to some authorities, the presence of a fair number of eosinophiles, are favourable signs. With this last point I do not altogether agree, as I have seen numerous eosinophiles shortly before death in a good many cases.

A constantly raised temperature, even though it be comparatively slight in degree, is unfavourable, especially if no definite cause can be found for it.

When a case has got over the first attack, and has begun to improve, one hopes to see the colour index become low, if possible below 1.0, for these cases usually have a long remission. Cases whose colour index remains high are apt to go downhill again rapidly, and on slight cause.

To sum up, then, what I want specially to impress upon you is that pernicious anaemia is by no means an uncommon disease, that many cases are undiagnosed because of the unusual way in which they commence or in which they run their course, and that there is no means of diagnosis except the examination of the blood. If blood examination were made more of a routine matter in ordinary practice, as it might quite well be, we should attain a much greater accuracy in diagnosis, not only of pernicious anaemia but of the most diverse conditions; and if we practise it constantly we shall not only save ourselves many unpleasant surprises, but shall attain a more comfortable certainty about cases which now trouble us unduly.

OBSERVATIONS ON THE POISONED SPINES OF THE WEEVER FISH (*TRACHINUS DRACO*).

By H. MUIR EVANS, M.D.LOND.,
LOWESTOFT.

THE painful wounds inflicted by certain fish which are commonly caught by fishermen around our coasts are well known to all who are in any way brought in contact with the men who supply our markets with fish. However, those who most frequently suffer from the pricks from spinous fishes are trawlers, because the weever is less frequently found in the nets of herring or mackerel fishermen. Amateurs are not infrequently stung through ignorance of the dangerous nature of these fish.

There has been comparatively little written by Englishmen on this subject, and those who would read an interesting account of fish with poison organs will find the whole subject treated historically and descriptively by Dr. Bottard in a book published in 1889 entitled *Les Poissons Vénimeux*.

It is important to bear in mind that the weever fish is not poisonous, although it has a poison organ; that is to say, the fish is perfectly good to eat, and has an excellent flavour, but has the power of inflicting dangerous wounds by certain spines on its dorsal fin, and on either gill cover or operculum.

The following account of the symptoms of a weever sting is quoted by Bottard:

I have been pricked several times by *Trachinus vipera*, and I am able to give an exact account of the various phenomena the result of such an injury.

The symptoms are almost always the same. They differ only according to the depth of the puncture and the site of the wound; also according to the time of the year and the nature of the spine, whether it be dorsal or opercular.

In the month of October, 1885, I was pricked fairly deep in the middle of the palmar aspect of the right thumb. The pain was similar to that of a burn. At the end of only a few seconds all the other fingers, as well as that pricked, had become immovable, and were painfully numb. In two or three minutes the numbness had reached the forearm. The numbness seemed to follow the course of the nerves. The joints were particularly tender. At the end of from five to seven minutes the poison made itself felt on the heart, as shown by somewhat severe palpitation. I then felt cold and shivered. The wounded hand all this time was very painful and tender to the least touch. I was unable to dress it until three-quarters of an hour after the accident, when it was impossible to move the arm, and I was unable to undress myself alone. Three days later the surrounding parts were still very painful, and the tissues were swollen.

Gressin describes the symptoms as follows:

One experiences at first a very acute pain, lancinating and paralyzing, often, among nervous persons, associated with fainting. Then a sort of painful tingling attacks the wounded part, which swells up and becomes inflamed, and if one neglects to attend to it this may go on to phlegmonous inflammation and gangrene. Frequently certain general symptoms, as fever, delirium, and bilious vomiting, accompany

this state. They may last two or three hours, or make themselves felt for several days.

The fishermen attribute, with some reason, this irregularity in the effects to the amount of poison inserted, or to the time of year, the spawning season being the most dangerous.

My investigations into the poison organ of *Trachinus draco* were the result of observing the pain and suffering among all associated with the fish trade, both fishermen and packers. I must express my indebtedness, in the first place, to Mr. Borley, of the Marine Biological Association at Lowestoft, who has examined with me the anatomy, naked eye and microscopic, of the weever, and has also helped me in getting together the literature of the subject; and, in the second place, to Professor Sidney Martin, in whose laboratory and with the help of whose assistant, Dr. Stevenson, the experiments on the physiological properties of the venom took place. I must further thank Dr. Stevenson for the great interest he has taken in the subject and the time he has willingly put at my disposal.

The most recent work on the subject of the venom of *Trachinus draco* is to be found in several communications by M. A. Briot.¹ My own experiments, several years later, were made in complete ignorance of this work, and therefore have the value of being free from any bias. This literature was not referred to in so recent a work as the *Cambridge Natural History* (1905).

The methods adopted by Briot to collect sufficient poison for investigation differed to some extent from those of Phisalix. The latter macerated the gland in chloroform water and glycerine, and the fluid resulting from the maceration was used in his experiments. Briot took the venomous spines and adjacent tissues, pounded them in a mortar, and made a glycerine extract. He employed the filtered liquid for his researches, and used the opercular and dorsal spines of 350 weevers caught between the end of May and the beginning of June and 70 caught in September.

The method that I employed was, I think, more scientific and accurate. I obtained freshly-caught weevers from a buyer who was in the habit of collecting fish for the Board of Trade, and could be thoroughly trusted. These I washed under a tap, and with pledgets of absorbent gauze wiped any mucus and dirt from the opercular spines. I then laid the fish in rows and again wiped the heads with wool. With a sterilized syringe I extracted from within the sheath, by inserting the needle along the groove of the spine, as much poison as possible, obtaining, no doubt, a certain quantity of cellular elements, but I avoided those heads in which there was *post-mortem* staining of the vascular tissue of the sheath. The fluid thus obtained—say, from 100 fish—was collected in a watch-glass and placed in a desiccator (Hempel's), the rim being filled with pure sulphuric acid and the air exhausted by means of a pump. In this way, from nearly 2,000 fish I obtained several grams of dried weever poison. This, before making experiments, was finely powdered in a mortar and dissolved in either distilled water or normal saline solution. These solutions were used either with a certain quantity of solid matter suspended, or filtered, or, better, after being centrifuged, to obtain a clear fluid.

The local effects of the poison injected subcutaneously are described in the passages quoted from Bottard and Gressin. It causes mortification of the tissues, and appears to produce a local paralysis which is very evident in experiments on fish. If the poison be injected in the region of the lateral line, the fish is sharply bent away from the lesion on account of the predominant action of the lateral muscles of the opposite side. These experiments have been repeated in the laboratory of University College, London.

A gold fish (A) 4 in. in length died in one hour and a half after injecting 0.015 gram dried venom.

Another (B) died in four hours with the same dose. Seven minutes after injection it lay on its left side, curved away from the site of inoculation into the muscles of the right lateral line.

Another (C), injected with 0.01 gram, showed the next day some local swelling at the site of injection, and continued to live until ten days after the commencement of experiment. *Post-mortem* examination showed extravasated areas of blood at the site of inoculation, and great congestion of the peritoneum.

Another (D) died three days after injection of 0.01 gram, and showed a marked haemorrhagic area along the track of the needle.

Gold-fish (E), injected with 0.005 gram, died eight days after

inoculation. There was a soft puffy area with swelling where the injection took place. On incision into this there was a small cavity in the muscles with ill-defined breaking down walls. It seemed as if there was a pure necrosis of muscles. The surrounding area showed bloody engorgement, well seen on the peritoneal aspect.

In a frog 0.015 gram caused death in two and a half hours with paralytic symptoms.

In a mouse (A) 0.02 gram dried venom caused death from a dry necrosis at the site of inoculation in two days. During the day after injection there was marked paralysis of the hind quarters the injection having been made under the skin of the back. The large vessels in the neck and axilla were engorged and the sinuses of the brain were full.

Mouse (B), injected with 0.01 gram dried venom, showed weakness of the hind quarters two days after injection, and five days after a small greenish area appeared on the skin at the site of inoculation. The animal died seven days after the injection with similar local symptoms as mouse (A), but the brain sinuses were not so engorged.

In a guinea-pig 0.05 gram injected intraperitoneally caused death in three hours. After preliminary pallor and convulsions, the animal became gradually weaker and showed paralysis of the hind quarters. After two hours the heart could not be felt beating, but catchy respirations continued for another hour. *Post-mortem* there was clotting in the superficial vessels and general venous engorgement.

Briot was the first to make intravenous injections in the rabbit; 0.5 c.cm. of his solution caused death so rapidly that he had not time to finish his injection. With 0.2 or 0.1 c.cm. death was slower, taking several minutes, and, according to this observer, it was death by asphyxia. He says:

On opening the animal immediately after the pupil reflex was abolished, one found that the blood was not coagulated in the portal vein or heart. The heart continued to beat for several moments, and artificial stimuli caused it to contract for some little time. There was no doubt that death was due to respiratory failure. I was not able to say precisely whether this was due to a direct paralysis of the respiratory muscles or to an effect on the respiratory centre. But I am inclined to think that it is due to a direct action on the respiratory muscles, as much from the rapidity of the death as from the fact that when death did not result in a relatively short time (eight to ten minutes) it did not occur later.

INTRAVENOUS INJECTIONS.

The result of experiments, as illustrated by simultaneous tracings of carotid blood pressure and respiratory movements, does not altogether agree with Briot's statement that death in the case of intravenous injections is due to respiratory failure. The action appears to follow the course of that which occurs with most snake venoms, that is, a slightly preliminary rise followed by a very rapid and deep fall in blood pressure.

Experiment 1.—Rabbit, weight 1,900 grams; anaesthetic, ether. After an injection of 0.05 gram dried venom there was a fall in blood pressure lasting 45 seconds. Then, after 20 seconds of irregular low pressure, the pressure gradually rose. After an interval of 7 minutes, during which the cardiac pulsations had steadied down to a normal rate, a further 0.05 gram of dried venom was injected into the femoral vein. Immediately the blood pressure fell rapidly for 20 seconds with almost imperceptible cardiac pulsations, and in 2½ minutes a further fall took place, immediately preceding complete cardiac failure and death.

Experiment 2.—Cat, weight 3,400 grams; 0.1 gram dried venom; ether. Immediately after injection marked fall of blood pressure, and 18 slow, powerful beats in 10 seconds, that is, 104 per minute (normal, 192 per minute). This was followed by a rise of blood pressure in 10 seconds to normal, and then another gradual fall with marked slowing of the beats; 33 seconds from first injection the cannula was washed out with 0.86 per cent. sodium chloride solution and the cardiac contractions became gradually small and the pressure continuously fell, so that 2 minutes from the commencement of experiment the heart had ceased to beat. During this time the respiration, although affected so as to become at first shallow and then every fifteenth breath a deep inspiration, continued, and there were nine deep inspirations during the period of nearly 2 minutes which elapsed between the last cardiac beat and the last respiration. This tracing shows in a very convincing way the typical fall of blood pressure the result of an intravenous injection of a venom, and controverts the theory that the cause of death is in the case of weever poison always due to respiratory failure.

Experiment 3.—Cat, weight 3,300 grams. Ether, injected with 0.1 gram dried venom dissolved in 5 c.cm. of 86 per cent. sodium chloride solution; the resulting solution was then centrifugalized and the clear fluid injected into the femoral vein. For 20 seconds from commencement of the injection there was a slight fall in blood pressure, followed during the next 15 seconds by a slight rise. Then, 40 seconds from the commencement of experiment, there was a gradual and deep fall of pressure, reaching its lowest point 2 minutes from the

commencement. During this fall the cardiac pulsations became less strong, the rate being 150 per minute as against the normal of 168 per minute. During the first 30 seconds the respirations were 8 per minute as against a normal of 9 per minute; they gradually quickened, until at the end of the second minute they amounted to from 14 to 12 per minute. Four minutes from the commencement the respirations had slowed down to 6 per minute, with cardiac pulsations stronger, 96 per minute. At the eighth minute the respiration became very slow, while during the ninth minute respirations were very irregular—4 per minute, with clonic twitching of abdominal and leg muscles. During tenth and eleventh minutes there were only 2 respirations per minute, with clonic twitchings; in fact, death almost resulted from respiratory failure. During the fifteenth minute the animal began to recover, 6 respirations per minute and heart 126. The experiment was then completed.

In this case death did not result from the immediate action on the cardiac system, but the later effects on the respiration nearly ended in death.

It would no doubt have been interesting, from a scientific standpoint, to have gone further and repeated these experiments after division of the nerves; but, inasmuch as the poison is not, in practice, ever injected in sufficient quantity to cause death either by heart failure or respiratory failure, it seemed more important to study the local effects and the blood changes which not infrequently result in man in phlegmonous inflammation and septicæmic conditions.

The neurotoxic effects of the poison have not been fully worked out at present, as I have been unable to spare the time away from practice, but I have devoted what time has been available to experiments on the hæmolytic properties of the venom.

HAEMOLYTIC ACTION OF WEEVER POISON.

This had been studied up to a certain point by M. Briot, but only as regards the blood of warm-blooded animals.

It has been established that snake venom only produces hæmolysis in certain animals when certain components of serum are also present. If red corpuscles be washed in normal saline and venom added, no solution occurs; but if normal serum heated to 62° C. be added, hæmolysis occurs. It is necessary to heat the serum to prevent an anti-hæmolytic present at low temperatures from protecting the erythrocytes against the solvent action of the venom. These are the views held by the school of Calmette. Briot has repeated Calmette's experiments on snake venom with weever poison. In a series of test tubes he put a drop of washed corpuscles in suspension in 2 c.cm. of normal saline; he then added normal serum heated to 60° C. for one hour, and variable doses of his solution of weever venom. Complete hæmolysis took place in 1½ hours.

Two controls were made, one without the venom and one without the heated normal serum; in neither tube did solution take place, nor did it take place when unheated serum was added. Briot also observed that the hæmolytic effect of the venom remained intact after heating for an hour at 75° C., and that even after heating for twenty minutes at 100° C. dissolution still took place, though slowly.

These observations were limited to the blood of certain warm-blooded animals. I have carried the investigations further by experimenting with the blood of the roach and perch. I took 2 minims of a solution of equal parts of fresh weever venom and normal saline, and added it to 2 c.cm. of normal saline in which were suspended 5 drops of the nucleated red corpuscles of roach which had been washed and centrifugalized three times with saline. Solution began to occur in from one hour to two hours, and no formed elements remained a few hours after commencing the experiment; the red corpuscles remaining as a sediment in the control tube with white clear supernatant fluid, while in the tube in which the weever venom was added no sediment remained, and the fluid was deep red. This experiment has been frequently repeated, and always with the same result. The same result was obtained with perch blood.

The explanation of this apparent difference in result is made clear by the work of Preston Kyes on the mode of action of cobra venom. It had been established by Flexner and Noguchi that snake venom is made up of a number of substances acting after the manner of amboceptors (or immune bodies) which are activated by certain complements of the serum. In fact, snake venom does

not act as a simple poison after the manner of toxins but in a complex manner, being identical in action with the haemolysins of blood serum, as this has been conceived by Ehrlich and Morgenroth. Kyes then experimented with cobra venom in the blood of different animals, and

solution pipetted off. This was repeated three times, and finally a 5 per cent. emulsion of red corpuscles was made in 0.85 per cent. salt solution. Either 2 c.cm. or 1 c.cm. of this solution was put in a series of tubes, the poison added in varying doses and the mixtures put into the incubator for

Fig. 1.—The weever fish (*Trachinus draco*). D, Dorsal spine; O, opercular spine.

found that there were two groups of blood cells: (1) Those dissolved by cobra venom; (2) those only affected by venom after the addition of other substances, complements, etc. He then showed that the red corpuscles in the first group were only acted upon by the venom

two or more hours at 37° C. They were then allowed to settle in a cool place for several hours.

With pigeon blood one drop of a 50 per cent. solution of fresh weever poison was sufficient in two hours to produce marked haemolysis. The poison solution used was

Fig. 2.—Tracing of carotid blood pressure and respiratory movements. Weever poison, 0.1 gram dried venom. Cat, weight 3,400 grams. Ether. A, Injection, lasting 14 secs; B, 36 secs. from the beginning of the injection. Cannula washed out with normal saline.

because they themselves contained endocomplements which allowed the amboceptor of the venom to act on the cell.

It is not for me to discuss the experiments which proved this point, but they are very convincing, and there seems no doubt that certain blood cells themselves contain complement-like substances or endocomplements. My own experiments with different bloods with weever poison at any rate support his views, and we see the venom-producing haemolysis in roach and perch blood without the intervention of complements of added serum.

It seemed important, having made these preliminary investigations with the blood of fish, to carry them further into the realm of warm-blooded animals. The blood of pigeon, guinea-pig, sheep, ox, horse, and man, was used. The more exact methods of haemolytic experiments, as employed by Ehrlich's school and fully described in an article by Morgenroth in Ehrlich's volume of collected studies on immunity, were used in these investigations.

The blood, having been defibrinated, was centrifugalized to remove the serum, which was carefully pipetted off; 10 c.cm. of normal saline was added to 0.5 c.cm. of corpuscles, and well mixed, centrifugalized, and the clear

obtained by extracting the venom with a needle and syringe previously sterilized; in order to get all the milky fluid from the syringe, an equal quantity of salt solution was added to wash it out, so that a 50 per cent. solution of fresh poison was obtained. This was kept in a cellar on a block of ice in the dark, and its virulence remained intact. From a great number of experiments I found that uniform results were obtained if the poison was removed from freshly-caught fish, and extracted at once. It appears, as the fishermen state, that the poison loses its effects soon after death. With one specimen of venom I got most contradictory results, due to my using a batch of 100 fish which were not quite fresh, although they had been kept on ice.

The effect on birds is interesting, inasmuch as a smacksman has related to me how he has seen gulls fall into the sea as if struck dead when he has thrown live weevers overboard to them.

With guinea-pig corpuscles, two drops of 50 per cent. weever poison were added to one tube containing 2 c.cm.

emulsion, and to two other tubes the same quantity of weever poison, with in one tube some normal serum, and in another serum heated for an hour at 62° C. In all three tubes marked haemolysis took place; in the control,

Fig. 3.—2 c.cm. of 5 per cent. emulsion guinea-pig corpuscles washed three times in normal saline; i, control; ii, two drops 50 per cent. weever venom added; complete haemolysis.

none. We see here, again, the presence of endocomplements in the guinea-pig corpuscles, and we have a slightly greater action with the presence of heated serum than with unheated serum.

A similar experiment was made with sheep's blood. In this case, with 5 per cent. emulsion and two drops of 50 per cent. weever poison, there was advanced haemolysis, and with the same quantity of poison, with both normal serum and serum heated for an hour at 62° C., there was complete haemolysis after two hours in the incubator at 37° C. We have here a very marked example of the amboceptor of weever poison combining with the endocomplement of the sheep corpuscles, resulting in rapid haemolysis. The increased haemolysis with the added serum was not affected by heating to 62° C.

With human blood we also find that there is haemolysis of the washed corpuscles without the addition of serum. But unless the venom is quite fresh 1 drop of the 50 per cent. solution is not sufficient to produce haemolysis, although 2 drops suffice. It seemed important, in the light of Briot's investigations previously referred to, to be quite sure of this. I have made seven separate experiments with human blood. The following experiment was very conclusive:

Six tubes, each containing 1 c.cm. of 5 per cent. washed human corpuscles, were numbered 0, 1, 2, 3, 4, and 5; 0 was the control.

1	contained 1 drop of 50 per cent. weever poison.
2	" 2 drops " " "
3	" 3 " " " "
4	" 4 " " " "
5	" 2 drops + 0.1 c.cm. normal serum.
- 0 underwent no change after two hours at 37° C.
 1 showed medium haemolysis with dark red sediment.
 2 showed well-marked haemolysis with dark pink sediment.
 3 ditto.
 4 showed very marked haemolysis, almost complete, the sediment being merely a faint pink.
 5 showed complete haemolysis; sediment yellow.

There seems no doubt that trachinus venom can dissolve human corpuscles without serum, and that normal unheated serum does not delay, but rather increases, the solution. Experiments were made to endeavour to activate venom insufficient in quantity to dissolve the human corpuscles by means of sheep serum and ox serum. A venom of which 1 drop did not dissolve washed human corpuscles, was mixed with 0.3 c.cm. sheep serum, and a control was made with sheep serum only. The result was that with 1 drop of weever poison in 0.3 c.cm. sheep serum there was marked haemolysis; the control showed agglutination but very faint haemolysis.

A similar experiment was made with ox serum, 0.1 c.cm. of which was used for activation. There was complete haemolysis with ox serum and 1 drop of weever poison, and slight haemolysis in ox serum and blood alone. In both the above experiments there were two other controls with the corpuscles and weever poison, and no haemolysis occurred. A further experiment was made with ox serum heated for an hour to 62° C.; with 1 drop weever poison the control gave no result, the unheated serum slight haemolysis, and the heated serum *nil*. With 2 drops the control had marked haemolysis; unheated serum, medium; and heated, slight. Apparently the heated serum delayed haemolysis.

I have made experiments with the idea of activating the venom towards human blood by means of lecithin, and have succeeded on two occasions; but as other experiments have not given uniform results, I am continuing this investigation with lecithin recommended by Preston Kyes.

Horse's blood gives the haemolytic reaction with washed corpuscles alone. Seven tubes were filled with 1 c.cm. of 5 per cent. emulsion. 1 w.p. = 1 drop 50 per cent. weever poison.

With 1 w.p.	slight haemolysis.
With 2 "	well marked haemolysis.
With 2 "	+ 0.05 c.cm. normal serum, strong haemolysis.
With 2 "	+ serum heated for half an hour at 62° C., slight.
- The controls were all negative.
 Ox blood gave similar results:
 With 2 w.p. well marked haemolysis.
 With 2 " + 0.5 c.cm. $\frac{1}{2}$ normal serum, well marked haemolysis.
 With 2 " + 0.5 c.cm. $\frac{1}{2}$ heated serum, strong haemolysis.

The results of these experiments establish (1) that weever venom has a haemolytic property capable of dissolving washed corpuscles of all the bloods so far experimented on without the addition of serums. (2) That the addition of the serum of the same animal does not inhibit, but

rather increases the activity of the venom. (3) That in most cases heating the serum for an hour to 62° C. diminishes its activating power. (4) That minimal doses of the venom can be activated by the serum of other animals in the case of man, or the corpuscles are rendered more susceptible to the poison. (5) That probably the poison is an amboceptor, which unites with the endocomplements of the blood cells.

REFERENCE.

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PRELIMINARY NOTE ON THE DEVELOPMENT OF PIROPLASMA CANIS IN THE TICK.

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THE only work which has hitherto shed any light upon the development of the piroplasmata in ticks is that of Koch on the piroplasmata of cattle in East Africa, and that of his colleague Kliene on the development *in vitro* of *Piroplasma canis*. But these observations still leave us very much in the dark regarding the cycle of development of piroplasma in the tick, and the life-history of the parasite in the main is still unknown. I have been fortunate

EXPLANATION OF FIGURES.

- Fig. 1.—Endocorpuseular forms. $\times 2,000$.
 Fig. 2.—Free form in gut of tick. $\times 2,000$.
 Figs. 3, 4, and 5.—Developmental forms in the gut. $\times 2,000$.
 Fig. 6.—Conjugation. $\times 2,000$.
 Fig. 7.—Developing club-shaped bodies from the gut. $\times 2,000$.
 Fig. 8. Fully developed club-shaped body. $\times 2,000$.
 Fig. 9.—Immature club-shaped bodies from the tissues. Living preparation. $\times 2,000$.
 Figs. 10 and 11.—Living club-shaped bodies from the ovary. $\times 2,000$.
 Fig. 12.—Club-shaped body showing the disc structure on the flat. $\times 2,000$.
 Figs. 13 and 14.—Club-shaped bodies from the tissues of nymphs of the second generation. $\times 2,000$.
 Fig. 15.—Parent forms (oocyst). $\times 2,000$.
 Fig. 16.—Binary fission of a parent form. $\times 2,000$.
 Fig. 17.—A group of "sporoblasts" from the salivary gland of nymph. $\times 2,000$.
 Fig. 18.—Two of the same. $\times 4,000$.
 Fig. 19.—A portion of a salivary cell of unfed nymph of second generation showing a few of the parasites embedded in its substance. $\times 500$.
 Fig. 20.—Division of the chromatin into two "sporoblasts." $\times 2,000$.
 Fig. 21.—Division of sporoblast into small forms. $\times 2,000$.
 Fig. 22.—Free small forms (sporozoites?). $\times 2,000$.
 Fig. 23.—A group of sporoblasts probably derived from a single parent form lying in connexion with the nucleus of a tissue cell and showing signs of division into sporozoites. $\times 2,000$.
 Fig. 24.—Sporozoites (?) in a cell of developing salivary acinus. $\times 300$.
 Fig. 25.—Large body with stellate chromatin lying in cell of limb of unhatched adult. $\times 2,000$.
 Fig. 26.—Sporulation of this form. $\times 2,000$.

enough, however, to have been able to trace in its entirety the life-cycle of *Piroplasma canis*, and presumably incidentally that of the other piroplasmata.

Canine piroplasmosis is described from Europe and Africa, but I have found it also endemic among the native dogs of India. Though it has been universally assumed on analogy that the disease is carried by ticks, the actual facts regarding its transmission have only been worked out with respect to the disease in the Cape, and the observations of Lounsbury and Nuttall yield the only precise information we have on the subject. In South Africa the disease is transmitted by the tick *Haemaphysalis leachi* (Audouin), the adults of the second generation only being infective. In Madras *Haemaphysalis leachi* is not found, and a preliminary investigation was undertaken to determine what species acted as the carrier here, and at what stages it took in and transmitted infection. These experiments will shortly be published at length; it is only necessary at present to note that the tick generally concerned is *Rhipicephalus sanguineus* (Latreille), and that infected adults give rise to infective progeny. The larvae hatched from the eggs of an infected mother are apparently unable to transmit infection, but the nymphs and adults reared from these do so. Though infection can pass through the egg, it can also be taken in during the nymphal engorgement and be transmitted later by the adult stage, a fact, I believe, Mr. Lounsbury had previously arrived at in regard to certain of the Cape piroplasmata