

CANINE PIROPLASMOSIS.

I.

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1. Introduction.

THE disease which is the subject of this paper is one of a group of diseases affecting different animals, and caused by parasites possessing very similar characters. The diseases produced by parasites of the genus *Piroplasma* appear to closely resemble each other, and for this reason it is well to follow the terminology adopted by French investigators in speaking of any one of the diseases in question as a

Piroplasmosis. Not only are the parasites similar in these diseases, but they also appear, in all cases where the subject has been worked out, to be transmitted by species of Ixodoidea or Ticks.

All of the diseases in question possess considerable economic importance, for they are amongst the most devastating which affect domesticated animals in many parts of the world, and there is reason to believe that man may also be attacked by parasites of a similar nature. It is possible that the parasites affecting a given animal, although apparently similar, may belong to different species according to the species of tick which act as carriers in conveying the disease. Further investigations are however required to determine this.

Bovine Piroplasmosis is perhaps the most important of this group of diseases. It will suffice here to mention that it has been observed in America over a large area in the United States, in the West Indies (Porto Rico, St Thomas), in the Argentine Republic, Southern Venezuela, Uruguay, and apparently in Brazil. The disease has also caused vast monetary losses in Australia, and in Africa, where it has been observed in the North, South, East and West (Algiers, Egypt, Uganda, Cameroon, Cape Colony, German E. Africa, etc.). In Europe the disease occurs in S.W. Russia, Bulgaria, Hungary, Roumania, Turkey, Italy, Sardinia, France, Germany, Finland, Norway, and there is reason to believe in parts of Great Britain. In all cases the disease has been found associated with the presence of ticks on the affected cattle. The species *Rhipicephalus annulatus* (Say) was proved to transmit the disease by Smith and Kilborne and numerous observers since. This species occurs in America (localities mentioned above), also in many parts of Africa and Asia, and in Australia. *Ixodes reduvius* would appear to play a similar part in Europe, where it is widely distributed. This species is also suspected of transmitting the canine parasite (see p. 223). Judging from the results of inoculation experiments on animals other than bovine, the parasites encountered in cattle are species peculiar to cattle. Theiler and Koch however distinguish the parasites causing Redwater from those causing Rhodesian fever, not only on morphological grounds, but also because animals which are immune for the one disease are not immune for the other. It would appear as if Rhodesian fever of cattle may be transmitted by *Rhipicephalus shipleyi*, a species described in 1903 by Neumann; Mr Lounsbury has, however, just informed me in a letter (24. III. 1904) from Cape Town that *Rh. appendiculatus* carries this disease, and that "a single pathogenic specimen has sufficed for an infection." Lignières (1900) believed

that there were two species of *Piroplasma* causing similar diseases in cattle in the Argentine, and that bovine piroplasmosis in France (mal de brou) was due to still another parasite. It is impossible for me to enter further on the subject here, but what I have said will suffice to show for the present that when we speak of *Piroplasma bovis* we may be speaking of different species of parasites, and that it is well in all cases to state with what species of tick the parasite is associated and where the disease it causes is prevalent geographically, and even then it is not impossible that one species of tick may be able to carry two different species of parasites, given a suitable opportunity. We shall see below that *Ixodes reduvius* is suspected of transmitting the canine parasite in Europe.

Bovine piroplasmosis is known by many names: Texas Fever, Tick Fever, Blackwater, Redwater; Mal de brou, Malaria des bovidés, in France; Piscia sanguie, Malaria bovina, in Italy; Weiderot, Rotnetze, Schwarzwasser, Maiseuche, Blutharnen, Waldkrankheit, etc., in Germany; Tristeza, Lomadera or Ringadera, in South America. All of which names suggest the prominent symptoms, which indeed very much resemble those of the canine malady especially considered in this paper.

Ovine Piroplasmosis was first observed in Roumania (Babès), subsequently in Italy, about Padua (Bonome), in Turkey, about Constantinople (Laveran and Nicolle), and in France (Leblanc and Savigné). Ziemann reported the existence of a similar affection in St Thomas, W. Indies, and Hutcheon and Robertson (1902) in S. Africa. Motas (1902—03) successfully infected four lambs through the agency of ticks (*Rhipicephalus bursa*) obtained from parent ticks fed on infected herds of sheep. He found these ticks in the larval stage to be incapable of communicating the disease. A perusal of Bonome's paper (1895) will show how very similar this disease, due to *Piroplasma ovis*, is to the canine malady. Like the canine and bovine malady, it is transmissible by inoculation with blood containing the parasites, and the disease occurs both in a mild and in a very fatal acute form (*see* Postscript).

Equine Piroplasmosis was observed by Bordet and Danysz; Theiler, Laveran, and others in South Africa; and in Germany by Ziemann, who moreover states that a similar disease prevails in Venezuela.

Human Piroplasmosis may prove to be the correct name for the disease called Rocky Mountain Fever, Spotted, or Tick Fever observed by Wilson and Chowning (1902), whose observations have received confirmation from Westbrook, Anderson, and others (1903). The disease

is probably transmitted by a tick (*Dermacentor reticulatus*¹), a species prevalent in the regions where the disease occurs, namely, in Montana, Idaho, Nevada, Wyoming, and Oregon in the United States. In all cases the attack was preceded by a history of tick-bites having been inflicted about a week previously. The disease only occurs in the spring and early summer, when ticks are active. The symptoms differ markedly from those observed in the bovine, ovine, equine, and canine maladies. Nevertheless the authors named describe and figure piroplasma-like parasites, and consider them to belong to the genus *Piroplasma* (personal communication; see Postscript).

There are other parasites which have been found in man by Leishman and since described by Donovan, Ross, Manson and Low, which Laveran named *Piroplasma donovani*. After seeing preparations of the parasite I quite agree with Ross and Manson in not considering them to belong to this genus. Ross has very appropriately named them *Leishmania* after their discoverer².

It seems in place here to refer to the pathogenic effects following the bites of different species of ticks in man and animals. Those interested can consult Nuttall (1899, pp. 43—49)³, some additional data are given by Sambon (1900)⁴. Particularly interesting are the recent investigations of Marchoux and Salimbeni (1903)⁵, which have demonstrated the part played by a species of tick (*Argas miniatus*) in the spread of a very fatal disease of the domestic fowl in Brazil. This disease is due to a protozoan parasite, a *Spirochaete*, similar to others found in man, in the goose (Sakharoff), and more recently by Laveran (1903)⁶ in cattle. The *Spirochaete* which causes the fowl disease evidently undergoes some development as yet unknown within the above-mentioned species of tick possibly after the manner of other

¹ Suspected in France of conveying *Piroplasma canis*, see p. 240.

² Leishman, W. B. (30. iv. 1903); *Brit. Med. Journ.* i. pp. 1252—1254; (21. xi. 1903) *Ibid.*; (6. ii. 1904) *Ibid.* i. p. 303. Donovan (28. xi. 1903), *Ibid.* ii. p. 1401. Ross, *Ibid.* p. 1401; (1904) *Ibid.* i. p. 160. Manson and Low (1904), *Ibid.* i. p. 183. Laveran (3. xi. 1903), *Bull. de l'Acad. de Méd.*; (7. xii. 1903) *Comptes Rendus de l'Acad. d. Sc.*

³ Nuttall, G. H. F. (1899), "On the rôle of insects, arachnids and myriapods, as carriers in the spread of bacterial and parasitic diseases of man and animals." A critical and historical study. *Johns Hopkins Hospital Reports*, viii. 154 pages, 3 plates. Published also in part in *Hygienische Rundschau*, 1899, Bd. ix. See especially pp. 402—408.

⁴ Sambon, L. W. (1900), "Ticks and Tick Fevers," *Journ. of Tropical Med.* ii. pp. 217—223.

⁵ Marchoux, E., and Salimbeni, A. (1903), "La spirillose des poules," *Annales de l'Institut Pasteur*, t. xvii. pp. 569—580.

⁶ Laveran (1903), "Sur la spirillose des bovidés," *Comptes Rendus*, p. 939.

protozoal parasites, such as those of malaria within the bodies of certain mosquitoes. It will be interesting to see if the *Spirochaete* recently found in cattle is also conveyed by a tick. In any case the work of Marchoux and Salimbeni is of fundamental importance, for it indicates in what direction research must be pushed to discover the mode of spread of relapsing fever in man, the parasite of which (*S. obermeieri*) has so long been known. Finally there are the investigations on "Heartwater" of goats and sheep in South Africa, a disease in which a tick (*Amblyomma hebraeum* Koch) is also regarded as the carrier of infection. I shall report upon this subject later.

Name of the Disease: Canine Piroplasmosis.

Previous to 1899 Hutcheon described the disease in South Africa as "malignant jaundice or bilious fever of the dog," stating that it was the most fatal disease affecting dogs in Cape Colony. It has also been called malignant malaria, hondziekte, malarial fever, and was formerly mistaken for distemper. In France it has been called infectious Icterus. All of these names more or less indicate prominent symptoms, but strictly speaking are unscientific. The jaundice is frequently absent, the disease may run a mild course, there may be but slight fever. I therefore prefer to name the disease after the parasite or parasites which cause it, namely Piroplasmosis, in accordance with the terminology adopted by French authors.

2. History, Geographical Distribution and Seasonal Incidence.

In considering canine piroplasmosis it will be well for the present to distinguish between the observations made in Europe and in Africa for the reason that the parasites causing piroplasmosis may belong to different species in different localities.

I. Canine Piroplasmosis in Europe.

Italy: The first to describe and figure the parasite were Piana and Galli-Valerio (1895). These authors, working in Milan, examined the blood of a dog suffering from fever, weakness, and slight jaundice after having hunted in marshy localities. They found the dog infested with ticks (*Ixodes redurivius* [L.]) and supposed that the latter might transmit the parasite whose resemblance to that of Texas fever was perfectly

evident to them. The parasites occupied 3—4% of the red blood corpuscles, and measured 3·5—2·5 μ , some parasites were encountered free in the plasma. The infected corpuscles contained 2—3—4—5 pyriform bodies. Some of the parasites showed amoeboid motion, this being excellently figured subsequently by Piana (1896) in a coloured plate, showing 24 sketches made of a parasite changing its form within 30 minutes, and protruding usually one or two pseudopodia from an ovoid or spherical body. Stained with thymol methylene blue, the parasites took a deep blue tint, the corpuscles being greenish. An unstained spot of round or oval form could be observed in the blue-stained protoplasm. The dog in question recovered—its blood showed leucocytosis, the erythrocytes were sometimes much enlarged, and nucleated corpuscles were encountered. In dogs previously examined, which had suffered from similar symptoms after hunting in certain localities, they noted at autopsy that the blood was less coagulable, that there was more or less icterus present, that the liver and spleen were congested. At times necrotic foci were observed upon the omentum near the pancreas. They noted the presence of haemoglobinuria, anorexia, prostration and emaciation, the fever recorded in one case reaching 40° C. It is perfectly obvious therefore that these authors were the first to observe the disease in Europe. They named the parasite *Pyrosoma bigeminum*, var. *canis*.

Celli (1900, p. 38) subsequently stated that similar parasites had been encountered in hunting dogs coming to the Campagna Romana from Lombardy.

In *France* the disease was first recorded by Leblanc (20. I. 1900) of Lyons. This author refers to “infectious icterus,” being chiefly observed in hunting dogs. On examining a dog suffering from severe infection he found numerous haematozoa similar to those of cattle and sheep (*Piroplasma bovis* and *ovis*) in its blood corpuscles and free in the plasma. His communication on the subject was very brief, but clear and to the point, scarcely deserving the remark of Nocard and Motas that “la description qu’il fait du parasite est si brève et si peu précise qu’on ne saurait l’affirmer.” Subsequently, after becoming aware of the investigations of Marchoux (mentioned below), Leblanc (17. II. 1900) reported having found similar parasites in four more dogs, the parasites according entirely with the description given by Marchoux.

Important contributions to our knowledge of the disease in France were those of Nocard and Almy (28. III. 1901) and Nocard and Motas (IV. 1902), in which a number of cases observed at the Alfort clinic

are described and much new matter presented which receives full consideration in the following pages. Five out of the seven cases occurring naturally in dogs had been already reported upon by Almy (10. x. 1901). Subsequently Nocard (1902) reported fifteen more cases of the disease in France, so we may conclude that the disease is fairly prevalent there. As Nocard (x. 1901) pointed out, the disease is quite different from the fatal icterus of young dogs, the cause of which is unknown.

II. *Canine Piroplasmosis in Africa.*

Canine piroplasmosis in *South Africa* was recognised as a distinct disease by Hutcheon as early as 1893 (p. 476). At that time he noted the destruction of blood corpuscles and the haemoglobinuria and dwelt upon the close resemblance between the canine disease and redwater of cattle. Cartwright-Reed (1893) reported upon a case of the disease at Herschel, Cape Colony, the diagnosis being arrived at by Hutcheon from the description given by the former. Hutcheon (30. xi. 1893) stated that the disease existed as a fatal epidemic affection in that region in the autumn and winter of 1893. Whilst Cartwright-Reed's note agrees fully with the classic descriptions of the disease, he noted that "the immediate cause of death was effusion of bloody serum into both cavities of the chest." As Hutcheon pointed out, the latter is not a constant phenomenon.

Both Hutcheon (1899, p. 399) and Robertson (1901, p. 331) state that Dr Carrington Purvis of the Bacteriological Institute, Grahamstown, was the first to discover *Piroplasma* in the blood of dogs affected with the disease. Robertson reported at the time that he had found the parasites in all cases of the affection. Shortly before this Koch (1898) appears to have seen similar parasites in *German East Africa*. In his report from Daressalam, he casually remarks that he had detected malaria-like parasites in one dog. He wrote (p. 107) that he had found a number of blood parasites "welche dem menschlichen Malariaparasiten mehr oder weniger ähnlich sind, so bei vielen Arten von Vögeln, bei Reptilien, auch bei einem Hunde und namentlich bei Affen..." It is obviously this passage which is referred to by Marchoux (1900) and Nocard and Motas (1902) as evidence that Koch observed canine piroplasmosis "several times" in dogs in East Africa, but the original statement, above quoted, is altogether too vague to permit of any such conclusion. I however wrote recently to Professors Kolle and Kossel, at present in Berlin, on the subject, and they inform me that the

parasite Koch saw was *Piroplasma canis*. Possibly Martini (1903)¹ also refers to the above passage (he gives no reference) when he states that Koch observed this parasite in the Transvaal in 1897. Martini moreover states (again no reference) that Bitter of Cairo subsequently observed the parasite in dogs in *Egypt*, and that he figured the parasite. I have unfortunately been unable to trace this reference.

In *Senegal* the disease was observed by Marchoux (27. I. 1900), who found the parasites in 11 dogs which showed slight fever but no icterus. The parasites were most numerous during the febrile stage. The disease appears to have been benign. One of Almy's cases (No. 1, see p. 241) appears to have originated in *Tunis*.

The communicability of the disease by blood inoculation was first demonstrated, according to Hutcheon (1899), by Dr Carrington Purvis in South Africa, the observation being confirmed by Spreull in the same year (1900, p. 45). The communicability of the disease in France was demonstrated in a similar manner by Nocard and Motas (1902).

The transmission of the disease by means of infected ticks (*Haemophysalis leachi* [Audouin]) was first demonstrated by Lounsbury in South Africa. A similar demonstration is still wanting for the disease in Europe.

To justify the statement that the *Piroplasma* of Africa may be different from that of Europe I would refer to the geographical distribution of *Haemophysalis leachi*. This species appears to be almost confined to the African Continent. Neumann (1897, pp. 347—350) states that it was found in Egypt by Savigny, Mozambique by Karsch, Eastern Ethiopia by Antinori, West Africa at Pangolin by Mocqueryson, in Algeria by Fayet, in Sierra Leone by Trouessart, and Doumargue, in the French Congo, Transvaal, Cape Colony. Two males were however collected on *Felis tigris* in Sumatra. Almy (1901, p. 375) suspects *Dermacentor reticulatus* (Fabr.) of being the usual carrier of the parasite in France, for the reason that he found this species on dogs which developed the disease there. Salmon and Stiles (1900, p. 452) state that this tick is widely distributed, occurring in different European countries. Railliet (p. 713) records its presence in France and Italy, where it has been found on the ox, sheep, goat, and on man. Salmon and Stiles add that it has been found on horses, deer, roe-deer, fallow-deer, and the rhinoceros. Its presence on the dog does not seem to have been recorded before. This tick has been found in different parts of

¹ Martini (1903), *Archiv f. Schiffs- u. Tropenhygiene*, Bd. VII. p. 504.

the United States (California, Texas, New Mexico, Indian Territory, Tennessee, and Oklahoma). *Ixodes redwivius* [L.], on the other hand, appears to be widely distributed in Europe, and is a species which is suspected of being the carrier of the parasite in Italy (Piana and Galli-Valerio, 1895). This species is very common in France and Germany; it was found in Finland by Kossel and Weber, and has been found frequently in England. It is suspected of also being the carrier of *Piroplasma bovis* in these countries, where it attacks cattle, sheep, and a variety of animals. This species occurs moreover in North Africa, and in North America. As I have already indicated, these two species of ticks possibly transmit two species of canine parasites, but at present there is no proof one way or the other.

Where the disease occurs it appears to be confined to certain localities. Hutcheon (1893, p. 477) in South Africa, stated that it was very common in coast towns and districts, but comparatively rare in higher inland districts of Cape Colony. Nevertheless it prevailed about Herschel in 1893. In 1899 he reported (p. 398) that the disease was confined chiefly to the Eastern coast districts about 1884, but that in 1899 it was met with all over Cape Colony and the adjoining States, chiefly along the main lines of traffic. He first observed the disease at Port Elizabeth in 1885. Lounsbury (1901, p. 11) states that dogs at Cape Town are practically exempt from the disease, but that when such dogs are infected, this can usually be traced to their having previously been taken out upon the veld. Robertson (1902, p. 682) refers to Constantia as being "a notorious place for this affection of the dog."

Seasonal Incidence. According to Robertson (1902, p. 327) the disease prevails chiefly in summer and autumn at the Cape. In Italy cases were observed in April by Piana and Galli-Valerio (1895), in France in September and October by Almy. Further data are required regarding the seasonal prevalence of the disease both in Europe and Africa.

3. The Cause of the Disease.

The Parasite and the Tick which transmits it.

Our studies upon the parasite (*Piroplasma canis*) and the species of tick (*Haemophysalis leachi* [Audouin]) which transmits the parasite to dogs in South Africa are not yet completed, and I shall therefore defer

their detailed consideration, describing them in a future communication. For the present it will suffice to state that the parasite is a Protozoon, and that it occurs in the blood, throughout the body, being most numerous in the internal organs. Most of the parasites are encountered in the red blood corpuscles, but some, presumably derived from ruptured corpuscles in which they have multiplied, are encountered free in the plasma. The general appearance of the parasites is shown in the Plate accompanying the paper by Bowhill and Le Doux on p. 217.

The species of tick above mentioned certainly transmits the disease when infected with the parasite. To be infective the tick must be descended from a mother tick which has sucked the blood of an animal affected with the disease. After the parent tick has gorged herself with blood, she falls to the ground, and after a variable time lays a large number of eggs from which hexapod larval ticks issue in due course. The larval ticks attack a dog when they have an opportunity, fill themselves with blood, and, after about two days, drop off. According to the observations of Lounsbury, these infected larval ticks do not transmit the disease. After lying in the ground for a variable length of time the larval skin is shed and the eight-legged nymphal tick issues forth. Lounsbury has also been unable to show that the infected nymphal tick is infective. The nymph in turn attaches itself to a dog for a few days, and having gorged itself with blood drops to the ground. Here it undergoes its metamorphosis, as did the larva, and after this is accomplished, it casts its nymphal skin, and issues as an adult sexually mature eight-legged tick. It is the *adult* tick and *only* the adult which transmits the parasites, according to Lounsbury.

At Mr Lounsbury's request I have undertaken to control his observations with regard to the infective character of the ticks at various stages of their development. For the present, I can only say that I have successfully infected three dogs with infected adult ticks which Mr Lounsbury has kindly sent me. These experiments should prove convincing to any persons who may doubt the part played by ticks in similar diseases. The disease in question is, as far as I am able to ascertain, unknown in England, and it has been reproduced in England by means of infected ticks sent from South Africa. The control of the rest of Mr Lounsbury's most interesting observations will necessarily take some time, since weeks must elapse for the ticks to undergo metamorphosis from one stage to another. The fact that the tick harbours the parasite after having undergone two moultings subsequent to leaving the egg, and that the adult tick is capable of infecting after

having starved for five to six months, as I have found, certainly shows that the parasite must undergo some cycle of development, as yet undetermined, within the tick's body. It should dispose of the hypothesis advanced by Lignières (1900) in relation to the bovine parasite of a similar kind which causes "Tristeza" in the Argentine. Lignières considered it possible that the parasite might adhere to the external mouth-parts of the mother tick after sucking infected blood, and be deposited from the mouth-parts on the egg-surface during the act of oviposition, when, as others have observed, the mouth-parts play about the vulva. In proof of this possibility he cites experiments in which the injection into animals of crushed infected ticks and their eggs gave a negative result. Five years before Lignières, Hunt (1895) in Australia also reported the negative result of infection experiments by means of inoculations with the crushed bodies of young infected ticks which carried Texas fever. But surely such a crude experiment cannot be accepted as offering any evidence of the absence of the parasite from the internal organs of the tick! Even with the malarial parasites, mammalian and avian, whose cycle of development is known to take place in different species of mosquitoes, we do not know if a similar experiment would or would not yield a negative result. It is an experiment which I have suggested to several observers, but nobody has yet undertaken to carry it out.

The observations of Lounsbury to the effect that infected ticks may harbour, and not convey, the parasite until they reach the adult stage appears to have gained support from observations on ticks which convey similar parasites to sheep and cattle. Whereas *Rhipicephalus annulatus* as far as we know remains on cattle whilst undergoing its development from larva to adult, Kossel and Weber have found that *Ixodes reduvius* does not do so, the larva when gorged leaving its host. Furthermore, Motas (27. XII. 02, and 27. IV. 03), working with the similar disease of sheep (due to *P. ovis*), successfully conveyed it to four lambs by means of infected ticks (*Rhipicephalus bursa*). Here other conditions prevailed, the larva did not infect, and it stayed on the same spot on the animal's skin until it metamorphosed into a nymph. The gorged nymph dropped off, and became infective only on reaching the adult stage. It is evident, therefore, that ticks may behave differently in this respect. It is a matter of considerable practical importance with regard to the efficacy of practical measures of tick extermination, apart from the interest to science, to determine how different ticks behave in this regard. At present we know that some

ticks stay on throughout their different stages of development, others only during one moulting, whilst the species *H. leachi* drops off for each moulting.

These main points in the life-history of *H. leachi* are illustrated by the accompanying photomicrographs, for which I have to thank Mr Walter Mitchell, who took them from some specimens I have prepared. As already stated, I defer a more detailed description for the present.

EXPLANATION OF PLATES XII AND XIII.

THE TICK, *Haemophysalis leachi*.

- Plate XII, Fig. 1. Blood-gorged female which has dropped from a dog and is beginning to lay eggs. The small legs are seen protruding from the distended body. When oviposition is completed the egg-mass may exceed the size of the original tick which lies beneath it half buried, finally the tick dies, having shrivelled like a raisin. Magnified about $2\frac{1}{2}$ times.
- Fig. 2. Larva with six legs, as it appears after escaping from the egg. Legs curled under. $\times 20$.
- Fig. 3. Nymph with eight legs, as it appears after escaping from the larval skin. $\times 20$.
- Fig. 4. In a row down the centre are three gorged nymphs, which give rise to adult ticks, the adult male being shown on the left, the female on the right. The male has a more glossy back, owing to the whole dorsum being covered by the dorsal shield. This is not the case in the female, as will be seen by referring to Fig. 6 which follows. The adult tick appears about the same size as the gorged nymph from which it issues. $\times 2$.
- Fig. 5. Mature male tick. $\times 20$.
- Plate XIII, Fig. 6. Mature female tick, body somewhat broader than in male, dorsal shield extending but about half-way along dorsum. $\times 20$.
- Fig. 7. Ventral view of capitulum of female, showing the hypostome with its rows of denticles lying centrally between the palpi. The denticles are directed backward, and together with the toothed mandibles situated dorsally (not seen in the photograph) serve to anchor the tick in the skin of its host. $\times 150$.

With the exception of Figs. 1, 2 and 3, the ticks have been prepared by maceration in caustic potash. The larvae in Fig. 2 are viewed dorsally, the rest ventrally, so that it is owing to the transparency of the chitinous structures that the dorsal shield is visible through the ventral wall in Fig. 6.

4. Incubation period.

In all cases a period of incubation of variable length precedes the febrile attack or onset of symptoms. This period can be most accurately determined under experimental conditions. Under natural conditions a dog showed symptoms 10 days after being on a tick veld (Robertson). In the cases reported from France by Almy, the period of incubation

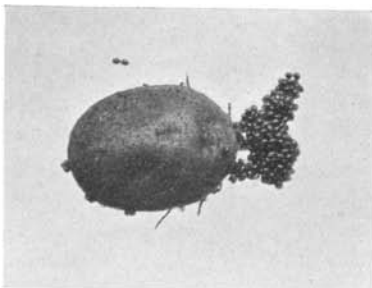


Fig. 1.

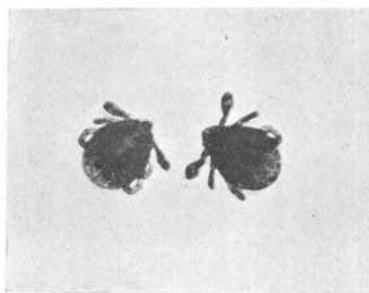


Fig. 2.

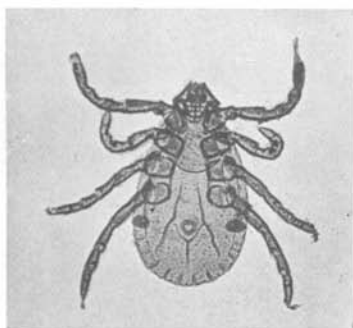


Fig. 3.

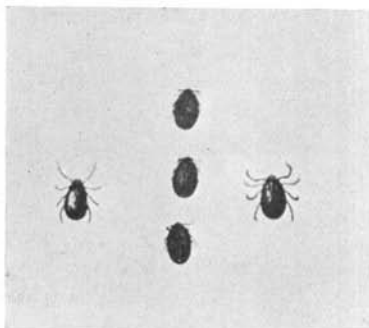


Fig. 4.

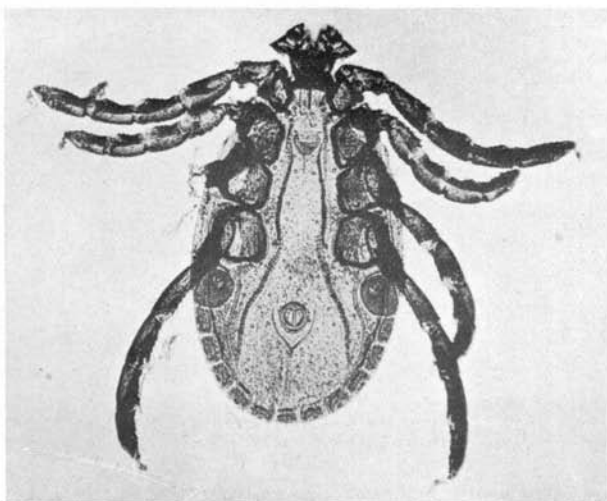


Fig. 5.

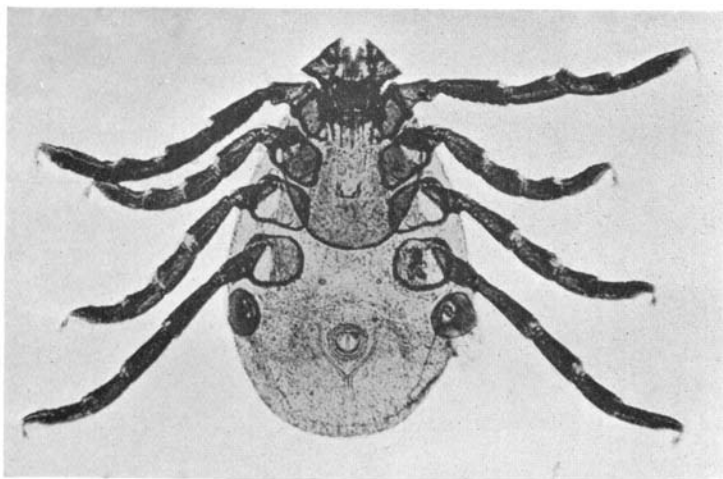


Fig. 6.

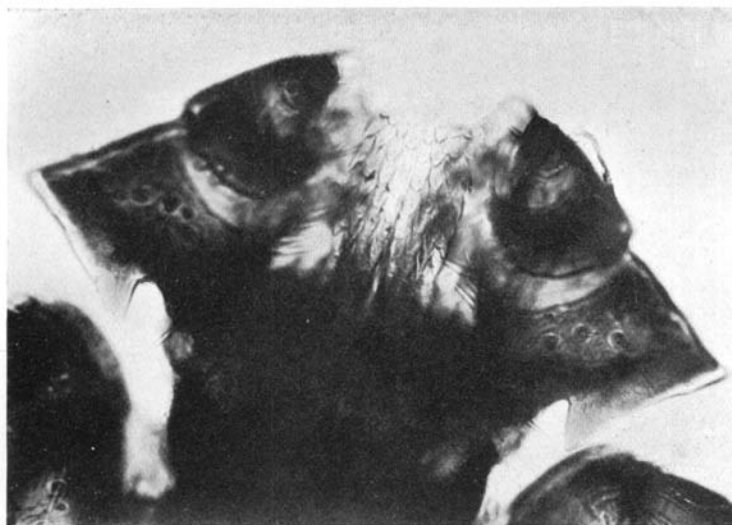


Fig. 7.

appears to have been 10 days in two cases, apparently some weeks in one case, uncertain in others.

In three dogs which I successfully infected in Cambridge by means of ticks sent me by Mr Lounsbury, fever made its appearance on the 13th, 15th, and 16th days respectively, and Mr Lounsbury has reported a case in which a dog showed symptoms on the 21st day after the ticks were applied.

In blood inoculation experiments the period of incubation varies according to the mode of inoculation employed. After subcutaneous inoculation Robertson observed febrile symptoms on the 3rd day in one, and on the 4th and 5th day in two cases, and Lounsbury on the 7th day in a case which proved chronic, and on the 6th day in another. In France, Nocard and Motas observed febrile symptoms on the 5th and 6th day in two cases. I have observed corresponding periods of incubation, but in one of my dogs (Chart IV) it lasted 10 days.

As Spreull, and subsequently Robertson pointed out, intravenous inoculation shortens the period of incubation, thus in two of the latter's cases (Chart VI) the period was reduced to 3 and 4 days respectively. Nocard and Motas record two cases, one of which proved chronic, in which the period of incubation, following intravenous inoculation, lasted 3 days.

5. Symptoms.

The disease is ushered in by fever and loss of appetite, followed by increasing prostration, ending in complete helplessness. The breathing and pulse are accelerated, the former becomes laboured and finally shallow. Beginning with the onset of fever, there is a loss of weight which is greatly increased during the final stages, and appears to be more marked in long-continued cases. The mucous membranes soon become pallid and may show icterus. The skin may also become icteric. Haemoglobinuria occurs in a certain proportion of cases.

In the table which follows, I have recorded in separate columns the symptoms of canine piroplasmiasis as it occurs in the South African and French diseases. With regard to the former, I have incorporated the observations of Hutcheon, Robertson, and Lounsbury, and added some of my own. The data regarding the French disease are condensed and tabulated from the publications of Nocard, Almy, and Motas. Although the diseases appear practically the same, judging from the symptoms recorded in both columns, it appears to me premature to conclude that they are identical until the subject has been further worked out.

SYMPTOMS OF
AS OBSERVED IN

SOUTH AFRICA

Loss of appetite	Apparently a constant and early symptom. Observed in all the dogs I have experimented on. All food refused in later stages, may drink much water (but refuse milk, Hutcheon, 1899). This symptom also noted by Lounsbury and Robertson.
Vomiting	An early symptom in some cases (Hutcheon, 1899, p. 400), but not observed by me.
Prostration	Soon after the onset, in acute cases, the animal appears dull, listless, it lies down more than usual, later it does not change its position for hours. Toward the end it totters in its gait, finally it cannot stand, becomes completely helpless, and dies comatose. These appearances observed by me are also recorded by Hutcheon, Lounsbury and Robertson.
Breathing	Accelerated, subsequently laboured irregular, finally very shallow.
Pulse	Weak and rapid.
Mucous membranes and skin: icterus, anaemia	Visible mucous membranes become pale and icteric, may become chrome yellow in colour, and also the hairless portions of the skin (flanks, belly, prepuce and inner edges of ears). [Lounsbury and Robertson.] In 6 dogs infected by me in various ways (see Protocols) in Cambridge, icterus was very slight or not evident, but anaemia was well-marked in all cases. The tongue foul and furred, the teeth dirty, gums and mucous membrane of the mouth pale, bloodless, at times ulcerated (often icteric according to Hutcheon, who with Lounsbury and Robertson, noted foetid breath).
Body temperature	Fever recorded in all cases, and may be present when the dog appears well, thus constituting usually the first symptom. Fever starts at 104.2°—105.4° and oscillates or rises to 105—106.6°, even 107° F. In chronic cases (Chart V) there may be great oscillations in temperature which may fall below normal (97—98° F., about 36° C.) and again rise. Toward death the rectal temperature gradually falls far below normal, in three of my dogs 98.2°, 97.2°, 90° F. (32.2° C.) were recorded respectively when last taken.
Loss of weight	A marked loss of weight was recorded in four of my dogs, amounting: in Dog 3 (died in 4½ days) to 6.5%; in Dogs 5 and 7 (died in 8—9 days) to 14.3% and 17.7% respectively; in Dog 1 the loss of weight amounted to 12% during the last three days alone. The loss occurs chiefly toward the end. Emaciation appears to be more marked in chronic cases, see Chart V: Dog a "living skeleton."
Urine: haemoglobinuria	All the cases I have observed have been acute and haemoglobinuria was present, also albuminuria. The urine was claret or brownish-red in colour, or resembling coffee-grounds. Lounsbury and Robertson consider this brown colouring an unfavourable symptom, indicating a fatal termination. Haemoglobinuria was noted by Hutcheon (1899). It may be absent in fatal cases as in redwater (Robertson, p. 329). In one urine I examined (Mr Graham-Smith will report upon the other cases later) I found the reaction acid, albumen, haemoglobin, bile salts and pigments, a considerable deposit consisting of spermatozoa (chiefly), granular casts, epithelium, leucocytes, granular detritus, crystals of salts and a few erythrocytes. There were no spermatozoa or bile salts and pigment present on the day preceding death (Dog 1). No haemoglobinuria was observed in the chronic case recorded in Chart V.

CANINE PIROPLASMOSIS

THE DISEASE OCCURRING IN

FRANCE

Loss of appetite at onset in acute cases, and appetite not regained. Anorexia also observable in chronic cases.

A rare symptom, occurs in acute cases, intractable when present. Greenish mucus expelled.

In acute cases dog dejected, lies in a corner. Sensibility greatly lowered. Movements difficult, muscular twitching, especially of hind limbs, followed by paresis. Dog rises with difficulty and may tumble over. When temperature becomes subnormal dog ceases to move. Dies comatose. Only once were tetaniform convulsions seen to precede death (N. and M. p. 261).

In chronic cases lethargy and muscular weakness accompany the profound anaemia.

In acute cases respirations 36—48 a minute (accelerated), laboured, gasping, and at times, especially in young dogs, accompanied by whining sounds. Examination of thorax negative.

In acute cases beats 120—160 a minute, rapid, weak, thready, often intermittent.

In acute cases visible mucous membranes become pale, then cyanotic, and in some cases (inconstant) icteric. Icterus noted in the mucous membranes of the eye, mouth, and skin in 30 out of 63 cases observed (p. 260).

In chronic cases profound anaemia, rarely icterus. Mucous membranes very pale, skin dry, coat dull.

In acute cases fever at onset may exceed 40° C. (104° F.), is maintained usually 2—3 days, then the temperature falls below normal, even down to 33° C. (91.4° F.). Rarely temperature is seen to oscillate, then gradually fall. In young dogs which die very quickly initial fever may be absent, parasites appear in the blood and temperature sinks until death.

In chronic cases fever usually absent, slight when present, rarely exceeds 40° C. (104° F.). May be overlooked, lasts 36—48 hrs., then falls. In one case a "quartan fever," with remissions as in the human malaria observed.

Nocard and Motas do not mention anything about loss of weight in acute cases, but in chronic cases they state there is emaciation.

In acute cases: urine *albuminous* at onset before parasites can be found in the blood. Albuminuria persists until death, increasing with number of parasites present. *Haemoglobinuria*: urine pink, dark red, blackish, like prune-juice or coffee-grounds according to its degree. No erythrocytes in urine. Oxyhaemoglobin may amount to 2.5%. Haemoglobinuria appears soon after parasites are seen in the blood and in very acute cases persists until death and is found in bladder at autopsy. Haemoglobinuria inconstant, noted in 3 out of 6 cases by Nocard and Almy, this may be due to its being at times very transitory. Nocard and Motas observed more or less lasting and severe haemoglobinuria in 43 out of 63 dogs. *Bile pigment* present in cases showing icterus and haemoglobinuria. *Reaction acid*, only found neutral twice, alkaline once. Polyuria rare.

In chronic cases: urine usually *slightly albuminous* at start, condition lasting 15—20 days. *Haemoglobinuria* very rare, lasts 1—2 days. Urine may be icteric. *Reaction acid*, only once found neutral, this attributable to other causes (sugar found).

The Duration of the Disease, reckoned from the appearance of the first febrile symptoms until the occurrence of death, varies according to the acute, subacute, or chronic character of the disease. In extremely acute cases the illness may last only 24 hours, but it appears more often to last 3 to 6 days. In subacute cases the disease lasts about 10 days, and in chronic cases anywhere from 21 to 62 days. These statements are based upon the following table in which I have included all recorded observations possessing a suitable character in point of accuracy.

Disease acquired	Duration in days	Authority	Remarks
Naturally by Tick infection	4	Lounsbury, Robertson	1 case in S. Africa
	5—6	Almy	1 case in France
Experimentally by Tick infection (<i>H. leachi</i>)	1, 1, 5, 11	Lounsbury, Robertson	4 cases, two first in puppies
	8, 9, 10½	Nuttall	3 cases in Cambridge
Experimentally by subcutaneous inoculation with infected blood	5	Spreull	1 case
	2—3	Robertson	A general statement
	4½	Nuttall	1 case in Cambridge
	9, 10, 11	Nocard and Motas	A general statement, disease in France
	21—30—40, 60	„ „	Ditto, in chronic cases
	62	Lounsbury	Case recorded in Chart V, chronic
Experimentally by intravenous inoculation with infected blood	3	Spreull	1 case in S. Africa
	4—5	Nocard and Motas	A general statement

For convenience' sake I have included the observations by Nocard and Motas (1902, p. 273) upon the French disease in the above table. They say that the disease may last 36—40 hours in very young puppies, but that it usually lasts 3 days after the appearance of the parasites in acute cases.

I have not as yet had an opportunity of observing the chronic type of the disease, but through the courtesy of Mr Lounsbury I am able to present an unpublished record (Chart V) of a case lasting as long as 62 days from the onset of symptoms, and ending in death after what appear to have been unsuccessful attempts at recovery. At the end of about a month the dog had become a "living skeleton," subsequently its temperature fell to subnormal on three days and then again rose, pursuing an irregular course throughout. Toward the end of the second month the dog became blind in both eyes. Robertson (5. VI. 1902, p. 682) says that many dogs, after practically recovering from

the jaundice, "died of anæmia and prostration long after all clinical symptoms of the disease had disappeared." Hutcheon (1899) attributed death to exhaustion. Others have attributed death to collapse and weakness due to the drain on the blood consequent on the hæmoglobinuria, but Lounsbury points out the fact that deaths occur in the absence of hæmoglobinuria. It appears from the experience of Nocard and Motas (1902, p. 258) in France that the acute disease is usually fatal, whereas the chronic type usually ends in recovery. In such cases recovery proceeds very slowly, lasting 6—12 weeks.

6. Pathology.

To avoid confusion between the canine piroplasmosis observed in Europe, notably in France, and the South African disease, I have compiled the following brief table relating to the pathology in the same way as I did the one for the symptoms. Mr Graham-Smith has made the pathology of the disease the subject of special study, and will report thereon in due course. The appearances at autopsy are identical in naturally and experimentally infected dogs. In dogs dying from the acute type Nocard and Motas state that there may be an entire absence of abnormal pathological appearances at autopsy. According to these authors lesions are most marked in chronic cases, and the changes observed in the various organs point to their depending upon the "extreme dilatation of the capillary network by the accumulation therein of corpuscles mostly gorged with parasites¹."

Haematology.

The changes in the blood in the South African disease are being made the subject of study by Dr Wright, so that I shall leave it to him to report thereon in a later paper. On the other hand I herewith append an abstract of the observations recorded by Nocard and Motas (1902) for the French disease. What observations I have made agree very closely with those of the French observers. This might be expected from the great similarity between the descriptions of the symptoms and pathology, etc.

In acute cases of the French disease (p. 261) the blood appears profoundly altered, pale and watery. Coagulation is retarded, the coagulum softer and paler than usual, the serum tinted more or less

¹ Refer to p. 223 for the earlier observations of Piana and Galli-Valerio in Italy.

PATHOLOGY OF

AS OBSERVED IN

SOUTH AFRICA

External appearances	Great emaciation, extreme pallor of all visible mucous membranes and hairless portions of skin (Robertson).
Blood	Thin and watery (Lounsbury, Robertson). The serum tinged with haemoglobin.
Spleen	Much enlarged, congested (Hutcheon). Slightly enlarged, soft, pale, bloodless, like other organs scarcely stains paper when smeared thereon. Parasites therein numerous (Robertson). Little change observed in our cases.
Liver	Usually congested, at times inflamed, mahogany or saffron-colour. <i>Bile</i> dark green, usually thicker than normal (Hutcheon). Liver enormously enlarged, soft, "distended with watery fluid" (Robertson). But slightly changed macroscopically in our cases.
Kidneys	Congested more or less, at times oedematous, cortex dark brown (Hutcheon). Kidney pale and friable (Robertson).
Bladder	Normal, urine generally dark brown like "pontac" (Hutcheon). Urine may appear normal (Robertson). <i>See</i> Symptoms p. 232.
Serous cavities	Peritoneal and thoracic cavity may contain fluid (Hutcheon). Sometimes brownish serous exudation in thorax. In Lounsbury's chronic case (Chart V) pericarditis and pyothorax.
Stomach	Fundus generally inflamed, pyloric end normal, occasionally a few ulcer-like spots on rugae of stomach (Hutcheon).
Intestines	Catarrhal inflammation of small intestine, more intense about duodenum. Lumen contains viscid mucus often mixed with blood. Large intestine slightly but not uniformly inflamed, contains much viscid mucus (Hutcheon).
Heart	Pericardium contains variable amount of serous fluid. Ecchymoses around heart, largely on left ventricle (Hutcheon).
Lungs	Rarely affected, according to Hutcheon. I noted oedema and pinkish frothy fluid in the bronchi and trachea.
Bone-marrow	Congested.
Lymphatic glands	In one of my dogs the retroperitoneal glands appeared injected, the others unchanged.
Central nervous system	Slight congestion in <i>some</i> of our cases.
Icterus	In some cases the tissues in general are yellowish (Hutcheon).

CANINE PIROPLASMOSIS.

THE DISEASE OCCURRING IN

FRANCE

Heart and large blood vessels contain soft clots composed mostly of fibrin, floating in haemoglobin-stained serum.

Often enlarged 3—4 × natural size, softened, dark, reddens on exposure to air. No changes may be noticeable in acute cases. Parasites numerous therein in either case. Splenic enlargement when present can be determined during life.

Usually congested, shows little change. Blood flowing on section is rich in parasites. *Bile* usually thick, syrupy, grumous or dark green, and distends gall-bladder.

In an acute case the size of liver was normal, colour yellowish, gall-bladder filled with syrupy bile.

Greatly congested usually, capsule easily stripped, revealing numerous petechiae. On section cortex seen to be congested, shows petechiae. Blood from kidney very rich in parasites. In an acute case yellowish-red fluid exuded on section.

Prune-juice-like urine found in an acute case.

Mucosa infiltrated and congested on a level with the duodenum in a few cases.

Pericardium contains yellow or bloody fluid. Not infrequently observe numerous petechiae about apex or beneath endocardium of left heart. Heart may be pale (acute case).

Often small apoplectiform foci. In young dogs dying quickly usually acute oedema and reddish foamy secretion in bronchi and trachea.

Usually intensely congested, foetal in appearance, soft, friable, rich in endoglobular parasites.

Rarely affected.

Nothing special, meninges slightly congested.

Intense icterus of mucous membranes, sclerae, skin and fat recorded in an acute case.

by haemoglobin. Where haemoglobinuria is succeeded by icterus, the serum appears deep yellow, showing a greenish reflex at times. Citrated blood, on standing, shows a layer of corpuscles corresponding to $1/5$, $1/10$, $1/15$ th of the plasma.

Whereas the erythrocyte count in the normal dog amounts to 6·5 to 7 million (Malassez method), a progressive decrease is observed in dogs affected with piroplasmosis. The decrease begins gradually with the onset of symptoms, and with the appearance of haemoglobinuria there is a sudden fall to 2 million or less. There is then a corresponding fall in the percentage of haemoglobin, namely to 13—12—6·4—3·5%. The erythrocytes are not simply reduced in numbers, they are altered, so that in stained films they appear of different sizes, some all the way from a third to two-thirds larger than normal, and taking the stain poorly. Nucleated erythrocytes are also observed especially in chronic cases. Erythrocytes containing many parasites are much enlarged, and, bursting, liberate the parasites. Parasite-containing corpuscles as well as parasites may be taken up by leucocytes (p. 271).

There may be considerable leucocytosis, the number of leucocytes being increased 2—3—4 times the normal, so that instead of having 7—8000 (normal) as many as 40,000 may be counted. The multiplication almost entirely affects the polynuclear elements, this being especially marked in slow running cases.

Nocard and Motas (p. 289) give the following record as typical for an acute case (see Chart VII, Case II).

Day	Erythrocyte count	Remarks
1	5,240,000	Intravenous inoculation with 2 c.c. of virulent blood
2	5,560,000	Dog well
3	5,960,000	Dog well apparently, temp. 40° C., few parasites
4	5,240,000	Dog dejected, pulse 124, many motile parasites
5 (morning)	2,600,000	Dog miserable, hind limbs paralyzed, temp. 35·6° C.
(afternoon)	2,200,000	Condition worse, temp. 33·5° C.
6	—	Found dead

In chronic cases (p. 264) the progressive anaemia is explained by the blood changes. Here the erythrocyte count falls to 2 or 1·2 million, a fall in numbers being especially noticeable after a fall of fever, and continuing after the parasites have apparently disappeared or are rare. A slow increase begins in 25—30 days in cases leading to recovery, a normal count being reached after 2—3 months. The loss of haemoglobin is less marked than in acute cases, thus, in a dog showing 2,760,000 erythrocytes the haemoglobin gave 9·5%. Stained films

show, even better than in acute cases, great differences in the size of the erythrocytes; they may be enlarged 2—3 times above the normal. The enlarged corpuscles take the stain feebly. Many nucleated corpuscles may be found, especially at the commencement of corpuscular decrease.

Leucocytosis is always marked, the count amounting to 15,000—30,000, and in one case even to 54,000. The increase of leucocytes is due equally to mononuclear and polynuclear elements. Where there has been fever, on the days succeeding a fall of temperature, leucocytes—mononuclears only—are encountered which contain erythrocytes laden with parasites, a condition rarely met with in acute cases. As recovery takes place, the erythrocytes increase, the leucocytes decrease, agglomerations of haematoblasts being however encountered.

The following record (p. 289), which I have also remodelled, is given as being typical of a chronic case, ending in recovery. The dog was inoculated intravenously with 2 c.c. of virulent blood obtained from one of Almy's naturally infected dogs (No. 3) (see Chart VII, Case I).

Day	Erythrocyte count	Remarks
1	5,840,000	—
2	—	Few parasites found
3	—	More parasites. Eating less
4	—	Dog dejected, appetite gone, numerous motile parasites
6	4,040,000	Few parasites found, lying down, mucous membranes pale
8	2,820,000	Profound anaemia, insensibility, very few parasites
10	1,520,000	Same condition, nucleated erythrocytes numerous, leucocytes = 54,000
15	1,200,000	Same condition, no parasites found, leucocytes = 10,000
18	2,120,000	Slight improvement, no parasites found
20	2,480,000	Distinct improvement, few nucleated reds, no parasites found
25	4,380,000	Improving rapidly, lively, hungry, mucosae pink, no parasites found
27	5,100,000	Haematoblasts numerous, but one parasite found
—	—	Dog considered cured

According to Nocard and Motas (pp. 277—278) it is especially in such cases as the preceding that phagocytosis is observed, that is in cases leading to recovery. The mononuclears may include as many as 2—6 infected corpuscles, which are more or less degenerated. Phagocytosis is observed in the peripheral blood, but is most marked however in the spleen, where it may be seen even in dogs dying of the acute disease. When the corpuscles included are numerous, the gorged leucocyte may simulate the cross-section of a capillary.

7. Natural Mode of Infection.

The part played by adult ticks, of the species *Haemophysalis leachi* (Audouin), in the transmission of the disease to healthy dogs has been mentioned in part on p. 226. A similar part has not been demonstrated for the European dog tick, but there is circumstantial evidence that it is capable of performing a similar rôle.

The following case, cited both by Lounsbury and Robertson (1902, p. 333), evidently serves as a type of the usual way in which the disease is acquired in South Africa: A small, aged pointer dog, belonging to a gentleman living in the suburbs of Cape Town, was taken out on the veld after having been kept at home for about two years. The owner stated that the dog had had the disease previously. Ten days before the last illness he took the dog shooting to Kuils, a very bad tick veld. The animal returned with ticks upon him, fell ill after a week, and died on the fourth day of illness.

There is no evidence pointing to any other mode of infection in nature. According to Robertson (1902, p. 327) the disease is not communicable by ingestion, nor when healthy dogs cohabit with diseased ones. As Lounsbury (1901, p. 10) points out, fleas (*Pulex serraticeps*) and dog-lice are evidently not suitable hosts for the parasites, for the disease has not been observed amongst dogs in places where these fleas abounded, and in which healthy and diseased dogs were housed together. The disease is moreover rare in suckling pups, although they suffer as much as adult dogs from fleas and lice.

As stated, there is no direct evidence to prove the agency of ticks as carriers in the disease observed in France by Nocard and Motas. Nevertheless they bring forward strong presumptive evidence. In all the cases recorded both by them (1902, pp. 274—275) and by Almy (10. x. 1901, p. 375), some 7 in all, the dogs which had acquired the disease under natural conditions had been recently covered by ticks, in most instances after hunting, about Alfort, in wooded or shrubby country, or being housed in tick-infested kennels. These authors suspect a certain species of tick, *Dermacentor reticulatus*, of probably being the usual carrier of the disease in France. Experiments carried out by Nocard and Motas with a view, if possible, of demonstrating this, gave negative results. This was doubtless due to their experimenting with the *larvae* of this tick, a stage which appears to be incapable of being infective, certainly in the case of *H. leachi*. The *larvae* of *D.*

reticulatus were observed to fall off soon after they were put on the dogs, and they were lost.

The histories of 5 cases of Piroplasmosis occurring in dogs brought to the clinic at Alfort between 2 Sept. and 8 Oct. 1901, recorded by Almy (10. x. 1901, pp. 375—379), are herewith appended in evidence of the part probably played by *Dermacentor reticulatus* in the transmission of Piroplasmosis in France:

Dog 1. Had been in Tunis¹, where it had fallen ill some weeks before, having been attacked by ticks there (species undetermined). When examined the dog was profoundly anaemic. Erythrocytes numbered 1,260,000. Piroplasma found. Dog died.

Dog 2. Had hunted in the department d'Eure-et-Loire, from 1 to 3 Sept. Attacked by ticks. Fell ill, 9 to 10 Sept., haemoglobinuria noted 11th and Piroplasma found in the blood. Icterus followed; and death on the 15th. Typical appearances at autopsy.

Dog 3. Had hunted two days before and numerous ticks were found on him. Came to Alfort 24 Sept., anaemic, erythrocyte count 2,500,000, Piroplasma found. 7 Oct. the erythrocytes numbered 4,100,000 and the dog was apparently recovering.

Dog 4. Received in the Alfort clinic 5 Oct., suffering from haemoglobinuria. Had been in the woods 10 days before, and been infected by many ticks. Piroplasma numerous. Icterus followed. 12 to 15 Oct. appeared to improve; 26th, temperature sank to 34° C., 27th, to 22° C. (per rectum), died during the night.

Dog 5. Admitted 8 Oct. to Alfort clinic, being off his feed. Many ticks observed on him during the previous days. Piroplasma found. Left clinic 24 Oct. apparently quite recovered.

It is evident from the foregoing, as well as from the observations in South Africa, that hunting dogs are chiefly affected, for the reason that they are more exposed to the attacks of ticks. It seems futile therefore to mention the objections advanced by Leblanc (10. x. 1901, p. 380), who claimed that the part played by ticks could not be important for the reason that dogs are frequently tick-covered without developing the disease. Leblanc's objections were fully met by Nocard (10. x. 1901, p. 381) when he stated that it was fallacious to conclude that all dogs must get piroplasmosis if attacked by ticks. To begin with, dogs are attacked by different species of ticks, some of which may not be suitable hosts for the parasite, and a suitable species of tick may frequently not be infected with the parasite. I would add that Leblanc's objection is of the same nature as that brought forward by certain persons against the part played by *Anopheles* in the transmission of malaria, and is based simply on ignorance of facts.

¹ May have been infected there.

8. Infection Experiments.

1. *Infection of dogs by blood inoculation.*

The first to communicate the disease from diseased to healthy dogs by means of blood containing parasites were Purvis and Spreull, in S. Africa. Hutcheon (1899, p. 399), who reported Spreull's observations, states that the injection of small quantities of infected blood caused the death of two dogs. The dog which received a subcutaneous injection of virulent blood showed fever on the 6th day and died on the 11th day after inoculation. The dog receiving an intravenous injection showed fever on the 4th day and died on the 7th day after inoculation. The more rapidly fatal disease following upon intravenous inoculation has also been observed by Nocard and Motas in the French disease. These observers (1902, pp. 263, 273) inoculated dogs subcutaneously, and intravenously and also into the muscles. Intravenous injection produced a more rapid and certain infection. Similar observations have been made by Robertson (1901, p. 331) and Lounsbury (1901, p. 9¹) at the Cape. Death followed 11—13 days after subcutaneous injection in their animals, the illness lasting 2—3 days. The observations made both in France and in South Africa show that the experimentally produced disease is in all respects similar to that naturally acquired. (Robertson, p. 329, Nocard and Almy (28. III. 1901).) I have considered the acute and chronic types of the disease on pp. 232—235. Also in Charts I—VII.

Robertson (1901, p. 331) transmitted the disease by inoculation through a series of 13 dogs and thought (p. 332) that the disease "was aggravated and rendered more virulent, by passage through dogs."

The amount of virulent blood required to produce infection appears to vary. In young dogs, which are more susceptible, Nocard and Motas (iv. 1902, p. 273) found the intravenous injection of 1 drop to be sufficient to kill, whilst adult dogs developed the disease after the

¹ Lounsbury (1901, p. 9) records two experiments made in conjunction with Robertson, in which death took place sooner in a dog inoculated subcutaneously. This is evidently due to this dog being young, and consequently more susceptible (see p. 245). The experiments were as follows:

- I. Irish terrier pup, 3 weeks old, received 3 c.c. of infected blood subcutaneously (9. ix. 1901). Parasites appeared in its blood after 6 days, death occurred on the 8th day.
- II. Adult dog, received 3 c.c. of infected blood into the jugular vein. Parasites appeared in its blood on the 4th, and the dog died on the 11th day after inoculation.

injection of 1 c.c. of virulent blood. Robertson (VI. 1902, p. 682) in Africa found the duration of the disease, ending in death, to vary "in certain cases with the quantity of infecting matter used." In the experiments reported by Lounsbury (1901), successful infection followed the injection of 2—3 c.c. of virulent blood.

2. *Infection of dogs by means of Ticks (Haemophysalis leachi*
[Audouin]).

The first experiments on the infection of dogs with ticks were made in South Africa by Lounsbury. The initial experiment, recorded by Robertson (1901, p. 333), was unintentional in so far as it was conducted with the view of studying the life-history of the tick.

A small rough-haired mongrel dog was kept for two months in close confinement in a glass case by Lounsbury, the dog serving as a host for ticks obtained from various domesticated animals. During the course of its confinement the dog developed piroplasmosis. Prior to the attack, which ended fatally, ticks had been placed on the dog, the ticks having been obtained from the vicinity of Cape Town. Some of these ticks were derived from salted or immune dogs, others from a dog affected with piroplasmosis.

Lounsbury (1901, p. 9) moreover reported 5 experiments on dogs, of which I append the following abstracts :

EXPT. I. *Irish terrier pup.* 23 Sept. 1901. Ten adult ticks (sexes equally divided) were placed upon the pup, which was kept isolated, and fed on condensed milk. 29 Sept., 20 more adult ticks were placed on the dog (10 ticks of each sex). 4 Oct., that is 12 days after the first lot of ticks were applied, the dog's temperature rose, and parasites were found in its blood. The dog remained active and showed good appetite until 8 Oct., when it showed signs of prostration. The dog died 9 Oct., that is on the 5th day after the onset of symptoms.

EXPT. II. *Adult dog.* 23 Sept. 1901. Ten adult ticks (sexes equally divided) were placed on the dog. 4 Oct., that is 11 days later, the dog's temperature rose, parasites being found in its blood. Fever continued for five days, then subsided, to return two days later, the dog becoming very ill. The dog was found dead on the morning of the 15th, that is 11 days after the onset of symptoms.

Two control dogs chained near to the preceding dog in the same shed, remained healthy, obviously for the reason that the infected ticks had not migrated to them from the animal upon which they had been placed.

EXPT. III. *Dog.* Showed distaste for food 21 days after application of infected adult ticks. On the 24th, refused food, on the 30th day parasites were found in its blood by Robertson. Diagnosis confirmed by autopsy.

The two following experiments were made on dogs, first with larvae, then with nymphs and adult ticks (Lounsbury, pp. 7—8). The ticks were derived from two blood-gorged females (*H. leachi*) found on the preceding dog (III.) after death.

EXPT. IV. 15—20. VI. Larvae placed on the dog dropped off after a few days. After undergoing metamorphosis into nymphs these

25. VII. Nymphs were placed upon the same dog. The nymphs dropped off, were allowed to undergo metamorphosis into adults (took three weeks).

The dog remained healthy.

27. VIII. Adult ticks were now placed on the dog. (This dog was probably bitten also by a second batch of larvae placed on Dog V. in the same pen, but which migrated to Dog IV.) Nothing noticed until

9. IX. Dog's temperature 105° F., appeared well, parasites in its blood. 12 hours later both this dog and No. V. became prostrated, showed haemoglobinuria.

10. IX. Dog dead.

EXPT. V. 15—20. VI. Dog kept in same cage as No. IV. It was probably bitten by the larval ticks applied to the latter. Dog V. was isolated when the nymphs were applied to Dog IV. (25. VII.), and again isolated (31. VII. to 3. VIII.) when a new lot of infected larvae from another source were applied.

27. VIII. Some of the adult ticks placed on Dog IV. on this date wandered on to Dog V.

(28. VIII. Nymphs derived from first lot of larvae placed on Dog IV. were put on Dog V.)

9. IX. Fell ill with same symptoms as Dog IV. (*q.v.*).

10. IX. Dog dead.

The negative results obtained in Experiments IV. and V., conducted with larvae and nymphs of *Haemophysalis leachi*, obtained from eggs laid by ticks removed from dogs suffering from piroplasmosis, led Lounsbury to conclude that *Piroplasma canis* is transmitted through the parent tick to its progeny. He wrote at the time (p. 10) that "such progeny normally remains incapable of transmitting the infection it inherits until it attains the adult stage. That the infection passes through the egg stage is a fact not unparalleled, and therefore not surprising; but that it is harboured through two feeding stages without being transmitted, is at present wholly unique, and is a fact that considerably strengthens the inference that in this disease and redwater, the ticks are not merely carrying agents but are secondary hosts of the protozoan parasites."

9. Immunity.

Animals affected: Robertson (1901, p. 332), has failed to infect other animals than the dog. He injected these animals with blood derived from a diseased dog, the blood showing parasites on microscopic examination. His results were negative with the following animals: horse, ox, sheep, cat, rabbit, guinea-pig, rat, mouse, fowl.

Similarly, Nocard and Motas (iv. 1902, p. 275), experimenting with the French disease, failed to infect the horse, ox, sheep, goat, cat, rabbit, guinea-pig, white rat, white mouse, fowl and pigeon. They never found parasites in the blood corpuscles of these animals although they received intravenous, intramuscular, and subcutaneous injections of infected dog's blood.

Influence of Breed and Age on Susceptibility: According to Hutcheon (1893), writing of the disease in South Africa, "imported and well-bred dogs are more liable to become affected than Colonial bred ones, but the latter become affected also. Dogs of all breeds appear subject to it." Robertson (1901, p. 327) says the disease attacks Cape bred and imported dogs indiscriminately, and (p. 336) that it affects both old and young dogs, as proved by inoculation. The South African observers do not state that young dogs are the more susceptible (see footnote, p. 242).

In France, Nocard and Motas (iv. 1902, p. 275) found young dogs to be much more susceptible than adults. Experimenting with puppies aged 2—12 weeks, they found not only that they were more readily infected, but that in them the disease ran a more acute course and that the termination was invariably fatal.

Immunity after Recovery: Both in the South African and French disease immunity has been seen to follow upon recovery. Thus, Lounsbury (xi. 1901, pp. 11, 12), in Africa, seems to have observed immunity, for he writes "one is inclined to infer the probability of practically continuous infestation by ticks being a necessary adjunct to complete immunity, as is known to be actually the case in redwater." Immunity would not appear to last long in the absence of "continuous infestation by ticks." Lounsbury cites the case of a dog which died from the disease having suffered from it two years previously.

The immunity which follows on recovery was tested experimentally by Nocard and Motas (iv. 1902, p. 277), in the French disease, on dogs which had passed through the disease both naturally or artificially

acquired. They inoculated large quantities of virulent blood into five such dogs after periods of time of 2, 2½, 2½, 3, 6 months respectively after recovery and found the animals in all cases to resist infection, control animals dying in every case.

It is worthy of note that in "salted" dogs in South Africa *the parasites evidently persist many months in the blood* after apparent recovery as proved by inoculations practised with their blood on fresh dogs. Thus Robertson (5. vi. 1902, p. 682) found that the fresh blood of a salted dog (No. 11), in which no parasites could be found by painstaking microscopic examination, was nevertheless capable of setting up infection in a fresh dog. The term "salted" is applied to dogs which have recovered from the disease and have acquired immunity. The salted dog had had the disease in the latter part of October. This dog's blood was used for inoculating fresh dogs as follows:

No. of dog inoculated	Date of inoculation with blood freshly obtained from salted dog	Amount of blood of salted dog injected in c.c.	Result
1	4 November, 1901	6	Dead in 10 days
2	17 " "	4	" 17 "
3	26 " "	4	" 8 "
4	3 December "	2	" 19 "
5	17 " "	6	" 13 "
6	6 January, 1902	7	Dead after many weeks
7	24 " "	5	Dead in 15 days
8	3 February "	6	" 18 "
9	25 " "	20	" 8 "
10	27 " "	27	" 3 " (from shock)
11	13 March "	2	" 34 "

These experiments show very conclusively that the blood of an apparently recovered dog may contain parasites for months (4 Nov. to 13 March) and there is no evidence that the parasites had become attenuated, for the last dog, which died on the 34th day after inoculation, it will be noted, only received 2 c.c. of the salted dog's blood, the smallest amount injected in the whole series excepting Dog 4. It is not impossible that the parasites may however become attenuated with time, but this remains to be proved. Dog 6 in the above table (constructed by me from Robertson's protocols) is obviously identical with the dog whose temperature etc. is recorded in Chart V, and for which I am indebted to Mr Lounsbury.

This persistence of virulent parasites, in the blood of apparently recovered or "salted" animals, has its parallel in what has been

observed in Texas fever by Schroeder (1900, pp. 42—43)¹ in the United States, only he has shown that the parasites (*Piroplasma bovis*) may be harboured for a very much longer time, namely from 1 to 6 years (!).

That the parasites may become attenuated is suggested by the observations of Nocard and Motas (1902, p. 273), who state that the blood of dogs suffering from the chronic disease is less virulent than in acute cases. Even allowing for the small number of parasites present in blood from chronic cases, large doses thereof usually produced but a mild form of disease. In one series of their experiments, moreover, the blood of a convalescent dog (parasites present) produced but a mild disease when injected into fresh dogs, all of the latter recovering.

Nocard and Motas made a number of observations which possess very considerable interest in relation to immunity. They found (pp. 278—281) the blood of immune dogs germicidal, for on mixing virulent blood with the serum of a recovered dog and injecting the mixture into a fresh dog, they failed to infect the animal. Such injections did not however confer immunity on dogs for they died as quickly as control animals, that is, 12 to 15 days after subsequent inoculation with virulent blood. Virulent blood and the serum of a recovered animal injected separately resulted in a fatal infection. Evidently then the immune-serum must act directly and in a certain concentration to be effective. The germicidal power of the immune-serum, absent in normal dog serum, was destroyed by exposure for 30 minutes to a temperature of 56° to 57° C. The germicidal power of the serum was greatly increased in recovered dogs which had been subjected to repeated inoculations with virulent blood.

Artificial Passive Immunity. Nocard and Motas (pp. 281—283) found that small doses of recovered dog's serum were not obviously protective, but large doses were markedly so, when injected 24—48 hours before infection with virulent blood². The immune-serum may retard the progress of the disease or completely check it. Much more potent was the serum of hyperimmunified animals, namely dogs treated with virulent blood inoculations after recovery. Here 3—5 c.c. of their

¹ Schroeder, E. C. (1900). A note on the persistence of the Texas fever organism in the blood of cattle. 16th Annual Report of the Bureau of Animal Industry for the Year 1899. U.S. Dept. of Agric., Washington, pp. 42—43.

² Carrington Purvis (cited by Hutcheon, 1899, p. 401) was the first to recommend a trial of this mode of preventive treatment, which is similar to that which has been tried in the case of Texas fever.

serum sufficed to protect dogs, control animals dying in 6 days. The immune-serum was still protective after having been heated to 56—57° C. A much smaller dose (p. 284) of heated immune-serum was required for immunization when it was mixed with virulent blood prior to injection. Infected erythrocytes exposed to the action of immune-serum, and then washed, were also capable of conferring immunity when injected; thus, in one experiment 20 drops of virulent blood were mixed with 50 drops of immune-serum and allowed to stand, being injected after they had been centrifugalized thrice and washed twice. Blood rendered avirulent by heat did not afford any protection.

The passive immunity obtained in the manner above described was of but brief duration. Whereas dogs receiving immune-serum injections 24 hours before those of virulent blood remained well, an inoculation practised 11—35 days after injection of immune-serum resulted in infection, the dogs showing few parasites in some cases, dying in others.

The serum of a sheep treated with virulent dog's blood developed germicidal properties, although the animal proved naturally immune to infection. The serum of the treated sheep did not afford any distinct protection when injected into dogs.

Immune-serum (p. 285) was proved to also possess curative properties when obtained from hyperimmunified dogs, and injected 24—42 hours after infection with virulent blood. The immune-serum was ineffective once parasites had appeared in the infected dog's blood.

10. Treatment.

Apart from the specific treatment recorded above, there is very little to note regarding treatment. In South Africa, Hutcheon (1893, p. 477, and 1899, p. 400) recommended the use of repeated doses of ammonium chloride and belladonna, a form of treatment tried by Borthwick at Port Elizabeth with "excellent results." Subsequently Hutcheon obtained encouraging results from the use of quinine, benzoate of soda, and carbolic acid. Robertson (1901, p. 332) states that he has tried quinine, calomel, ammonium chloride, extract of belladonna, carbolic acid, and finally benzoate of soda without satisfactory results. Carbolic acid appeared in fact to hasten death. He obtained the best results from a "calomel pill to start with, then a calomel and quinine pill four times a day." Without stating the dose, he says that very large amounts of calomel are needed. Hutcheon does not appear to approve of the calomel treatment. In other words the

evidence as to treatment in South Africa appears to be somewhat contradictory.

In Europe, Piana and Galli-Valerio (1895) attributed the recovery of the one dog they saw suffering from Piroplasmösis to the use of quinine. Almy (10. x. 1901, p. 379) treated his dogs with quinine bromhydrate, but observed no effect therefrom, the remedy being as ineffective as quinine has been shown to be in the treatment of Tristeza (*Piroplasmösis bovis*).

Evidently there is no known remedy for canine Piroplasmösis, and it is open to question whether or no the dogs which have been successfully treated would not have recovered anyhow.

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So as to avoid overloading the text I have omitted to mention my authorities for the statements I have made regarding this disease. Some 250 papers have appeared on the subject, most of these are given in the above bibliographies. For the reason that no complete bibliographies exist for the following diseases, I give them as follows:

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¹ I am indebted to Prof. Galli-Valerio for kindly sending me reprints of these otherwise inaccessible papers.

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Postscript.

Whilst this paper was in the press I had an opportunity of consulting a further publication by Wilson and Chowning (2. i. 1904. "Studies in Pyroplasmosis hominis ['Spotted Fever' or 'Tick Fever' of the Rocky Mountains]." *Journ. of Infectious Diseases*, vol. i., No. 1, pp. 31—57. Map, Charts, 2 Plates). The coloured Plate shows parasites stained by modifications of the Romanowsky method and the figures are much more convincing than those which appeared in the earlier paper by these authors, cited by me on p. 221. We may therefore accept the name (*Piroplasma hominis*) which they give to the parasite.

Wilson and Chowning (p. 52) cite H. P. Johnson (*Proc. Am. Veter. Med. Assoc.* 1903) as having observed *Piroplasma ovis* in sheep suffering from "pyroplasmatic ictero-haematuria" in Deer Lodge Valley, Montana, U. S. A.

CHART I. Tick infection experiment. Dog 1. Mongrel Terrier. 40 ticks placed on him 1st and 6th day caused onset of fever on 16th day. 11 ticks put on after onset (18th day) so that they might become infected from the dog. First ticks began dropping 13th day. Dog off his feed 18th day. Very weak 23rd.

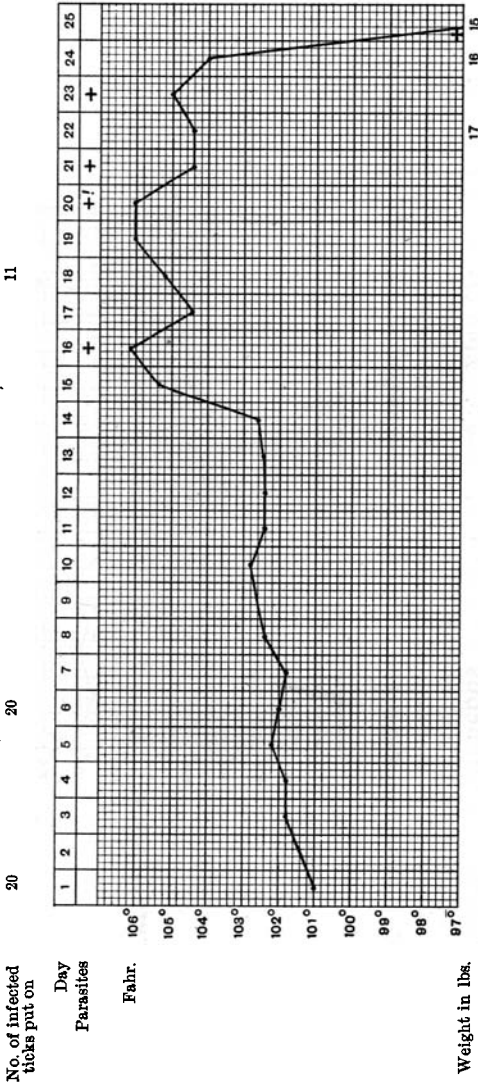


CHART II. Tick infection. Dog 5. Fox-terrier (long-haired). 26 ticks placed on dog on days 1, 7 and 12, the last near the onset of symptoms. Appetite poor 14th, food refused 19th and after, when grew rapidly weak.

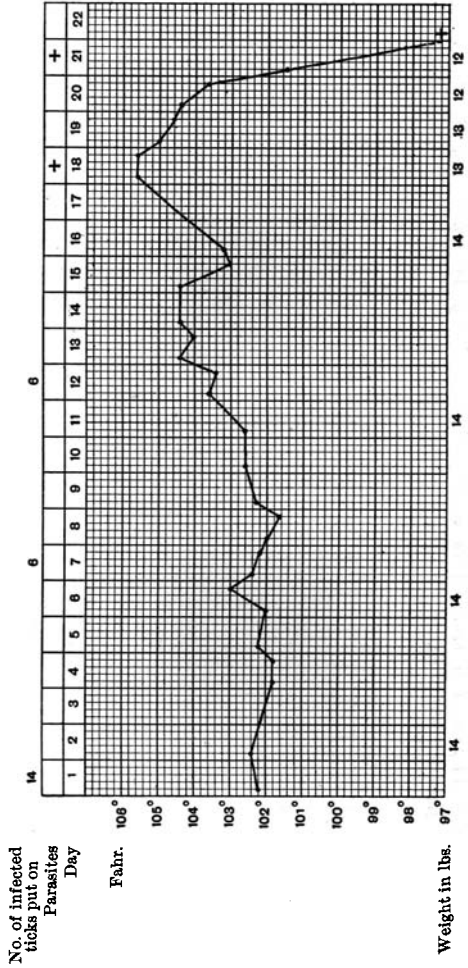


CHART III. Tick infection. Dog 7. Mongrel terrier, aged, long-haired. 22 ticks, sexes equally divided, were put on on days 1, 4 and 11; and 6 more ticks were put on on day 16 when dog showed fever. Dog ceased feeding 17th day until death 23rd day.

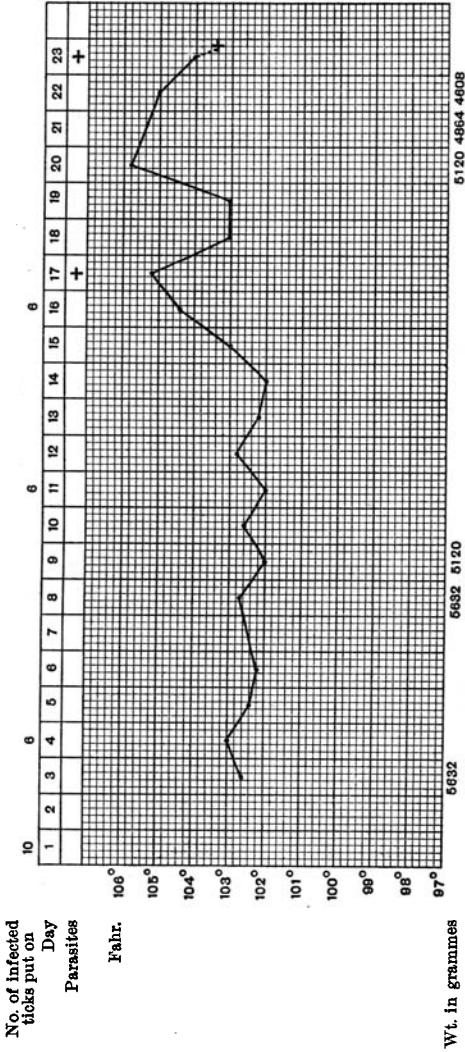


CHART IV. Infection by subcutaneous inoculation. Dog 3. Bitch (Fox- and Bull-terrier cross). Subcutaneous inoculation with blood (6 c.c.) of Dog 1 found dead on morning of 1st day. On the 10th day (after onset) 20 ticks placed on bitch so that they might become infested. 11th day bitch off her feed; 12th refused food, weak; 13th weaker, mucous membranes pale; 14th tottering, haemoglobinuria, rectal temp. 90° F., sinking.

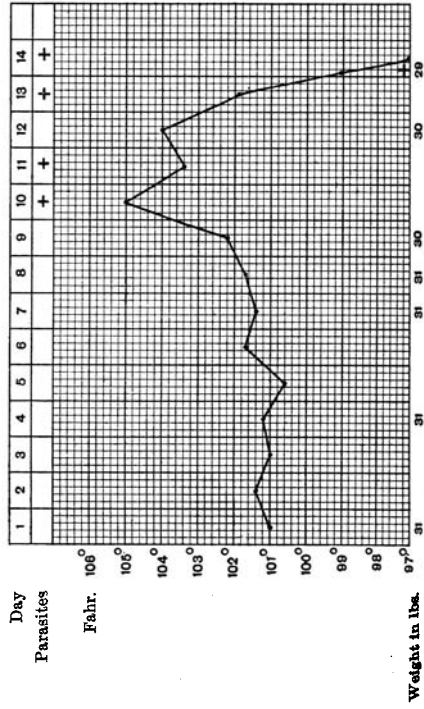


CHART V. Infection by subcutaneous inoculation. Chronic type. Mr Lounsbury's Case. Small adult male dog (No. 17) inoculated 7. 1., died 16. III. 1902. Received 7 c.c. of citrated blood (under shoulder) fresh drawn from Dog 11, recovered animal, sick in Oct. 1901. Fed on dog biscuit and condensed milk during illness. Fever began 7th day, died 62nd day of illness, no haemoglobinuria throughout. (Through an oversight the sign + was omitted in the parasite column on days 11—14, 28, 30, 31.)

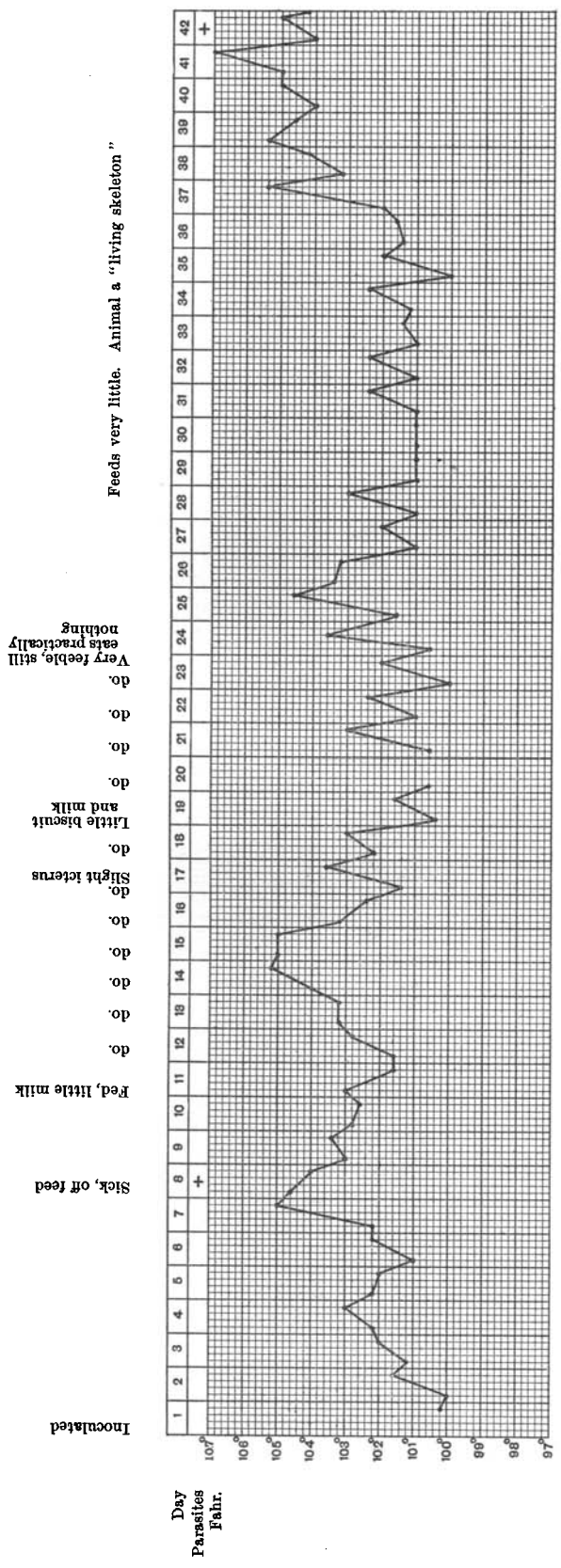
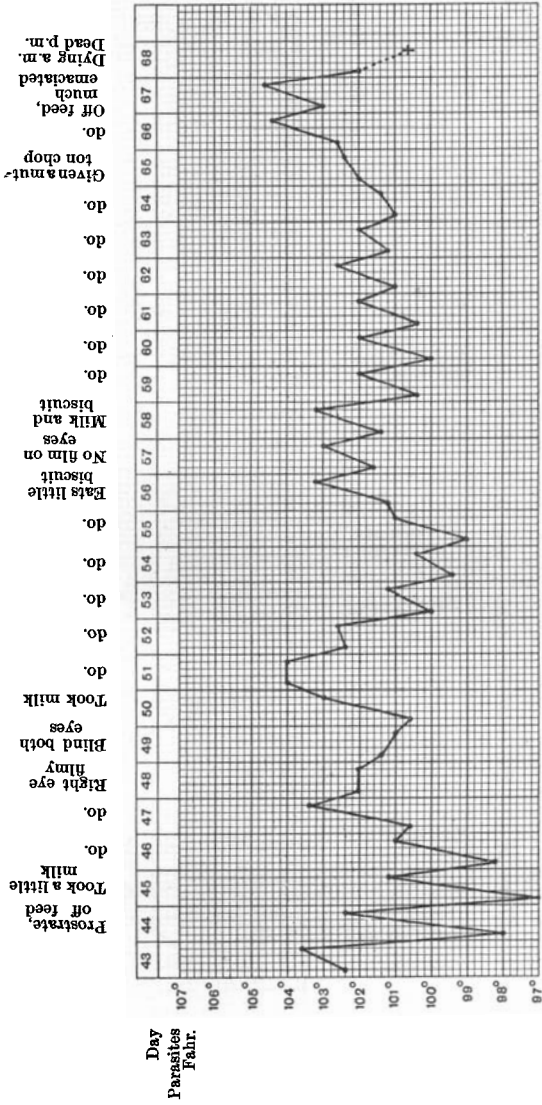


CHART V. (continued)



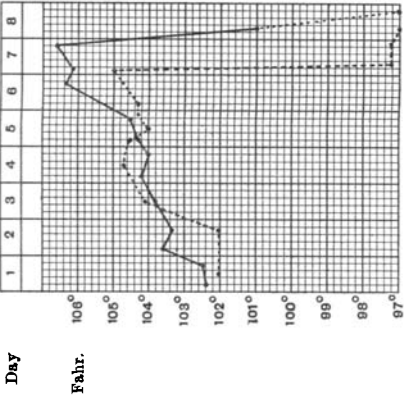
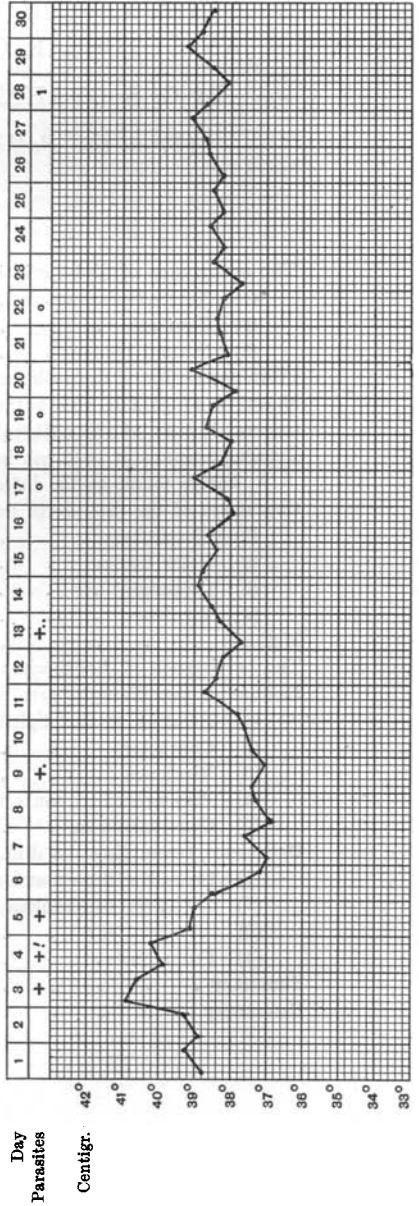


CHART VI. Infection by intravenous inoculation. Combined temperature chart for two dogs which received an intravenous inoculation of 5 c.c. of the blood of a naturally infected dog. After Robertson (1901, pp. 328, 329.) Death on the 8th day after injection.

CHART VII. Infection by intravenous inoculation. Case I. chronic, II. acute, French canine piroplasmosis. After Nocard and Motas (1902, pp. 288, 289). The blood counts etc., are given on pp. 238—239.

Case I. Parasites & fever 3rd day. Case ended in recovery.

Parasites { + moderate
 +! numerous
 +. few
 +.. very few
 | single one found
 0 nonefound



Case II. Acute, death during night of 5—6th day

