Inoculability of the Disease.*—In order to prove conclusively that a given micro-organism is the *causa causans* of a disease which is characterised by its presence, the inoculation of it into other animals should produce the same disease again, provided the animal operated upon is not immune against the disease. I repeatedly injected horses with the blood of equine malaria which contained the pyroplasma, but up to the present I have been unable to transmit the disease in this way from one animal to another. At first I injected small quantities of blood subcutaneously, later intravenously into the jugular vein; then, seeing that all these efforts failed, simply connected the jugular vein of a healthy horse by means of indiarubber tubing to the vein of a sick one, and let the blood run into the former for a period of two minutes. The amount of blood thus infused I calculated to be about one litre. Even this procedure failed to reproduce the disease. This failure is somewhat disheartening, seeing that the other diseases caused by pyroplasmata, namely pyroplasma bigeminum and pyroplasma canis, can be transmitted by injection of blood which contain them. But it must be borne in mind that these two diseases of cattle and dogs, respectively, can only be transferred in this way to *susceptible* specimens of their species. For instance, I have repeatedly tried to produce redwater by injecting virulent blood into oxen and calves born in and around Pretoria (a badly tick-infected area), and I have never succeeded in producing the disease, whereas freshly imported cattle from the Cape peninsula easily contract the disease. The same fact has been repeatedly proved for cattle born in other redwater countries.

This analogy points to the fact that my experimental horses must have had an immunity against biliary fever; in fact, all the animals experimented upon were South African veldt horses, and it is not improbable that they had either an acquired immunity, from a

previous attack of the disease, or a natural immunity. In order to show that a horse can be infected by the injection of infected blood, freshly imported horses will have to be injected. The failure to infect South African horses with equine malaria—by analogy with the other pyroplasmic diseases—would point to a high degree of immunity in the locally bred horses.

The question arises, how does a horse become infected by the *pyroplasma equi*?

The conclusion has again to be drawn from the analogy of the similar diseases in cattle and dogs. It has been proved that redwater is carried from a sick animal to a healthy animal by ticks. In America it was found that the tick *rhhipicephalus annulatus* is the propagator, and in Australia the tick *rhhipicephalus australis*. In these countries the disease has always followed the ticks, and the conclusion drawn therefrom could be proved by experiments. In South Africa the tick was known before redwater, and it only became infected after the importation of cattle suffering from the disease. The farmer, therefore, would not allow that the ticks had anything to do with the disease, and generally ridiculed the idea. It has, however, now been proved that the common blue tick of South Africa, *rhhipicephalus decoloratus* (Koch), is really the propagator of redwater. Recently the entomologist of Cape Colony, Lawnsbury, has proved that malignant malaria in dogs is due to the tick *hæmophysalis leachi* (Audin). The experiments with ticks show that the female tick transmits the infection into the eggs, from which it passes into the larvæ and shows in the pupa. Larvæ and pupæ can feed on healthy dogs without transmitting the disease, but as soon as they moult the imago or adult tick transmits the disease.

We have to conclude therefrom that in the different stages of the tick the *pyroplasma* undergoes changes of form in the process of reproduction.

It has been shown that the resemblance between the different forms of pyroplasmic diseases is very great; for two of them it has been proved beyond doubt that they are transmitted by ticks; the conclusion is, therefore, that the propagation of the third disease, caused by pyroplasmata, viz., malarial fever in horses, is also due to the same agent. It should be possible to prove this by experiment, but, as I have said, I have up to now been unable to do so, on account of the want of freshly imported horses. Observations made during the recent campaign go to support the idea. We noticed that biliary fever was nearly exclusively confined to imported horses; nor did it seem to matter from what country the horses were derived, for, so far as I can find from the literature, malaria in horses is unknown in all the countries from which they were derived. I had the opportunity on several occasions of observing that imported English horses which were in Pretoria, and which were kept under circumstances similar to those at home, viz., stabled and properly groomed, fell sick with biliary fever when they were turned out to graze and allowed to become tick-infected. In two instances the observation was so striking that it could be quoted as an experiment. The ticks commonly found, viz., the blue ticks (*rhhipicephalus decoloratus*), are very likely also responsible for the propagation. This arachnid has already been shown to convey redwater. The question, therefore,

whether these two diseases are identical naturally arises. The pathological appearances are also very similar, and the fact that hæmoglobinuria is not seen in horses can be explained in the same way as those cases of redwater in which the discolouration of the urine is absent, namely, to a greater resistance of the red corpuscles. Although I do not believe that the two diseases are identical, I inoculated horses several times with blood from cattle suffering from redwater without ever noticing any subsequent disease. The geographical distribution of horse malaria and redwater, moreover, points out that they do not result from the same cause. According to Hutcheon, biliary fever can be observed in the Cape Peninsula, a territory which, as far as I am aware, is not yet infected with redwater.

I also tried injecting blood from horses suffering with malaria into cattle, but without effect; and I have at different times tried to infect the ordinary experimental animals, such as rabbits, dogs, and guinea-pigs, but I failed to produce any reaction. The conclusion may therefore be drawn that malarial fever in horses is a specific disease peculiar to the species *equus caballus*. The injection of the blood of horses suffering from malaria into the peritoneal cavity of guinea-pigs was without effect, whereas the blood from cattle containing pyrosoma bovis had an immediate effect on these animals, producing symptoms of spasm, cramps, and even sudden collapse.

Before concluding these notes on pyroplasma equi, I wish again to refer to the immunity of horses against this disease. I have no proof that malarial fever leaves any immunity whatever, but we must assume that such is the case. The immunity of South African veldt horses has, however, to be explained differently, and not by assuming that they have really passed through the disease. The analogy to redwater again gives the clue to the solution. We know that calves possess a great resistance against redwater, and when they are born in a redwater country we know that they acquire immunity which lasts for a considerable time, probably for life. This is due to the constant infection during the early life, when they are already very resistant. The same will hold good for biliary fever. The foal, whilst running with its mother in the fields, becomes tick infected, and whilst highly resistant on account of its youth is gradually vaccinated. When full grown it is thus immunised against the disease.

### *Sequelæ of Malarial Fever.*

From the above notes it can be seen that I consider that the pyroplasma equi is the *causa causans* of biliary fever. I have stated that it is to be found in the early stages that it is not found in healthy horses or in horses suffering from other diseases; and I have demonstrated how quickly it leaves the animal body (the resistant forms excepted) when the horse is recovering. In some horses which do not recover I have noticed that the pyroplasma is present in the blood and internal organs up to the time of death; in others, however, it is absent.

It is exceedingly rare to find that only the pyroplasma is found in a horse suffering from and dying of biliary fever. In nearly every such case I found a bacterium which was present sometimes in

the blood, and always in the spleen, and which very often could be traced *intra vitam* by the agglutinating properties of the serum of such animals as were infected by it.

Before entering, however, into the life history and the characteristics of this bacterium, it will be advisable to quote illustrative cases.

(1). 14th July 1900. Argentine gelding, six years old. This horse was observed to be ill, temperature  $40.5^{\circ}$  C. The pulse was very rapid and feeble. The respiration was accelerated, and finally "pumping." The mucous membranes were spotted with red. On auscultation crepitation could be distinctly heard. The animal died during the night.

A *post-mortem* was made early in the morning. The lungs were in a state of inspiration. The interlobular tissue was œdematous at the apices and roots of the lungs and at the base of the heart. The bronchi were filled with white foam. The blood was coagulated. In the right ventricle a solid clot was found, and there was a smaller one in the left one. A few ecchymoses were found in the endocardium. The spleen was much enlarged; the pulp, however, was fairly solid. The stomach was normal. In the small intestines a few red patches were seen and injection of the blood vessels. The liver was jaundiced. A general slight jaundiced condition of all the organs was present.

The microscopic examination showed the absence of endoglobular parasites; in smear preparations from the spleen the bacterium could be found, and cultivations made from the blood and the spleen on agar-agar proved it to be present in pure culture. From the state of the lungs, some doubts were entertained whether this was not a case of "dunkop" (horse-sickness), although the season in which it occurred was against it. Some blood was therefore injected into a horse; horse-sickness did not follow, however; and, as the experimental horse died from a subsequent inoculation from horse-sickness, it was proved that the lesions in the lungs of the former horse had nothing to do with this disease.

(2). 2nd August 1900. Chestnut gelding, seven years old. Arrived the day before into the sick lines with symptoms of "pulmonary œdema." The respiration was very much accelerated and the nostrils wide open. Crepitation could distinctly be heard in both lungs. The pulse was imperceptible. The heart beats exceeded 80 per minute. The conjunctival mucous membrane showed ecchymoses. Temperature  $38.5^{\circ}$  C. There was a serous discharge from the right nostril. Blood was taken both for examination and culture. The horse died during the night.

*Post-mortem* early in the morning. The lungs were in the extreme of inspiration. The bronchi were full of foam. On the inner surface of the lungs on both sides it was seen that the pleuræ were infiltrated with yellowish lymph. The interlobular tissue at the edges of the apex were distended with the same lymph. The mediastinal glands and those of the lungs were enlarged and gorged with serous fluid. The pericardial sac contained serous fluid. The heart was one mass of petechiæ; the heart muscle between the hæmorrhages was of a greyish colour, and appeared as if it had been boiled. In both ventricles were yellow clots of coagulated blood. The liver was enlarged, of a dark yellow colour, and had a granular appearance. The bile



ducts were gorged with greenish-looking bile. The spleen was very much enlarged, the pulp fairly hard; the lymphatic glands were enlarged and hæmorrhagic. The kidneys were normal. There was no pathological change in the stomach or intestines.

The microscopic examination of the blood and spleen showed the absence of endoglobular parasites.

No cultures were made from the spleen, but from the blood the bacterium was found in pure culture.

There was a doubt whether the case was not one of horse-sickness. Two experimental horses were therefore injected with blood, but horse-sickness did not follow the injection.

(3.) 26th August 1900. English mare. This mare had been some time in the sick lines, and had been treated for a wound on the left fetlock. Beyond the wound nothing was noticed to be wrong with her until the morning of the 25th August, when she was found on the ground, with symptoms of spasm, which returned periodically. The head and legs were alternately drawn up under her belly and suddenly extended. A peculiar rolling of the eyes could be noticed, thereupon the horse moved its legs as if to walk, finally it became comatose and died during the night.

*Post-mortem* early next morning. The mare was in pretty good condition. With the exception of a very large spleen, nothing pathological seen with the naked eye. From both ventricles long yellow clots of coagulated blood were drawn.

Microscopic examination of smear preparations from the spleen showed the presence of the bacterium, which was cultivated in pure state on agar-agar.

(4.) 15th September 1900. English gelding, eleven years. This horse arrived the previous evening in the sick lines and died shortly afterwards.

The *post-mortem* was made early on the morning of the 15th. A general jaundiced condition of the flesh and all internal organs could be noticed. The spleen was enormously enlarged; it reached backwards nearly as far as the ileum. The pulp of the spleen was soft and black. The liver was also enlarged and yellow, the bile ducts filled with bile. The kidneys were anæmic. The lymphatic glands of these mentioned organs were enlarged with blood infiltrations. The urinary bladder contained brownish-looking urine. The stomach and intestines were empty; the mucous membrane of the stomach was very pale; the mucous membrane of the small intestine looked like eel-skin. The lungs were in inspiration, contained much blood, and were œdematous. The heart was found in diastole, and was filled with black blood clot. Preparations of the spleen showed the presence of the bacterium.

(5.) 3rd October 1900. Chestnut gelding, eight years old. Died to-day with convulsions.

*Post-mortem* about three hours later. Condition good. The spleen was about three times its normal size, the pulp was very soft; its lymphatic glands enlarged and infiltrated with blood. The liver was also enlarged and very yellow. The kidneys were normal. The stomach was normal; the mucous membrane of the small intestine was of slate colour, and that of the colon was deep black in parts. The lungs showed emphysematous spots, but were otherwise normal.

The heart was in diastole, and both ventricles contained well coagulated clots. The myocardium looked as if it had been boiled.

Microscopic examination of smear preparations showed the absence of endoglobular parasites, and the presence of the bacterium, which grew in pure culture on agar-agar.

(6.) 8th October 1900. Brown pony gelding. Arrived in the sick lines on the previous evening with a temperature of 103·6° F. and died soon afterwards.

*Post-mortem* was made early on the 8th. The skinned cadaver and all internal organs showed a generally jaundiced condition. The spleen was enormously enlarged, its weight being 8·5 lbs., and the pulp was soft. The liver was also enlarged, very yellow, and friable. Both kidneys were enlarged and very pale. The lymphatic glands of these three organs and of the stomach were enlarged and infiltrated with blood. The mucosa of ileum, jejunum, and colon was of a slate colour; the pericardial sac was filled with brownish-yellow fluid. The myocardium looked as if it had been boiled. Subendocardial petechiae were very marked in the left ventricle. Both ventricles contained yellow clots.

In smear preparations of the spleen no endoglobular parasites could be seen under the microscope, but cultivations made on agar-agar proved the presence of the bacterium in pure culture.

(7.) 24th October 1900. English chestnut mare, nine years old. She showed all the symptoms of biliary fever, and the microscopical examination of the blood proved the presence of pyroplasma equi. Symptoms of pneumonia were also present, and the neck was swollen. The horse died in the night.

*Post-mortem* was made about nine hours later. There was a generally jaundiced condition of the flesh and all the internal organs. The spleen weighed 9 lbs. 4 ozs.; the pulp was very soft. The liver was enlarged and yellow. The kidneys very pale. The pericardium was covered with a yellowish dry mass. In the pleural cavity serous fluid was found. The lungs around the heart were in a state of engorgement, and surrounded by oedematous infiltration. The pleura at this particular point was thickened and injected. Foam was present in the trachea. In both ventricles were yellow clots. The myocardium appeared as if it had been boiled, and was very friable. All the lymphatic glands were enlarged and infiltrated with hæmorrhages.

Smear preparations from the spleen showed the presence of the pyroplasma equi, and the bacterium was cultivated in pure culture.

(8.) 24th October 1900. English chestnut mare, thirteen years old. Died very suddenly in the night.

*Post-mortem* early in the morning. The mare was in pretty good condition. Rigor mortis was present. The spleen was very much enlarged, and weighed 8 lbs. 2 ozs. The mucosa of the stomach was of a slate colour. The small intestines were slightly congested. The liver was very large and congested. In the left ventricle, some white fibrous streaks. The remainder of the organs were normal.

The bacteria were found by culture in a pure state.

(9.) 1st November 1900. English mare, seven years old. On 24th October 1900 the diagnosis of biliary fever was made both clinically and by microscopic examination. The horse was treated with quinine, and showed improvement for some days. On the 28th

October no more pyroplasmata could be found in the red corpuscles, and the animal had a very good appetite. On the 29th October, however, a change for the worse took place; the evening temperature rose to 40° C., the hind legs began to swell, and the pulse was very feeble. The animal died during the morning of the 1st November.

*Post-mortem* was made about one-and-a-half hours after death. The cadaver was in a poor condition. The flesh had a brownish tint. All the fasciæ and serous membranes were yellow. The blood was very well coagulated. The peritoneum contained yellow fluid. The spleen was slightly enlarged, and weighed 2 lbs. 4 ozs.; the pulp was of a brownish colour and rather hard. The lymphatic glands of the spleen were enlarged and infiltrated with hæmorrhages. The liver was jaundiced and contained much blood. Both kidneys were enlarged and surrounded by œdematous capsules; the cortex seemed to be broader than usual and yellow. The subrenal glands were swollen. The stomach and intestines were empty. The mucous membrane of the colon was slightly reddened. The lungs were in inspiration and emphysematous at the edges. In the middle of the right lobe there was a pneumonic patch. The left ventricle contained subendocardial hæmorrhages.

Microscopical examination of smear preparations made from the spleen showed the absence of endoglobular parasites and the presence of the bacterium.

This bacterium also grew in cultures made from blood taken on the 27th October, on which day a striking decrease of endoglobular parasites could be noticed.

(10.) 2nd November 1900. English gelding, eight years old. This horse arrived in the sick lines the previous day with a temperature of 105·4° F. The respiration was accelerated; auscultation revealed increased vesicular sound on both sides. The pulse was 60. Conjunctiva bulbis was yellowish, with hæmorrhagic spots. The lower part of the head was swollen, and the head was held in a hanging position. In the evening of the same day the thermometer registered 104° F., the swelling increased. The horse became comatose and died in the morning of 2nd November 1900.

The *post-mortem* was made one-and-a-half-hours after death. Cadaver was in a poor condition. The flesh and all the internal organs were jaundiced. The blood was only partially coagulated. Incutting the axillary vein a brown serum ran out. The spleen was enormously enlarged and weighed 10 lbs. The pulp was black and protruded when a cut was made into the capsule. The liver was also enlarged, of a brownish-black colour, and strongly congested. The kidneys were enlarged and also of brownish-yellow colour; the cortex was pale. All the lymphatic glands were enlarged and infiltrated with blood. The pericardium contained serous fluid. The heart was flabby, and there were subendocardial hæmorrhages in the left ventricle. The lungs were emphysematous. The mucous membrane of stomach and intestines was brownish-yellow; no hæmorrhages were present, but there was a layer of tough mucus.

Microscopic examination of blood from the heart and of smear preparations from the spleen showed the absence of the pyroplasma equi. The bacterium, however, could be found in cultures made from the blood, spleen, kidney, and liver.

(11.) 4th January 1901. Horse 1050. English gelding, dun-coloured, nine years old. This animal arrived four days previously into the sick lines with high fever. The mucous membranes were yellow, and in the course of the succeeding days became spotted with hæmorrhages. The pulse was very bad from the beginning. The condition changed rapidly for the worse. Swellings appeared around the mouth, and a yellowish diarrhœa set in. The horse died in the night of the 3rd January.

The *post-mortem* was made early in the morning of the 4th. The cadaver was in poor condition. Rigor mortis was present. The flesh was sepia-colour. The spleen weighed 5 lbs; its lymphatic glands were enlarged, œdematous, and partially infiltrated with blood. The liver was enlarged and yellowish-brown, of nutmeg character; all the lymphatic glands were enlarged and œdematous. The kidneys were enlarged and very pale. The mucous membrane of the small intestines showed slate colour. The lungs were in inspiration, and at both apices were capsulated abscesses. The heart muscle was also of a sepia-colour. In the left ventricle were numerous subendocardial hæmorrhages.

Microscopic examination showed the absence of the pyroplasma equi in smear preparations of the spleen. The bacterium was obtained therefrom in pure culture.

(12.) 5th January 1901. Horse 17. Gelding, eight years old. This animal was for some time in the sick lines suffering from laminitis. It was seen to be very ill. The respirations were accelerated and pumping, the pulse imperceptible, temperature  $41^{\circ}$  C., mucous membrane of the eye reddened. The horse was obviously dying; it dropped and showed convulsions of the neck and legs, and it was therefore killed.

*Post-mortem* about one hour later. The condition was good. The spleen was enlarged and weighed 5 lbs; the pulp was soft. The kidneys were enlarged. The malpighian bodies were very distinct. The lymphatic glands of the kidneys were very much enlarged and infiltrated with hæmorrhages. The liver was greatly congested and of a dark brown colour. The mucous membrane of the small intestines was reddened in patches and injected. The lungs were in semi-inspiration, and spotted with small hæmorrhages. The heart seemed to be normal. The blood was not properly coagulated and was black.

From the spleen the bacterium was obtained in pure culture.

(13.) 19th January 1901. English mare, nine years old. This horse was for some considerable time in the sick lines, and was in very poor condition. A definite diagnosis was, however, not possible. It became lame all of a sudden on the near fore leg, and at the same time an acute iritis was observed. The animal was killed on the 19th January 1901, as *exitus letalis* was anticipated.

*Post-mortem* immediately after death. In the lungs numerous small encapsulated abscesses and emphysematous spots could be observed. The endocardium of the left ventricle was thickened and white. Slight tumour of the spleen was present; its weight was 4 lbs. The kidneys were dark red and markedly congested; there was some difficulty in removing the capsule. In the medulla white streaks extending into the cortex could be seen. The liver was enlarged and

friable. The stomach and bowels were normal. Under the coronary band an abscess was found. In the frontal chamber of the eye was a yellow coagulum.

Cultures made from the spleen gave the bacterium in pure state.

(14.) 12th March 1901. Horse 34. Gray mare, ten years old. This horse arrived in the sick lines on 2nd March, when both clinically and microscopically bilious fever was diagnosed. It was treated at once with quinine, and the pyroplasma soon disappeared. It did not, however, improve, but a general marasmus supervened. The horse died in the night of the 12th March 1901.

*Post-mortem* was made early in the morning. The cadaver was thin. The flesh was of a yellowish colour. The spleen was enlarged and weighed 4 lbs.; the pulp was soft and of a brownish-yellow colour. The liver was enlarged and nutmeg in character, very fatty and friable. The kidneys were enlarged and pale, not unlike the colour of burnt sienna. The stomach and bowels were normal. The lungs were œdematous. On the surface of the left lobe was a circumscribed pleuritis fibrosa and fibrinosa, underneath a grey pneumonic patch. In the left ventricle were subendocardial hæmorrhages. The pericardium was injected.

In smear preparations from the spleen only bacteria were found, which were obtained in pure culture.

(15.) 21st January 1902. Australian mare, eight years old; in very poor condition. The mucous membranes were very pale and showed petechiæ. The horse died during the night of 20th January 1902.

The *post-mortem* was made early next morning. A general anæmia of all organs was very well marked. The endocardium was full of hæmorrhages. The spleen was enormously enlarged, and the pulp soft.

Smear preparations from the spleen showed the absence of the pyroplasma. The bacterium was obtained in pure culture.

(16.) 12th February 1902. English mare, six years old. This horse showed clinically all the symptoms of biliary fever. Microscopical examinations proved the presence of the pyroplasma equi. The horse was treated with quinine and stimulants, and under this treatment it improved. A change for the worse took place on the 19th, and the horse died on the morning of the 20th February 1902. During the whole time the pyroplasma equi was present in the blood, but was less numerous at the end of the disease than at the beginning.

The *post-mortem* was made shortly after death. The blood was well coagulated. The flesh was of a brownish-yellow colour. The spleen weighed 7 lbs.; its pulp was soft, and the lymphatic glands were enlarged and infiltrated with blood. The liver was enlarged, of a reddish-brown colour, and of nutmeg character. The kidneys were pale. In the stomach and bowels nothing pathological could be found. The lungs were in half inspiration and were slightly œdematous. The heart muscle appeared as if it had been boiled. There were subendocardial hæmorrhages in the left ventricle; all the lymphatic glands were generally enlarged.

In smear preparations of the spleen the pyroplasma equi was present; the bacterium was obtained in pure culture.

(17.) 13th March 1902. Chestnut pony gelding. This horse

arrived some time previously from Heidelberg, where it had been suffering from bilious fever five weeks before this second disease. For a few days it had high fever; it was impossible, however, to arrive at a diagnosis. The mucous membranes of the eye were very pale and spotted with hæmorrhages.

The animal died during the day of 13th March 1902, and a *post-mortem* was immediately made. The blood was very thin and of a greenish colour, containing when defibrinated a very small deposit of red corpuscles. The cadaver appeared as it had been bled to death. The spleen was enormously enlarged and weighed 13 lbs., 10 ounces; the lymphatic glands of the spleen were enlarged and infiltrated with hæmorrhages. The kidneys were œdematous, enlarged, and very pale. The liver was very friable and fatty. The myocardium was of a sepia colour, and very friable. Subendocardial hæmorrhages were present. The mucous membrane of the stomach had a few erosions which were covered with coagulated blood. The mucous membrane of the bowels was swollen and had a yellowish glass-like appearance. In the colon ecchymoses could be found on the mucous membrane. The lungs were also spotted with very fine hæmorrhages.

Microscopic examination of specimens from the different organs showed the absence of any endoglobular parasite. From the spleen the bacterium was obtained in pure culture.

(18.) 22nd March 1902. Horse. S.A.C., dun. This animal had been suffering for some time from malarial fever. A diagnosis to this effect was made by Vet. Captain Christy of the S.A.C.

*Post-mortem* was made about two hours after death. The cadaver was in very poor condition and anæmic. The flesh and all the organs had a generally jaundiced condition. The spleen was very large and soft. The liver was also enlarged, quite yellow, and friable. The kidneys were pale, but looked otherwise normal. In the lungs capsulated abscesses were found.

Smear preparations from the spleen showed the absence of the pyroplasma equi. Cultures showed the bacterium.

### *Analysis of the foregoing Cases.*

These sixteen cases may be divided into four groups:—

(1.) The cases in which the pyroplasma equi was found in the blood both during life and *post-mortem* (4, 7, and 15).

(2.) The cases in which the pyroplasma equi was found *intra vitam*, but was not found *post-mortem* (8).

(3.) The cases in which no pyroplasma was found shortly before death, and none *post-mortem* (1, 2, 5, 6, 9, 10, 16, and 18), but where the characteristics of the *post-mortem* lesions left no doubt that the animals had suffered from biliary fever.

(4.) The cases in which no pyroplasma was found on *post-mortem*, and where the characteristic lesions were not so pronounced as to justify a certain diagnosis of biliary fever (3, 11, 12, 14). I consider these cases, notwithstanding the absence of the most striking symptoms of biliary fever, as due to previous biliary fever, and I base my opinion on the presence of the splenic tumour which was found in all cases, and upon the general degenerations of the internal organs. In these particular cases icterus was absent.

*Description of the Bacterium.*

Since the presence of the particular bacterium of which I have spoken was demonstrated in every case, although the pyroplasma equi was absent in the majority of these cases, a description of the bacterium may here be given before discussing the question of experimental pathogenesis.

*Morphology.*—It is a small rod, with rounded edges, resembling the so-called "coccobacillus," and is about the size of the bacillus hæmorrhagicus (chicken cholera). The size varies, however, according to the medium upon which it is grown. The bacillus may therefore appear to be of a iso-diametrical form, not unlike a coccus, and as a rod, about three to five times as long as broad. The former are usually observed when cultivated in broth. The staining is very often most marked at both poles, especially in young forms, and the bacilli in smear preparations of the spleen and blood show typical bipolar staining. The bacillus takes all the basic stains, but the best preparations are obtained with fuchsin gentian violet; methylene-blue stains very faintly. In smear preparations the best results are obtained with carbol-thionin blue. When treated by Gram's method the bacillus does not retain the stain.

*Motility.*—The bacillus shows distinct though not rapid motility.

*Cultural Reactions.*—When the spleen juice or blood is first inoculated the growth is not abundant. Several drop-like colonies appear, which gradually increase in size, and eventually become wrinkled on the surface. Subsequent sub-cultures result in abundant growth.

*Broth.*—This medium soon becomes turbid, and sometimes a pellicle is formed on the surface: a deposit is constantly formed, which is considerable in old cultures.

*Gelatine.*—It grows very slowly in stab-cultures in this medium, and never liquefies it. Along the stab a row of fine isolated colonies can be observed. On gelatine plates only very small drop-like colonies can be observed.

*Agar.*—On slanting agar the growth is very abundant after the first generation. It begins with a number of very fine drops, which become confluent; the resulting growth may become wrinkled when the growth does not reach the glass, or the growth may be thin and with a bluish iridescent lustre. Under all circumstances the growth is transparent.

*On glycerine agar* the growth is typical. The shrinking is extremely pronounced, and the layer is nearly translucent. The water of condensation in both agar and glycerine agar tubes becomes only slightly turbid. When touched with the platinum needle whole pieces of the shrunk culture can be removed. After several sub-cultures, however, this particular characteristic is lost, and the growth has an even surface and is translucent.

Grape-sugar agar, milk-sugar agar, and cane-sugar agar become broken up when inoculated. This is very pronounced in grape-sugar agar.

Milk is as a rule not coagulated. One sample, however, did so.

On potato there is a growth of a slight brownish colour, and the potato itself is subsequently stained the same colour.

On coagulated blood serum a translucent growth without any special characteristics is to be observed. In liquid serum the growth takes place only at the bottom of the test tube, and the serum never becomes turbid.

The bacillus is an obligatory aerobe.

No indol is formed.

*Inoculation of the Culture into Horses.*

(1.) 30th October 1900. South African pony, six years old. This animal was inoculated into the jugular vein with 18 cc. of a bouillon culture, fourteen days old, two generations from the original culture. Directly after injection most alarming symptoms were observed: accelerated respiration, colic, diarrhoea, and collapse, which ended in death after eight hours.

The *post-mortem* was made at once. The spleen weighed 2 lbs., 10 ounces. The liver was enlarged and strongly congested. The kidney was also markedly congested. The mucous membrane of the colon and cæcum had a generally reddened appearance, with still deeper red patches. The mucous membrane of the small intestines was here and there of a slate colour. The mucosa of the stomach was slightly reddened. The lungs were emphysematous. In the left ventricle there were subendocardial hæmorrhages. The bacillus was again recovered from the spleen.

(2.) 8th November 1900. Horse S. H. English mare, six years old. Injected with 5 cc. of a bouillon culture into the jugular vein. Symptoms of colic soon supervened; the respirations were accelerated, the temperature rose to 39·8° C. The horse seemed to be all right again on the following day.

20th November 1900. The animal received 20 cc. of a bouillon culture, four generations from the original, into the jugular vein. Again signs of uneasiness were noticed, and the temperature rose to 40° C, and during the next two days kept oscillating, always slightly above normal.

30th November 1900. The animal had completely recovered, and was again injected into the jugular vein with 20 cc. of a bouillon culture. Only a slight uneasiness could be observed, and the temperature never rose above 39·5° C.

12th December 1900. On this day the animal was injected with 40 cc. of a bouillon culture about one month old. The inoculation was made very carefully and at intervals. The animal, however, showed the most alarming symptoms of congestion of the lungs, and died suddenly a few minutes after the injection.

The *post-mortem* was made immediately. Hyæmia of the lungs was present. The spleen weighed 2 lbs. 12 ounces. All the organs were normal. The bacillus could not be found in the spleen by culture.

(3.) African pony, twelve years old. This horse received on 12th March 1901 30 cc. of a culture taken from the horse which died on 27th February 1901. Directly after the injection symptoms of colic came on, and the horse died during the night.

The *post-mortem* was made about twelve hours later. The whole of the small intestine showed hæmorrhagic inflammation; the mucosa of the stomach was also reddened. Some parts of the



intestinal mucosa was of a slate colour, among other parts the whole of the cæcum. The kidneys were markedly congested, and the surrounding tissue oedematous. The subrenal glands showed hæmorrhages on section. The spleen was slightly enlarged and soft; the lymphatic glands of this organ were enlarged and infiltrated with blood. The liver was hard. The lungs were normal. The heart was in diastole. The blood was badly coagulated. In the right ventricle were a few hæmorrhages.

(4.) 19th October 1900. Horse 27, pony four years old. 20 cc. of a broth culture of the second generation were injected into the jugular vein. This was followed by very alarming symptoms, accelerated respiration, and diarrhœa. The temperature rose above  $40^{\circ}$  C, and remained so for a few days. General depression was also observed, but the horse finally recovered.

(5.) African pony. This horse was injected into the jugular vein on 18th October 1900 with a culture (third generation) four days old. 9 cc. were injected. Symptoms of a slight colic with repeated evacuation of the rectum followed, but the animal recovered.

(6.) Horse 45, a pony. Injected 22nd October 1900 with 10 cc. of a bouillon culture into the jugular vein. The culture had been first passed through a rabbit. Symptoms of colic followed, the animal lay near constantly, and the temperature rose to  $40^{\circ}$  C.

(7.) Horse 32. Six years old, grey gelding. On 30th October 1900 it was inoculated with 18 cc. of a bouillon culture, five days old. The injection was made subcutaneously. Only slight evidence of uneasiness was observed. At the site of inoculation a large swelling was formed, apparently very painful to touch. The bacillus was obtained from the swelling in pure culture.

On the 5th November 1900 this animal was injected into the jugular with a further 18 cc. of a bouillon culture, taken from a rabbit. Symptoms of uneasiness supervened. The temperature rose the next day to  $40.8^{\circ}$  C. On the morning of the 7th November the animal was found in the paddock in a comatose state, entangled in wire fence, and was therefore killed.

The *post-mortem* showed no characteristic changes. Bacteria were found in the spleen.

(8.) 11th March 1901. Horse 85, South African mare. The object of this experiment was to predispose the horse to the action of the bacterium. For this purpose blood was withdrawn, then the jugular of horse No. 34, which was suffering from malaria sequelæ, was connected with the jugular of horse 85 by india-rubber tubing, and for a few minutes the blood was allowed to run from horse 34 into horse 85. The infusion was followed by no untoward symptoms. At the same time 85 cc. of the blood of horse No. 34, collected and defibrinated the previous day, were injected subcutaneously.

13th March 1902. Horse 85 was injected subcutaneously with 30 cc. of a bouillon culture three days old. Symptoms of a very acute disease soon developed. The pulse was  $76^{\circ}$ , the temperature rose to  $103.8^{\circ}$ , the respirations to  $68^{\circ}$  and very superficial. The mucous membrane of the eye became very red. The mare ceased to feed, and became semi-comatose. The temperature for the next day kept at  $104.4^{\circ}$  in the evening, and the pulse rose to  $90^{\circ}$ . Respirations receded to  $26^{\circ}$ . The animal, however, recovered. The site of in-

oculation was marked by a swelling of the size of two hands, and was hot and painful. It did not suppurate. This horse was again injected, and after this injection suppuration and necrosis of the subcutaneous tissue supervened.

(9.) Grey gelding, fifteen years old. On the 21st March 1901 this animal was inoculated with 50 cc. of the filtered toxin of a fourteen days old bouillon culture. Filtration was effected by a Chamberland filter, and the toxin proved sterile when planted on agar. Soon after the injection the horse sweated profusely, the respiration was accelerated, and dung was repeatedly voided.

About an hour later the animal was injected with a further dose of 90 cc. of the same toxin. Similar symptoms were observed, but the perspiration on this occasion was so severe that the water dropped from the animal. The temperature rose to  $39.5^{\circ}$  C. The horse sat on his haunches, and his eyes were suffused with tears. The pulse disappeared. On the morning of 23rd March 1901 the horse was found in agony, and was therefore killed.

The *post-mortem* was made directly after death. The epicardium was dotted with hæmorrhages, and there were subendocardial hæmorrhages in the right ventricle. The atrio-ventricular valves were oedematous, but otherwise normal. No culture could be obtained from the spleen.

*Experiments with Rabbits.* 19th October 1900. Rabbit F.a. was injected with 5 cc. of a bouillon culture into the peritoneum. 20th October 1900. The rabbit died during the night.

At the *post-mortem* examination there was very little fluid in the peritoneal cavity. There were hæmorrhages on the rectum. Bacteria were present in the peritoneum, but only a few were found in the blood from the heart.

3rd September 1900. 4 cc. of a bouillon culture were injected. The animal died next day. There was a peritonitis fibrinosa, and bacteria were present in the peritoneal cavity and the heart blood.

Two more rabbits were injected under the skin with 1 cc. of the culture, but death did not ensue; nor did the rabbit injected with 1.5 cc. intraperitoneally die.

*Pigeons.* Two pigeons were injected with 2 cc. of a one-day-old culture. These birds refused to eat for the next two days, but recovered.

*Rats.* A white rat was injected subcutaneously, and another one intraperitoneally. Both showed symptoms of illness during the following days, but recovered.

*Sheep.* A young sheep was injected with 2 cc. of a bouillon culture into the jugular. It showed symptoms of illness during the following days, but recovered.

*Goat.* A young goat was injected with 5 cc. of a bouillon culture in the leg. She went very lame and was seedy, but recovered.

#### *Analysis of the above Experiments.*

The bacillus described above could not be identified as one of the already described ones. It is easily distinguished from the hæmorrhagic septicæmia group, otherwise called *pasteurella* by Lignières. In Lehmann and Neumann's classification of bacteria it would fall under the group of *bacterium coli*, although it differs from it by not

producing indol. In Flüggé's classification of micro-organisms it would fall into the hæmorrhagic septicæmia group, to which belong motile bacilli which do not exactly cause septicæmia, but which are found in the tissues herded together in masses, resembling in this particular the typhoid bacillus. The bacillus in question shows a tendency to form colonies in the spleen; smear preparations from the spleen show the bacilli always grouped together. This, and the fact that it is constantly found in the spleen, makes the resemblance to the bacillus typhi abdominalis more marked.

It has been demonstrated that the bacillus is only slightly virulent for the smaller laboratory animals, and the injection of such large quantities of culture produces death rather by toxæmia than by the multiplication of the bacteria.

The bacillus is, however, decidedly pathogenic for horses. Death after injection into the jugular is, in my opinion, due to the presence of toxins, which, when isolated by filtration, produce an almost equal effect. Subcutaneous injection is sometimes followed by symptoms, which, however, never resemble the original disease.

Severe symptoms were produced in a healthy horse after it had received blood from a horse suffering from malaria sequelæ. This experiment is important, as it demonstrates that a more pronounced pathogenic result is obtained when the bacillus is injected into a horse which stands under the influence of the pyroplasma. The fact that the subcutaneous injection of a pure culture produces suppuration and necrosis is another indication of the pathogenicity of the bacillus in the horse. These experiments also point to the conclusion that the bacillus is not the *causa causans* of the disease described, but that the disease is due to the symbiotic effect of both, or to a secondary infection by the bacillus after the animal has been primarily affected with the pyroplasma equi.

In this symbiotic disease one part of the infection is due to a micro-organism belonging to the animal kingdom, and the other to one belonging to the vegetable kingdom. The impossibility of cultivating the former in artificial media makes it impossible to produce the disease experimentally by the inoculation of cultures.

As has been already mentioned, the pyroplasma equi has never, until now, been transmitted to healthy horses by the injection of blood, and the explanation of this failure has been already given.

When, however, the presence of the bacillus in the system of horses which show all more or less the same *post-mortem* symptoms is proved, and when this absence from horses which died from other diseases is shown, we obtain indirect evidence as to the conditions which the bacillus requires for its development. Although at one time it was thought that the presence of a specific bacterium elsewhere than in a typical disease of which it was presumably the *causa causans*, was evidence against the specificity of the bacterium, this view has not stood the test of recent experience. We know now that a micro-organism may be present in healthy animals without producing any ill effect, and that special conditions are required for its development. Such is especially the case in symbiotic diseases similar to the one under review.

One fact I have observed, that the bacillus I have described may occasionally be present in horses which die but do not show the

typical symptoms I have described, but this was rare (only two cases).

The following is a record of the bacteriological examinations of the spleens of horses dead from other diseases than the sequelæ of malaria:—

1900.			
1.	Sept. 15	.	Enteritis; <i>post-mortem</i> shortly after death. Bacterium found not identical with the one in question.
2.	" 21	.	Pneumonia. —
3.	" 24	.	Enteritis; <i>post-mortem</i> shortly after death. Bacterium as in No. 1.
4.	" 26	.	Chronic enteritis; <i>post-mortem</i> ten hours later. Nothing found.
5.	Oct. 1	.	Enteritis; <i>post-mortem</i> fourteen hours after death. Bacterium found, but not identical with the typical one.
6.	" 3	.	Pneumonia, endocarditis, enteritis; <i>post-mortem</i> half an hour later. —
7.	" 9	.	Colitis; <i>post-mortem</i> four hours after death. —
8.	" 9	.	Enteritis; <i>post-mortem</i> five hours after death. —
9.	" 10	.	Colic; <i>post-mortem</i> ten hours after death. —
10.	" 12	.	Enteritis; <i>post-mortem</i> four hours later. —
11. }	" 13	.	Cast horses killed; <i>post-mortem</i> —
12. }	" 13	.	six hours after death. —
13.	" 14	.	Experimental horse - sickness; <i>post-mortem</i> immediately after death. —
14.	" 17	.	— — —
15.	" 18	.	— — —
16.	" 22	.	Experimental enteritis; <i>post-mortem</i> directly after death. —
17.	" 24	.	Isolation. Bacteria found, but not identical with the typical one.
18.	" 30	.	Experimental horse - sickness; <i>post-mortem</i> directly after death. —
19.	" 31	.	— — — None present.
20.	Nov. 1	.	Cast horse killed; <i>post-mortem</i> directly after death. Bacteria present not identical. —
21.	" 7	.	Chronic pneumonia. —
22.	Dec. 12	.	Horse-sickness. None present.
23.	" 17	.	Gastritis, enteritis, nephritis. Bacteria present not identical.
24.	" 31	.	Anthrax. Anthrax bacterium.
1901.			
25.	Jan. 5	.	Experimental horse killed with bacillus typhi abdominalis. Bacillus typhi abdominalis.
26.	" 14	.	Experimental horse killed with bacillus coli; <i>post-mortem</i> three hours after death. Bacillus coli
27.	" 22	.	Horse-sickness. None present.
28.	" 24	.	— — — Bacteria present not identical.
29.	" 24	.	— — — Bacillus coli.
30.	Feb. 6	.	Pneumonia. None present.
31.	" 6	.	Horse-sickness; <i>post-mortem</i> five hours after death. Bacillus coli found.

32.	Feb.	13	.	Horse-sickness ; <i>post-mortem</i> six hours after death.	Bacterium fluorescens foetidum.
33.	"	8	.	Horse killed with typhoid bacillus.	Bacillus typhi abdominalis.
34.	"	14	.	Horse-sickness ; <i>post-mortem</i> twelve hours after death.	Bacteria found not identical.
35.	"	16	.	Horse-sickness ; <i>post-mortem</i> twenty-four hours after death.	—
36.	"	18	.	Horse-sickness.	None found.
37.	"	19	.	—	Bacteria found not identical.
38.	Mar.	27	.	Pneumonia.	Bacillus coli
39.	"	29	.	Nephritis.	Bacteria found not identical.
40.	{	" 20	·	} Pneumonia and influenza.	None found.
41.	{	" 20	·		

The following are the cases in which the typical bacterium was found:—

(42.) 21st September 1900. Experimental horse 25 for horse-sickness. This horse was suffering from a big abscess in the post-pharyngeal gland, which prevented the animal from eating. On *post-mortem* the spleen was found normal. Cultures revealed the presence of the typical bacillus.

(43.) 22nd September 1900. Very poor horse. *Post-mortem* showed general anæmia. Pneumonia cavernosa. Pleuritis fibrosa, spleen normal. Contains the specific bacterium.

A summary of the above observations shows that out of forty-three *post-mortem* examinations upon horses which did not show symptoms of the sequelæ of equine malaria only two cases were found to show the specific bacillus ; this corresponds to 3·2 per cent. The bacillus was found in eighteen cases of typical malarial sequelæ, corresponding to 30·5 per cent., and was not found in 41 similar cases, corresponding to 69·4 per cent. of all *post-mortems* made which were bacteriologically examined. These figures do not include the *post-mortems* of horses which were killed by the injection of the typical bacillus. These statistics prove that the bacillus which I have described is not merely a casual concomitant, but is a specific factor in a certain series of pathological changes commonly found in malaria sequelæ. In cases 40 and 41 the horses were in very low condition ; it may therefore be allowed that they offered a suitable medium for the development of the bacillus.

The fact that the *post-mortems* were made at varying intervals after death—even as late as twenty-four hours—proves that the invasions of the bacterium into the spleen was not a *post-mortem* phenomenon, it having been found *intra vitam*, and at all times after death in the typical cases, and not in others. Some of the bacteria found in the spleen were certainly due to *post-mortem* invasion, or rather to a preagonistic invasion. They were all closely studied, but most of them could not be identified, and as they differed one from another the study was not continued.

*Agglutination.*—In addition to the evidence already brought forward as to the specificity of the coccobacillus described, we have a still further proof in the fact that the serum of a horse suffering from malaria sequelæ does very often agglutinate the bacilli, when mixed with a bouillon culture. This phenomenon served in the first instance for the identification of a bacterium after it was isolated from

the spleen, and in all instances the typical precipitation of the culture took place when mixed with a corresponding serum. The serum was prepared by subcutaneous injection (horse 185) or by intrajugular injection (S.H.).

Before the serum horses were injected, their serum was tested with as many different sub-cultures from different horses, and then it was found that the normal serum did not agglutinate the cultures. One single injection of a culture is sufficient to produce the agglutinating power. Further, the injection of the filtrate of a bouillon culture produces an agglutinating serum.

In highly immunised horses this power is so high that agglutination takes place very rapidly after the mixture with the culture. The agglutination was tested in the proportion of one part of the serum to one hundred parts of bouillon culture; for this purpose, to 5 cc. of bouillon culture one drop of serum was added. But the phenomenon could still be observed when the dilution was made 1:1000; the precipitation took place after some time. In the proportion 1:100, the phenomenon began shortly after the mixture, and was generally complete in one hour; it rarely took two hours. The bacteria collected in big flakes and subsided, leaving the bouillon as clear as sterile bouillon. The clumps massed closely together, and it was only by shaking the test tube vigorously that a mass could be whirled up. In order to ascertain whether apparently healthy normal horses possessed an agglutinating serum, thirty-four horses were tested at three different times; when it was found that only two horses possessed a serum which agglutinated in a strength of 1:100. I also tested the specific serum on other cultures than the one in question, namely, on chicken cholera, on bacillus coli communis, bacillus typhi abdominalis, and on all the different bacteria isolated from the spleens of the different horses which on microscopical examination and culture proved not to be identical with the one in question. In no instance did the serum in a strength of 1:100 show an agglutinative power. Hence the conclusion that the serum obtained by the injection of the bacterium of malaria sequelæ is specific for this one bacterium.

In order to ascertain the time required by a horse to produce an agglutinating serum, a horse (No. 90) was injected with 5 cc. of a bouillon culture into the jugular. This horse was tapped before the injection, and on each day after the inoculation up to the twelfth day, and the different samples of sera were then tested. It was found that from the fifth day the precipitation began, that on the sixth, seventh, and eighth day it continued to increase in intensity, but after the eighth day there was no further increase. The horse appeared to retain the agglutinating power for a considerable time. One horse (S.H.) was repeatedly tested for a period of a year, and its serum was always found active. Serum kept in test tubes at ordinary temperature, however, had lost this power after four months.

The phenomenon of agglutination is a sign of infection by the bacterium which produces it, and can therefore be utilised for diagnostic purposes. It was therefore utilised in the case of several horses which were suspected to be suffering from malaria sequelæ, or which were actually suffering from bilious fever.

(1.) Horse 34 (14 of the S.U. list). This horse was found suffering

from malaria on 2nd March. On 10th March blood was collected in test tubes and allowed to coagulate. The next day the serum was tested on all the previously isolated cultures of the bacillus in question; it was found that all of them were typically agglutinated. The horse died on the night 11th March, when the bacterium was isolated from the spleen.

(2.) Horse 1050 (No. 11). This horse died on the night 3rd January 1901. During the 3rd January blood was collected. On 7th January the serum was tested on a bouillon culture, and it was found that it agglutinated very rapidly and typically. The bacillus was obtained from the spleen.

(3.) Horse. Dr T. English mare. On the 5th April this horse showed symptoms of malaria and the pyroplasma equi was found. The horse was treated, and recovered. But as, from previous experience, symbiotic infection with the bacillus of malaria sequelæ might be anticipated, the horse was tapped daily, and the serum tested for agglutination. The serum of the first day and the following day showed no agglutinative power whatever. The pyroplasma had disappeared. On the 16th April the serum of the horse showed distinct agglutinative power, and the power increased during the following days.

(4.) Horse S.A.C. 585. (Case 17). The serum of this horse was taken two days previous to its death on the 13th March 1902, and was tested on the 15th March. The precipitation was distinct. The bacillus was isolated from the spleen.

(5.) 25th March 1902. A horse of the S.A.C. belonging to Colonel St., showed distinct symptoms of biliary fever. It was treated by Captain Christy, and recovered. Captain Christy was good enough to put some serum at my disposal on the seventh day after the animal had entered the sick lines. This serum did not agglutinate. The animal was tapped again on the 29th March, and tested, when it was found that it agglutinated promptly.

(6.) 15th April 1902. Horse 24 of the S.A.C. suffering from bilious fever, but recovered. The case was diagnosed by Captain Christy and tested. After recovery the serum was tested, and the test repeated with serum taken on the 17th April. No reaction whatever took place.

(7.) 3rd June 1902. Horse belonging to the T.C.S. Company. This animal showed all the symptoms of biliary fever. The pyroplasma equi, however, was not found. Blood was collected, but the serum did not agglutinate. On the 13th June blood was obtained, the animal having recovered, and the serum was found to agglutinate promptly.

This is all the evidence which I have been able to collect, but from this evidence we may conclude that an animal which is suffering from malaria sequelæ produces an agglutinative serum. For production of this serum a time must elapse of at least five days between the date of infection and the appearance of the agglutinins in the blood. From Cases 3 and 7 we may gather that even horses which suffer from malaria fever and which recover may become infected by the bacterium of the sequelæ, and that they do not necessarily succumb. This is further evidence of the symbiotic, or rather, secondary infection in bilious fever.

Case 6 proves that there may be infection with the pyroplasma equi, but no secondary infection.

Cases 3 and 7 illustrate also the observation quoted above, where, out of thirty-six apparently healthy animals, three had a distinct agglutinating serum. These two animals probably had some time in their earlier life suffered from bilious fever and the sequelæ, and recovered, and the sera had retained the agglutinating power.

We may further conclude that symbiotic and secondary infection due to the pyroplasma equi on the one side, and to the bacterium on the other side, are not necessarily fatal.

To what recovery is due, I am unable to say. The serum of a horse which has received several injections of bouillon cultures of the bacillus of malarial sequelæ, and which is highly agglutinative, has no noticeable bactericidal action *in vitro*. Agglutinated cultures when transplanted into fresh agar developed even after a contact of fourteen days. I am not yet in a position to state what effect the serum of a highly immunised animal has on infected animals, when used as a curative agent.

It remains now to trace the origin of the bacterium, and how it comes into the animal's body. The idea that the infection takes place from the intestines will at once suggest itself. Seeing that it is, so to say, constantly found in the biliary fever cases, one is entitled to believe that the bacillus may be fairly constantly present in the horse, but that it enters the animal's blood and becomes pathogenic when the vitality of the antitoxic and bactericidal power of the animal juices is reduced. I was unable to isolate the bacillus by plate cultures on gelatine or agar from the dung, even though I experimented both with healthy horses and with horses whose sera agglutinates cultures of the bacillus.

The most common bacterium was a bacillus coli communis which corresponds to the typical bacillus coli communis of the text books.

#### *Summary of Conclusions.*

- (1). Equine malaria or biliary fever is due in the first instance to an invasion by the pyroplasma equi.
- (2). The pyroplasma equi predisposes to a symbiotic and secondary infection due to a specific bacillus.
- (3). The secondary infection can be traced by the agglutination test *intra vitam*, provided that the infection has existed for five days.
- (4). The secondary infection can take place even after complete recovery from the biliary fever.
- (5). The secondary infection is not invariably fatal.
- (6). When a healthy horse possesses agglutinating serum it is probable that it has suffered at some time from equine malaria.

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### SOUTH AFRICAN HORSE-SICKNESS.

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THE worst part of the horse-sickness season of 1903 being over, I propose to give an account of *post-mortems* made on forty-eight cases of this disease, these cases all occurring in the space of about two



months. Attached will be found a table in which an endeavour has been made to classify the cases according to the most prominent lesions found on *post-mortem* examination. The classification is only a rough one, as one cannot draw a fine distinction.

In the attached table it will be seen that Classes I., II., and V. differ only in the extent of the bowel lesions. In Class I. the only marked bowel lesion is a very intense inflammation of the mucous membrane of the pyloric portion of the stomach. In Class II. the inflammation of the mucous membrane is present in the small and large intestine, also to a varying extent. In these cases the inflammation of the mucous membrane is in the majority of cases most intense at the pelvic flexure of the large colon and also in the cæcum. At these parts the inflamed membrane is frequently of a blood red or purple colour, in many cases as intense as that of the pyloric membrane of the stomach. A quantity of gravel is frequently present in the cæcum and large colon, but this is a common occurrence in cases other than horse-sickness, and has no direct bearing on the disease. Occasionally, but rarely, there are noticeable in the mucous membrane of the intestines small, slightly depressed, ulcer-like lesions which are small areas of surface necrosis.

Before proceeding any further I should like to make a particular point of the fact that not in one single case of the forty-eight *post-mortemed* was an intense inflammation of the mucous membrane of the pyloric portion of the stomach absent. This organ was in every case made the object of very careful inspection, in view of the fact that this particular lesion was said by some observers not to be a common one in South Africa. I was fortunate enough to be present at *post-mortems* made on several cases of experimental horse-sickness at the Royal Veterinary College, London, by Prof. M'Fadyean, and the invariable presence of this lesion was very striking.

A close examination of the pyloric mucous membrane of the stomach in these cases *post-mortemed* by myself was not necessary to detect this lesion, although occasionally the membrane was covered by a thick layer of a white mucus-like material, which, however, is easily removed by gently passing the edge of the knife over the part, disclosing the intensely inflamed membrane beneath. The colour of this inflamed pyloric membrane varies from blood to purple, and always suggests that some very irritant poison has been at work. The inflammation of the mucous membrane of the small intestine usually takes the form of a slight muco-enteritis, but occasionally it is very much more marked, and in the form of ring-like bands (zebra marking). In some cases blood extravasations are present. I have never met with a case of very intense inflammation of the mucous membrane of the floating colon, and when present at all the colour of the membrane is of a rose pink.

In Classes III., IV., and VI., the intestinal lesions in the majority of cases were more marked than in Classes I., II., and V., and the lesions in the thoracic cavity correspondingly less marked.

*Symptoms in Classes I., II., and V.*—In these cases the animals were usually not brought to the sick lines until they had commenced to show symptoms of distress, *viz.*, accelerated or difficult breathing; and death usually took place within a few hours, the disease being so far advanced as to render treatment hopeless. Other symptoms shown are: intense

CLASS I.	CLASS II.	CLASS III.	CLASS IV.	CLASS V.	CLASS VI.
<p>1. Pneumonic symptoms and rapid death after their onset.</p> <p>2. Discharge of frothy serum after death from nostrils. (Sometimes also before death.)</p> <p>3. Lungs extremely cedematous. A quantity of clear amber-coloured gelatinous exudate on their surface.</p> <p>4. A clear amber-coloured or blood-tinged exudate in pleural cavity.</p> <p>5. A clear amber-coloured or blood-tinged exudate in pericardial cavity.</p> <p>6. A very intense inflammation of the mucous membrane of the pyloric half of the stomach.</p>	<p><i>Ditto I.</i></p> <p>Inflammation of the mucous membrane throughout the intestinal tract.</p>	<p>1. Death not usually so rapid after onset of clinical symptoms.</p> <p>2. Edema of the tissues of the neck and filled condition of the supra-orbital cavities usually present.</p> <p>3. Slight cedema of lungs and small quantity of gelatinous exudate or absence of cedema.</p> <p>4. Pleural exudate as in Class I.</p> <p>5. Pericardial exudate as in Class I.</p> <p>6. Very intense inflammation of the mucous membrane of the pyloric half of the stomach and throughout the intestines.</p>	<p><i>Ditto III.</i></p> <p>Inflammation of the mucous membrane of the intestines.</p>	<p><i>Ditto I.</i></p> <p>Intense inflammation of the mucous membrane of the large colon only.</p>	<p><i>Ditto III.</i></p> <p>6. Except inflammation of the mucous membrane of the pyloric half of stomach and large intestine only.</p>
<p>Colour. Sex. Age. Admtd. Died.</p> <p>1. Roan Mare 6 10/2/03 11/2/03</p> <p>2. Bay Geld. 6 14/2/03 14/2/03</p> <p>3. Bay Geld. 6 16/2/03 17/2/03</p> <p>4. Bay Geld. 6 19/2/03 19/2/03</p> <p>5. — Mule 7 22/3/03 22/3/03</p> <p>6. Bay Geld. 7 22/3/03 22/3/03</p> <p>7. Bay Mare 8 25/2/03 25/2/03</p> <p>8. Ch. Mare 7 28/2/03 28/2/03</p> <p>9. Bay Geld. 7 3/2/03 6/8/03</p> <p>10. Ch. Geld. 7 11/2/03 12/3/03</p> <p>11. — Mule 12/2/03 18/3/03</p> <p>12. Bay Geld. 18/2/03 18/3/03</p> <p>13. Roan Geld. 7 19/3/03 19/3/03</p> <p>14. Ch. Geld. 7 7/4/03 7/4/03</p>	<p>Colour. Sex. Age. Admtd. Died.</p> <p>1. Bay Geld. 7 21/2/03 21/2/03</p> <p>2. Br. Geld. 6 22/2/03 22/2/03</p> <p>3. Bay Geld. 7 22/2/03 22/2/03</p> <p>4. — Mule 7 22/2/03 22/2/03</p> <p>5. — Mule 7 25/2/03 25/2/03</p> <p>6. Bay Geld. 7 28/2/03 28/2/03</p> <p>7. Bay Mare 8 28/2/03 28/2/03</p> <p>8. Ch. Mare 7 3/2/03 6/8/03</p> <p>9. Bay Geld. 7 11/2/03 12/3/03</p> <p>10. Ch. Geld. 7 18/2/03 18/3/03</p> <p>11. — Mule 18/2/03 18/3/03</p> <p>12. Bay Geld. 7 19/3/03 19/3/03</p> <p>13. Roan Geld. 7 7/4/03 7/4/03</p>	<p>Colour. Sex. Age. Admtd. Died.</p> <p>1. Bl. Mare Aged 19/2/03 21/2/03</p> <p>2. Bay Mare Aged 24/2/03 24/2/03</p> <p>3. Bl. Geld. 8 23/2/03 24/2/03</p> <p>4. Grey Mare 7 24/2/03 21/2/03</p> <p>5. Br. Mare 5 22/2/03 22/2/03</p> <p>6. Ch. Geld. 8 24/2/03 27/2/03</p> <p>7. Bl. Mare 6 12/3/03 13/3/03</p> <p>8. Grey Geld. Aged 17/3/03 17/3/03</p> <p>9. Bay Geld. 7 17/3/03 17/3/03</p> <p>10. Bay Geld. 6 18/3/03 18/3/03</p>	<p>Colour. Sex. Age. Admtd. Died.</p> <p>1. — Mule 19/2/03 22/2/03</p>	<p>Colour. Sex. Age. Admtd. Died.</p> <p>1. Roan Mare 7 22/2/03 22/2/03</p> <p>2. Ch. Geld. Aged 22/2/03 25/2/03</p> <p>3. Bl. Mare 8 24/2/03 25/2/03</p> <p>4. Bay Geld. 7 27/2/03 27/2/03</p> <p>5. — Mule 6 10/3/03 19/2/03</p> <p>6. Bay Geld. 6 21/2/03 21/2/03</p> <p>7. Bay Geld. Aged 26/3/03 27/3/03</p> <p>8. Ch. Geld. 7 26/3/03 27/3/03</p> <p>9. Ch. Geld. 7 30/3/03 30/3/03</p> <p>10. Grey Geld. Aged 31/3/03 31/3/03</p>	<p>Colour. Sex. Age. Admtd. Died.</p> <p>1. Bay Mare 7 25/2/03 25/2/03</p> <p>2. Bl. Geld. Aged 27/2/03 28/2/03</p> <p>3. Bay Geld. 11/3/03 11/3/03</p> <p>4. Bay Mare 5 25/3/03 25/3/03</p> <p>5. Bay Mare 7 18/3/03 28/3/03</p> <p>6. — Mule 6/4/03 6/4/03</p>

congestion of the conjunctival membranes, and the invariable presence on them of red or purple petechial spots. This condition of the membranes is a characteristic feature. The pulse is always quickened, very weak, or almost imperceptible. The temperature is usually high ( $105^{\circ}$ ), but in cases where death is imminent it may be apparently normal, though in reality running down just before death.

Frequently about half-an-hour before death the animal commences to discharge from both nostrils huge quantities of a yellowish or white frothy serum, but if this does not occur before death it invariably does after death in this class of case. Usually these are the only noticeable symptoms.

On *post-mortem* one finds the lungs to be greatly enlarged from oedema, the serous fluid being present in such quantities as to run away on section. This fluid is turned into froth in the lungs themselves, as well as on coming in contact with the atmospheric air, this being due to the churning process of respiration. A quantity of a clear amber-coloured gelatinous exudate is almost invariably present on the surface of and between the lungs, also intersecting the lobules of the lungs near the surface. A dropsical transudate is almost invariably present in the pleural and pericardial cavities. The quantity varies from a few ounces up to 2 or 3 pints, and is usually of a clear amber colour, free from any signs of putridity; occasionally it is blood tinged. The intestinal lesions vary in extent, as shown above.

*Symptoms in Classes III., IV., and VI.*—Frequently in a careless examination the only symptoms that would be observable would be dulness and a tendency to lie down. This tendency to lie down is always present, and indicates a condition of uneasiness akin to colic. If a closer examination of the patient be made the conjunctival membranes will be found to be very dark and intensely injected, showing petechial spots. The pulse will be found to be quickened and very weak. This weakness of the pulse I put down in the majority of instances to the mechanical interference of the dropsical transudate with the heart's action, this being frequently present in such quantity as to be retained under pressure within the pericardial membrane. The temperature is always raised unless death be imminent. Death usually does not take place so rapidly as in the pneumonic forms. The patient may live for several days, or death may take place within a few hours of clinical symptoms being noticed by the ordinary layman. Probably, however, if observation had been carried out for several days previously to the animal being brought to the sick lines by an experienced eye, the animal would have been noticed not to be in his usual health.

The symptoms shown in this class of case are always indicative of intestinal trouble, the animal sometimes grunting and turning his head towards his abdomen. In fact, the patient shows the symptoms of enteritis.

It is not always easy to diagnose cases of this class in their early stages, but the condition of the membranes is a good guide. In this country cases of fever with discoloured membrane are common, being probably malarial fevers, but it is possible to draw the conclusion that more animals than is at present supposed become affected with horse-sickness, but recover in the early stages of the disease.

Other symptoms more frequently shown in this class of case than in the pneumonic form are œdema of the tissues of the neck and filling of the supraorbital cavities, so that they become convex instead of concave. When this œdema of the tissues of the neck is present in a minor degree, or so as not to be observable without manipulation, it can be frequently noticed by a peculiar, slightly indurated, cord-like feeling of the sternomaxillaris muscle; and later the tissues of the neck, instead of having the loose feeling when the front of the normal neck is grasped and moved from side to side, seem closer knitted together and firmer, and the trachea particularly prominent.

At the *post-mortem*, as previously mentioned, the lesions in the abdomen are usually more marked than the corresponding lesions in the acute pneumonic form. There is usually present slight œdema of the lungs and tissues on the chest wall. The lesions of the pleural and pericardial cavities are the same as in cases of the pneumonic forms, and I have never yet met with a case where there was no fluid in either of these cavities.

These two forms of horse sickness, viz., that in which the marked symptoms are pneumonic, and that in which the marked symptoms are abdominal, appear to me to be distinct enough to warrant one speaking of a pneumonic and an intestinal form. I have had no experience with the dikkop form, which I presume is quite distinct from the form in which one gets œdema of the tissues of the neck, but no swelling of the head except the filling of the supraorbital cavities. I should have mentioned that in the intestinal form, or, better, gastro-enteric form, the animal usually lives long enough to lose condition, whereas in the other form animals at the time of death are usually in excellent condition.

The liver in neither form appears to show characteristic lesions, nor do the kidneys. In only one case did I find the spleen distinctly enlarged, in which case it was quite twice its normal size. In other cases it may have been slightly enlarged and heavier, but, not having had scales to hand, it is useless to assert that the spleen is usually enlarged.

*Treatment.*—I must admit that I have not been very successful in the treatment of the disease. In the majority of cases when the patient is first seen the disease is too far advanced to allow any hope of successful treatment. The gastro-enteric form, on account of its slower progress, is more amenable to treatment. I have used antiseptics given as drenches, and also injected subcutaneously and intratracheally. Carbolic acid as an emulsion with linseed oil may be given in ʒii doses twice or thrice daily, but drugs given by the mouth do not give satisfactory results. Carbolic acid may also be injected into the trachea with glycerine as the vehicle. This causes a discharge from the nostrils from irritation of the tracheal mucous membrane, but causes no distressing symptoms from its irritation, and the discharge soon ceases after the injections are discontinued. Intratracheal injections of hydrarg. biniod. and pot. iod. have afforded me the most satisfactory results. The injection of this mixture also causes discharge from the nostrils, but the cause of the discharge is purely local. The percentage of recoveries by any method of treatment is small, provided that there is no doubt as to the case being one of horse sickness. Out of forty-eight cases, four recovered under

treatment, and one was well on the road towards recovery, but had a relapse and died. The following is a brief account of these cases:—

I. Bay gelding, six years old, admitted 12th March 1903, showing the following symptoms: conjunctival membranes extremely injected, showing purple petechial spots; great dulness and difficult breathing; supraorbital cavities filled; swelling of neck, due to oedema of the tissues. Temperature, A.M.,  $104^{\circ}$ ; P.M.,  $104.3^{\circ}$ ; pulse, 65; respiration, 24.

*Treatment.*—One injection intratracheally morning and evening of acid. carbol. pur.  $\frac{3}{4}$ ss, glycerine  $\frac{3}{4}$ ss. This caused a clear discharge from the nostrils, but no coughing or injection.

13th March 1903.—Membranes not so deep a colour; petechiæ still present. Temperature, A.M.,  $103.6^{\circ}$ ; P.M.,  $103^{\circ}$ ; pulse, 60; respiration, 20. Having run out of carbolic acid, two injections of the following administered intratracheally: hydrarg. biniod. grs. iii, pot. iod. grs. xv, aquæ  $\frac{3}{4}$ ss. Discharge from nostrils continues.

14th March 1903.—Temperature, A.M.,  $102.2^{\circ}$ ; P.M.,  $102^{\circ}$ ; pulse, 52; respiration, 20. Condition about the same. Treatment continued, but dose of hydrarg. biniod. reduced to gr. i at each injection.

15th March 1903.—Temperature, A.M.,  $101.6^{\circ}$ ; P.M.,  $100.2^{\circ}$ ; pulse, 40; respiration, 18. Decided all round improvement; petechial spots disappeared. Oedema of neck dispersing. Intratracheal injections continued.

16th March 1903.—Temperature, A.M.,  $100.6^{\circ}$ ; P.M.,  $100^{\circ}$ ; pulse, 40; respiration, 18. Animal brighter; feeding moderately. Injections discontinued. From this date improvement continued. Belladonna administered to stop the nasal discharge. The patient was discharged cured on the 1st April 1903.

II. Bay gelding, aged, admitted 18th March, 1903.—This case was practically the same in its course as case I., and also treatment, except that hydrarg. biniod. and pot. iod. were used from the commencement. The patient was discharged cured on the 6th April 1903.

III. Bay mare, seven years old, admitted 18th March 1903.—Symptoms and treatment precisely similar to the previous cases. Continued to progress satisfactorily up to the 26th March 1903, when she was suddenly taken worse, and died on the 28th March 1903.

IV. Chestnut gelding, seven years old, admitted 17th March 1903.—Discharged cured on 13th April 1903. Symptoms, treatment, and course of disease similar to previous cases. This animal had a large hard swelling at the seat of injection in the neck, due to some of the liquid injected having got into the space between the skin and trachea. This swelling, however, gradually disappeared without any unfavourable symptoms.

V. Mule, admitted 20th March 1903, showing following symptoms: Temperature, A.M.,  $107^{\circ}$ ; P.M.,  $105.6^{\circ}$ ; pulse almost imperceptible; heaving respiration accompanied by grunting. Oedema of tissues of neck. Supra-orbital cavities filled. Two intratracheal injections administered, each containing hydrarg. biniod. grs. iii, pot. iod. grs. xv, aquæ  $\frac{3}{4}$ ss. The injection caused the mule to cough; at the same time brought up about one pint of white frothy material.

21st March 1903.—Temperature, A.M.,  $104.6^{\circ}$ ; P.M.,  $105^{\circ}$ ; still

grunting, and breathing heavily. Treatment continued. Case given up as hopeless. Besides the intratracheal injections, the same dose was injected at the same time as the second intratracheal injection on either side of the neck subcutaneously, so that on this day the patient received altogether hydrarg. biniod. grs. xii.

22nd March 1903.—Temperature, A.M.,  $104^{\circ}4'$ ; P.M.,  $103^{\circ}6'$ ; heaving respiration; pulse very weak. Two injections intratracheally.

23rd March 1903.—Temperature, A.M.,  $103^{\circ}$ ; P.M.,  $102^{\circ}$ . Two injections.

24th March 1903.—Temperature, A.M.,  $101^{\circ}6'$ ; P.M.,  $100^{\circ}4'$ ; respiration, 32; pulse very weak. Lost a lot of condition.

25th March 1903.—Temperature, A.M.,  $100^{\circ}2'$ ; P.M.,  $99^{\circ}$ . From this date the mule continued to improve, and is now practically fit and rapidly putting on condition. The only reason that the patient is still in the sick lines is that the injection on one side of the neck caused a large area of skin to slough, and that is not yet quite healed.

This was a remarkable case, and I feel convinced that recovery was due to the almost poisonous doses of hydrarg. biniod. administered. Another peculiar feature is the fact that whereas the hydrarg. biniod. injected into the trachea caused no serious damage except a discharge from the nostrils, when injected under the skin it caused the latter to slough. It only proves how very tough and resisting the tracheal mucous membrane must be.

*Cause and Prevention.*—Several theories have been put forward as to the cause of this disease, but up to the present nothing is definitely known as to the actual causal organism, if organism there is; and there seems to be little doubt that the disease is caused by a micro-organism, but all the efforts with the present powers of the microscope have been unavailing to discover it. Animals may be readily infected by inoculation with blood of a horse affected with the disease, the period of incubation in these experimental cases being almost invariably eight or nine days. Probably the period between infection and death is the same in natural cases, since the *post-mortem* lesions in experimental and natural cases are similar. It is said that if animals are prevented from grazing after sundown, and in the morning before the sun is well up and has dried the veldt, the disease will be prevented, and also if a nosebag be worn at night for a similar purpose. But in my opinion this theory is absolutely untenable, and is not borne out by facts. The animals under my charge after the first few cases had occurred were prevented from grazing at all, and were not even allowed to water until within the suggested preventative hours, yet the disease was in no way checked. I have also heard it expressed by other observers who have experienced several seasons that, even when these commonly suggested precautions were taken, cases occurred just as frequently. There is no doubt that some low-lying parts are very infective, especially where water and herbage is abundant.

The method of infection also is not known, but the mosquito inoculation theory seems to fit in best with observed facts in connection with the disease. Animals kept in stables and not taken out at night contract the disease far less commonly. The idea amongst farmers seems to be that the disease is contracted one night,

and the animal dies the following day, and little attention seems to be paid to where the animal happened to be, and what he was doing a reasonable time beforehand. The inoculation theory fits in with the observed fact that the disease is more commonly contracted in marshy districts and also at night; and the fact of the horse sickness season being practically over with the first severe frost would be difficult to explain, except for the possibility of the causal organism resting in some secondary host during the winter months in a state of inactivity.

### DERMOID CYSTS.<sup>1</sup>

By J. R. U. DEWAR, F.R.C.V.S., Royal (Dick) Veterinary College, Edinburgh.

THE subject of dermoid cysts, whether dentigerous or not, has been very little studied by English veterinary surgeons. In fact, the literature of the subject is of the most scanty description. In looking up the subject, I have searched the *Veterinarian* back to the thirties, the *Veterinary Journal* since its commencement, and some volumes of the *Veterinary Review* without finding much of importance. Within the past few years rather more attention has been directed to them, but it seems certain that numbers of cases that must be observed are allowed to slip away into the past without being recorded, consequently without inciting to further study.

*Etiology.*—The etiology or causation of these cysts or congenital abnormalities has been very much disputed, and it is doubtful whether their “raison d’être” has yet been satisfactorily explained.

Dr Payne<sup>2</sup> rejects the hypothesis of the older writers, that they are the remains of blighted embryos included during development in the more perfectly normal individual.

Without being at all dogmatic, he states that “it would rather appear as if a portion of embryonic tissue from the upper and middle germinal layers became displaced at an early period of development.”

Somewhat similar views were very generally held. Thus we find Professor Mettam, in describing two dentigerous cysts,<sup>3</sup> stating: “It is generally considered that this form of cyst is due to an error in development; that in the closure of the branchial clefts—in this case of the first—a small portion has been separated off, and along with it part of the tooth-bearing segment of the palato-pterygoid division of the arch bounding the cleft has been included.” He, however, admits that this theory does not account for the presence of cysts containing teeth in the ovary, testicles, and distant parts of the body. These, he states, “are generally considered as forms of teratomata, or embryonic tumours.” But we fail to perceive why a cyst containing teeth and other tissues should be held to have an entirely different origin if situated in the temporal region, from what it would have if in an ovary.

<sup>1</sup> A Paper read before the South Durham and North Yorkshire Veterinary Medical Association.

<sup>2</sup> “Quains Dictionary of Medicine,” 1883.

<sup>3</sup> “*Veterinarian*” for May 1899, p. 309.