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.—Endocorpuscular 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

There is in this case no question of mere direct transmission, for the same cycle of development of the parasite occurs whether infection is transmitted hereditarily or not.

As already stated, the life-cycle has been followed in its entirety, though no doubt many details are still to be added and additional facts accumulated. In the present account I intend only to indicate the appearances seen, reserving a fuller description to a later date. In the gut of adult ticks and also in that of nymphs fed on infected dogs one sees a varying number of free forms resembling those seen in the blood. These in many cases have enlarged considerably (Figs. 3 and 4) and the chromatin has become more distinct. Certain forms are seen with processes, but I do not find this is a necessary stage of development, nor have the forms seen by me ever exhibited the irregularities in the chromatin figured by Koch and Kliene. The result of the initial changes leads to the formation of the very characteristic bodies delineated in Fig. 5. These measure about 3.5μ in

of the tail. Many of them possess at the anterior extremity a disc-like structure provided with distinct cusps (Figs. 8 and 12). Occasionally this structure is seen on the flat, when a central and four or five peripheral processes can often be made out. In the tissues of nymphs from infected mothers one finds these forms to be present, though they are as a rule swollen and otherwise altered. In some cases what is undoubtedly the altered disc structure can still be seen. In a great many instances the tail is flexed upon the body, and the parasite is apparently becoming the form we are about to describe.

In the tissues of the unfed nymphs (examination of infected larvae since my discovery has not yet been possible) bred from infected mothers there are, especially in the cells of the salivary glands, numerous, often swarms of, comparatively large, round, oval, or even pear-shaped forms having a general resemblance to piroplasma in the blood, except that they are much larger, and that the chromatin is arranged in a very characteristic way. These

diameter, and are more or less globular, though they very frequently show two or three blunt processes at one side. Numbers of these bodies are at certain times found in couples, the appearances suggesting conjugation rather than fission. As a final result of development in the gut large club-shaped bodies are formed, which may be seen in every stage of development, from those shown in Fig. 7 to the vermicule-like forms shown in Figs. 8 to 12. These are the bodies described by Koch in the egg, though, as a matter of fact, they are not by any means confined to this situation. In the case of the adult they leave the gut to enter the ova, and may be seen in fresh preparations in numbers, penetrating the walls of the oviducts and ovary. In the nymph, whether hereditarily or directly infected, they are found in the salivary gland and other tissues. Observed in the living condition, these bodies are seen to be endowed with pretty active movements. In what appear to be the less developed forms the movements are euglenoid in character, but the more matured forms have a fixed body shape, and exhibit, in the main, side-to-side movements

bodies, 4μ or more in diameter, are not very readily stained, so that they have a peculiar light blue transparent appearance when coloured by Giemsa. The most striking feature is the chromatin, which shows in a very marked degree an appearance very like that recently described by Nuttall in the parasite in the blood, and which I have also noted in the Madras parasite. Passing out from a dense mass of chromatin there is an area of loose chromatin reticulum, or even of separate woolly strands (Fig. 17). These forms are aggregated especially in the large cells of the salivary glands (Fig. 19). In heavily infected ticks hundreds of parasites are often present in a single cell. It is probably such a cell which Koch has figured, and which he erroneously thinks to be a cell of the gut. The bodies are the result of fission of a similar but somewhat large parent form, each of which is derived directly from a club-shaped body. Single parent forms are often seen embedded in the protoplasm of young cells close to the nucleus, and groups, clearly the result of fission, are seen in the same position.

These bodies, packed as they were in thousands in the

salivary cells of the nymph, gave rise to the belief that this was the final stage of development. Such, however, is not the case, for in nymphs undergoing changes into the adult further developmental stages are seen. It is probable, however, that the bodies I have already described can give rise to infection in the dog, since the nymph is highly infective, the disease being induced with an incubation period sometimes so short as three and a half days. The body of the adult tick is in great part formed independently of the original tissues of the gorged and the inactive nymph, and the salivary glands of the nymph are soon lost among the active tissue changes which go on at this time. If squash preparation be made of the already partly recognizable tissues of the unhatched adult, the groups of fission forms will be found to have become disseminated among these occurring even in situations which will eventually form the limbs. The changes at this time are very striking. The chromatin of each body becomes split up and arranges itself peripherally, often mainly upon one side (Fig. 20). The strand-like processes of chromatin already referred to are also dragged about, and portions are seen attached to, or in attendance upon, each of the denser masses. This arrangement is the prelude to the division of the bodies into from three to ten small forms resembling very exactly piroplasma in the blood (Fig. 22). Groups of as many as fifty of these small forms are to be seen, usually lying within some kind of cell, and it is probable that these represent the progeny of a single parent form (Fig. 24). Many such groups are found in tissue which will eventually become the salivary glands of the adult. The fate of the bodies in cells other than those eventually forming the salivary gland is doubtful, but I have seen single bodies embedded in cells and they often show what appear to be amoeboid processes, so that it is quite possible such parasites eventually reach the gland.

This appears to be the usual cycle of development in the tick, but I have reason to think that another cycle leading to a similar result exists. At present observations are too imperfect to enable me to speak with precision, but in the cells of the tissue of the developing adult one sees large forms with a stellate arrangement of the chromatin, very like certain forms sometimes seen in the gut, which do not appear to enter into the formation of the club-shaped motile vermicules. These bodies increase greatly in size, measuring in some cases so much as 15μ , and they then break up into a rosette of from 50 to 100 small forms, such as result from the previously-described cycle. It is premature to discuss yet the full significance of the changes here described, but it appears at present reasonable to conclude that a true conjugation takes place in the gut, and that the travelling club-shaped bodies are the ookinete. The bodies formed in the salivary gland of the nymph are possibly homologous with the sporoblasts of the malarial zygote, and the further subdivision of these into sporozoites would account for the small forms with a simple chromatin mass. That the salivary glands of the nymph are stocked with one type of body and those of the adult with another is a very singular fact.

CRAIG COLONY FOR EPILEPTICS.—According to the thirteenth annual report of the Craig Colony for Epileptics, recently issued, the colony occupies 1,895 acres of land with 76 houses; 6 more, providing accommodation for 200 additional patients, are being erected. The number of patients in the colony is nearly 1,300; when the new buildings are completed the census will be approximately 1,500. There are now about 1,000 epileptics in New York State who cannot be received because there is no room. A considerable amount of surgical work is done at the colony, as is shown by the fact that anaesthetics for surgical purposes were given 172 times during the year. The cost of maintenance last year was 141.38 dollars for each patient. The establishment of a second colony for epileptics nearer New York is said to be contemplated.

LEPER POLITICIANS.—The lepers on the Island of Molokai have, according to *American Medicine*, for the first time in the history of the settlement, a real election. Under the new County Act the settlement is created a county, but all the functions of government are given to the Board of Health, except the election of a deputy sheriff. A large number of candidates for this office has arisen, and a campaign which surpasses anything ever known anywhere else is in progress. The people have absolutely nothing to do but take part in political campaigns. They are housed and fed at public expense, and a political campaign affords them excitement and relief from monotony.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL.

THE TREATMENT OF TETANUS.

On August 18th, 1906, a young man, aged 19, was admitted to the Faversham Cottage Hospital suffering from a gunshot wound of the left leg, inflicted fourteen days previously, and situated on the outer side of the left leg just above the external malleolus. About 5 in. long and 3 in. wide, it was in a foul, sloughy condition. The fibula was bare in the bottom of the wound. There was slight difficulty in opening his mouth, but no rigidity in the limbs.

During the week following admission the patient rapidly became worse. The spasm of the jaw and the muscles of the neck became intensified; there was well-marked opisthotonos and risus sardonius. The temperature varied between 101° and 102° ; there was great restlessness and all the symptoms of a severe attack of tetanus. The spasms were induced by the slightest touch or attempt to take food, and became more frequent in number. On August 29th I trephined him over the left motor area and injected under the dura mater 20 c.cm. of antitetanic serum, obtained from Messrs. Burroughs, Wellcome and Co.

The piece of bone removed by the trephine was replaced and a cyanide gauze dressing applied. The wound healed by first intention, and the sutures were removed on the tenth day.

The day after the operation the patient was markedly better; the spasms were less intense, and his general appearance showed great improvement. He continued to improve, and was discharged from the hospital on October 10th in a convalescent condition.

Drug treatment consisted of bromide and chloral in full doses both before and after the operation, but I attribute his recovery solely to the effect of the antitoxin.

Faversham. F. W. GANGE, M.D. BRUX.

PARTIAL DISLOCATION OF THE LARYNX.

THE following accident, from its nature and the method of its production, seems to me to be sufficiently interesting and unusual to warrant description:

H. L., a veterinary surgeon, while driving in a high dog-cart, on July 25th, 1906, along a narrow street, failed to notice the presence of a double clothes-line stretched tightly across the road. The first intimation of its existence—that he received was a sense of being struck forcibly across the throat, and thrown very violently against the back of the gig. He at once experienced a terrifying feeling of suffocation, heard something loudly “click” in his neck, and became very dizzy, and almost blind in his left eye. Having a very definite surmise of human anatomy, deduced from his own professional knowledge, and a wonderful presence of mind, he diagnosed his condition, and, putting his hand on his neck, felt the thyroid cartilage, and particularly the pomum, displaced from the median line at least 1 in. to $1\frac{1}{2}$ in. to the left. This displacement he at once attempted to rectify, with some success, and the replacement was again accompanied by a very definite “click.” He was driven home four miles, and I saw him about an hour and a half later. At that time he had greatly recovered from his shock. I examined his larynx very carefully. There was no doubt that the pomum was displaced at least $\frac{1}{2}$ in. to the left of the mid-line. The right ala of the thyroid cartilage was felt to be indented though not fractured. The hyoid bone was in position. The cricoid cartilage and upper part of the trachea were carried slightly to the left with the thyroid cartilage. With the laryngoscope I could not obtain a good view of the vocal cords, owing to an apparent bulging towards the left of the upper part of the larynx. By grasping the thyroid cartilage firmly in the hand it could easily be brought back into position, but on doing this a peculiar crepitation could be felt and heard, and seemed to be caused by the grating of the cartilage over torn ligaments, and to be produced in the space situated between the larynx and oesophagus. His voice was but little affected, there was no spitting of blood, no emphysema, no swelling, no oedema. Being an active man of strong will he insisted on continuing his work, and was very averse