AFRICAN TICK FEVER

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by

RONALD RODERICK MURRAY, M.B., Ch.B.
F.R.C.S.(Ed.), D.T.M.(Liv.)
INTRODUCTION.

The importance of a thorough knowledge of the various forms of relapsing fever occurring in tropical countries cannot be too greatly recognized. Owing to their wide geographical distribution, the diversity and number of the transmitting agents, and often, the severity of the clinical features, resulting, not infrequently, if the disease be not early recognized, in distressing sequelae, medical men resident in the tropics, especially in tropical Africa should be thoroughly conversant with the various types of the disease, its prophylaxis and treatment. In view of our occupation of German East Africa this disease assumes additional importance - for this region of Africa abounds in the tick which conveys the spirochaete of relapsing fever. In some districts Koch found that 50% of the ticks were infected.

Various names are given by the Natives to the tick, in different countries of Africa. The great travellers, Livingstone and Kirk, in the year 1865 described a disease called "Carrapata" with symptoms resembling those of relapsing fever, which was endemic in the Zambesi basin. Mesnil has in more recent/
recent times described this disease in the same locality. The disease has also been described by Ross and Milne in Uganda and British East Africa, by Dutton and Todd in the Belgian Congo, by Koch in German East Africa, by Wellman in Angola and by Drake-Brockman in Somaliland. Outside Africa, a relapsing fever produced by the bites of ticks has been described by Franco Robledo in Colombia, in 1865, by Büldow in the Andes, and more recently, Wosnissenky's and Dschunkowsky's observations seem to make it clear that the spirochaete causing the disease in Persia is conveyed by a tick of the genus Ornithodoros. Alleman in Mexico has also described a form of relapsing fever, a tick being suspected of conveying the infection, and the disease has also been met with in North Africa, South Africa and Madagascar. Lastly, I have to record my own cases which were met with in Northern Rhodesia.

Since the discovery of the spirochaete in the blood, and the fact of its conveyance by a tick, by Nabarro, also by Ross and Milne in 1903, much work has been done on the subject of the etiology of the disease, chiefly by Breinl and Kinghorn at Runcorn, Koch in German East Africa, also by Leishman and Hindle who studied the life history of the spirochaete/
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spirochaete in the gut of the tick. Balfour has studied the phenomenon of "granule shedding" in liver puncture and dark field method in infected fowls. Ross and Milne, Dutton and Todd, proved that *Ornithodoros moubata* conveyed the disease. This placing of the etiology of the disease on a sound basis has resulted in great benefits in the direction of prophylaxis and treatment.

My own experience of the disease has been gained in treating cases, both European and Native, during a period of residence in Northern Rhodesia of six and a half years. During this time I travelled over a very wide stretch of country, at first being attached to the Anglo-Belgian Boundary Commission operating on the borders south of Lake Bangweulu and later, between Lakes Mweru and Tanganyika. Here we crossed some of the old caravan routes where many of the villages were infested with Argasine ticks and I was privileged to treat several cases of the disease. Later I was stationed at Kawambwa in North East Rhodesia, near the Congo border where I found the spirochaete in the blood in several other cases and was able to get a history of a tick bite in most of the cases; on more than one occasion seeing the very tick which had bitten the patient and caused the disease.
disease. Finally, at Abercorn, in the early days of the war in 1914, I met with several cases which had been contracted in the native gaol. The vast majority of my cases were Natives, of whom I have records of thirty-nine cases, but I have also treated the disease in six Europeans.

The scope of the present thesis is to give an account from personal experience, of the clinical types of tick fever as it occurs in both Europeans and Natives. I shall also refer to the etiology, the prophylaxis and treatment.
HISTORICAL OUTLINE.

The two great explorers, Livingstone and Sir John Kirk, both described the disease in the year 1865. Livingstone was the first to describe a "human tick disease" in Portuguese East Africa endemic in the Zambesi basin. From time immemorial the natives of certain countries of Africa have attributed to the bite of a tick, a sickness of longer or shorter duration which may end in death. In August 1903, Nabarro was the first to observe a spirochoete in the blood of human beings in Uganda. In 1904 Ross and Milne, also Cook, in Uganda, and Dutton and Todd in the Congo Free State, found the cause of Tick Fever to be a spirochoete, and they showed that it was introduced into the blood of the patient by the bite of a tick, Ornithodoros moubata. Previously to this, Marchoux and Salimbeni had experimentally demonstrated the transmission of spirilla by ticks to fowls, and further, that a tick could transmit the disease for five months. Following on the work of Marchoux and Salimbeni, the researches of the above-mentioned African observers, whose attention was drawn to this tick by the well-known evil reputation which it enjoyed/
enjoyed among the natives, who attributed their illness to its bites, stimulated the further study of the disease by other workers. Koch, in 1905, found that ticks were infective to the third generation. Markham Carter, in 1907, and Kleine and Eckard in 1913 have since corroborated Koch's work on the spirochoetes in the tick eggs. In 1906 the disease was studied in America by Novy and Knapp, who gave to the organism the specific name Spirochaeta duttoni. This specific name was also given to the parasite in the same year, 1906, by Breinl and Kinghorn working in Liverpool, in honour of Dr Dutton who died of the disease while working at Liverpool. These observers differentiated the African spirochoete from S. recurrentis (obermeieri), which causes European relapsing fever, by differences in its clinical features, animal reactions and relative active immunity. They found that a considerable number of animals, especially rabbits and guinea-pigs, which were susceptible to the African spirochoete were refractory to the organism of relapsing fever; that each of S. duttoni and S. recurrentis confers a relative active immunity against itself but not against the other; and that a more severe illness was produced by S. duttoni in monkeys. Breinl and Kinghorn also showed/
showed that S. duttoni passed through the placenta from the circulation of the mother to that of the foetus. They also showed that splenectomy had no effect on the course of the disease. As the result of their investigations, Dutton and Todd stated that the transmission of the spirochaetes by ticks was not merely mechanical, but that some developmental process took place in the tick. The passage of the spirochaetes from the alimentary canal of the ticks to the ovary and eggs suggests that the organism is protozoal. Breinl stated that just before the crisis in African tick fever, S. duttoni becomes thinner in the spleen and bone-marrow and rolls up into skein-like forms, which are surrounded by a thin "cyst" wall (probably the periplast). Inside the cyst the spirochaete breaks up into granules.

Balfour and Sambon have described similar forms, breaking up into granules, inside the red blood cells of Soudanese fowls in the case of S. granulosa (variety of S. gallinarum). The granule phase is an essential one in the case of the invertebrate transmitter. Workers have shown that when the tick takes in the spirochaetes from the blood of its host granules are given off from these spirochaetes. These granules are coccoid in form, and are found in/
in various organs, especially in the Malpighian tubules, and are termed "infective granules". Leishman has shown that in ticks which were kept at a low temperature, $21^\circ - 24^\circ$ C., spirochoetes were not seen except in the gut, and even these disappeared after about 10 days from time of feeding; but in ticks incubated at $35^\circ - 37^\circ$ C., for two or three days, spirochoetes reappeared in its various organs, presumably having been formed by the transformation of the granules. Leishman and Balfour regard these granules as being the infecting phase, but the French workers, Marchoux and Couvy, say that these "infective granules" can be found in almost all ticks whether fed on an infected host or not. Of late years, the spirochoetes causing relapsing fever in man have been differentiated into various species. The morphology of all of them being so similar, it is difficult thus to differentiate them, and the following tests are used:—

1. Cross immunity experiments.

2. Varying receptivity of various laboratory animals.

3. Agglutination tests.

Darling, working in the Panama zone, has shown that the cross-immunity experiments are not entirely reliable,
reliable, as there is a certain individuality in the
different strains of spirochoetes. S. duttoni has
been cultivated by Noguchi by utilizing his methods
for cultivating the S. pallida. Workers in various
fields have given their names to the spirochoetes
causing diseases they have worked at, thus we have,
besides S. duttoni.—

S. recurrentis (obermeieri) causing European
relapsing fever.

S. berberi causing North African and Egyptian
relapsing fever.

S. carteri causing the Indian variety of relapsing
fever.

These three forms are usually transmitted by lice,
but the bed-bug has been suspected as being a trans­
mitter of S. carteri, and Nuttall has succeeded in
transmitting S. recurrentis from mouse to mouse by
the bites of the same bug.

S. novyi causing relapsing fever in Colombia is
transmitted by O. turicata.

In Persia relapsing fever is transmitted by O. tholozani.
Drake-Brockman thinks that the spirochoete causing
relapsing fever in Somaliland is transmitted by
O. savignyi.

Nicolle showed that the method of infection when
lice are the carriers is by the scratching of the
skin and crushing of the lice containing spirochoetes
on the excoriated surface of the body. An emulsion
of infected lice placed on the conjunctiva of monkeys
has/
has caused fever. As regards the mode of transmission when ticks are the carriers, Leishman has shown, (and Fantham and Hindle have independently repeated successfully his experiments) - that if infected ticks were interrupted while feeding, no infection resulted in monkeys. He demonstrated that infection proceeds, not from the salivary gland, but from the excrement, i.e. the thick, white Malpighian secretion voided by the tick while feeding. This infected excrement, diluted by the coxal fluid passes into the wound caused by the bite.

Dutton and Todd proved early that ticks could remain infective for months, and that the tick eggs could be infected and the disease handed on to the next generation.

Recently Frankel has proved by biological tests that the spirochete of East African relapsing fever differs from S. duttoni as observed in West Africa and Nuttall has proposed the name S. rossii Nuttall 1908 for the new species.
The protozoal diseases of man and of other vertebrates are illustrations of the great powers of adaptability of their environments on the part of the responsible Protozoa. Parasitic life did not commence as such. It has been gradually evolved from a free-living independent existence, and only the more adaptable and resistant forms have been able to withstand the vicissitudes of the change from a free life of unlimited range and possibilities to a more restricted life of saprozoism, where the provision of at least partly digested food was a necessity and, finally, to a life of parasitism, or living at the expense of another organism, (the host), and being subjected to violent reactions on the part of its host, as the attempts of the leucocytes to combat certain blood parasites show. Under such conditions wherever large bodies of men are collected together on ground that has been fought or is being fought over, unsanitary conditions are inevitable. Lack of convenience for cleanliness, the character of the land, and the action of the guns in opening up old cesspits, graveyards and the like, together with conditions of overcrowding all aid in promoting a condition/
condition favourable to parasites and unfavourable to man.

Epidemic diseases due to protozoa inhabiting the intestine or the blood of man have been prevalent in certain war zones, especially those having tropical or subtropical situations.

Representatives of the chief groups of Protozoa are very common, the Sarcodina occurring in Amoebic Dysentery, flagellates in various diarrhoeas and infections such as Trypanosomiasis and Leishmaniasis, spirochaetes in relapsing fevers, sporozoa in the various malarial fevers and Coccidioses, and ciliates in the case of balantidian dysentery.

SHORT CLASSIFICATION OF PROTOZOA, invading and parasitic on man.

1. Sarcodina (Rhizopoda)
   a. Entamoeba histolytica causing amoebic dysentery.
   b. Entamoeba coli causing (?) mucous colitis.

2. Mastigophora (Flagellata)

Trypanosomes
   a. T. gambiense causing sleeping sickness.
   b. T. brucei causing nagana. Analogous to T. rhodesiense.

Herpetomonas.
Herpetomonas.
   a. Leishmania donovani causing kala azar.
   b. L. tropica causing oriental sore.
   c. L. infantum causing mediterranean or infantile kala azar.

Intestinal Flagellates.
   a. Giardia (Lamblia) intestinalis, found in many cases of diarrhoea and dysentery.
   b. Trichomonas hominis causing dysentery.
   c. Chilomastix mesnili causing diarrhoea.

Spirochoeta.
   a. Spirochoeta duttoni causing African Recurrent or Tick Fever.
   b. S. ictero-haemorrhagiae - called Leptospira causing Weil's disease because small spiral throughout the body.
   c. S. bronchialis
      Bronchial affections in the Soudan.
   d. S. schaudinii
      found in Ulcus Tropicum.
   e. S. vincenti
      Found in Vincent's Angina.
   f. S. dentium and Sp. buccalis
      found in the mouth - non-pathogenic.
   g. S. novyi
      causes relapsing fever in America.
   h. S. rossii causes East African relapsing fever.
   i. S. berberi causes North African relapsing fever.
   j. S. carteri - Indian relapsing fever.
   k./
k. S. euryzyrata – in human stools.
l. S. urethrae – in " urethra.
    Macfie (1917)
m. S. morsus muris – rat bite fever.
n. S. hebdomadis – causing 7-day fever.

   a. Intestinal coccidia
      Eimeria stiedae or Coccidum.
      C. oviforme
      causing chronic diarrhoea.
   b. Malarial parasites.

4. Infusoria (Ciliata)
   a. Balantidium coli produces ciliate dysentery.

EVOLUTION OF PARASITISM IN SPIROCHOETES.

Spirochoetes present an interesting study in the evolution of parasitism, for they afford striking examples of the graduation from life in water to saprozoism and thence to parasitism.

The type species of spirochoete, Sp. plicatilis, from which Ehrenberg founded the genus in 1833, occurs in stagnant pond water; another species, Sp. gigantea is found in sea-water. Spirochoetes occur in the digestive tract of many bivalve molluscs. Gross (1911) suggested the generic name Cristispira for molluscan spirochoetes, because they possess a well-marked membrane/
membrane or "crista". The alimentary tracts of most animals, apparently, may contain spirochotes which they have swallowed with their food or drink.

Spirochotes of aquatic animals ingested by higher animals can become habituated to the new environment of the alimentary tract, and their power of adaptability determines whether they will be pathogenic or non-pathogenic to their new host. In general terms, the newer a parasite is to its host, the less is it in harmony with that host and the more pathogenic are its effects, e.g. Trypanosomiasis in wild and domestic animals. Again, free living spirochotes found in water may be ingested by a host and find their way into the body cavity of that host. Many generations of such forms may result in the production of a spirochete quite tolerant to such a host as an insect or tick.

The acquisition of the sanguivorous habit of life by an insect is secondary, and when this mode of life has become established, the spirochotes of a sanguivorous ecto-parasite have already become accustomed to life in a medium containing some blood.

The transition to life in the blood of vertebrates is then relatively easy via the wound made when the insect bites or is crushed on the abraded skin of the vertebrate host. The parasitism and pathogenicity of *Sp. recurrentis* and *Sp. duttoni*, the/
the excitants of relapsing fever in man, may have arisen in this way.

It is sometimes made a matter of argument as to whether the spirochoetes are Protozoa or Bacteria. There is no sharp line of demarcation, and one can argue either way. Novy and Knapp are chief among those who consider them to be bacteria, chiefly on account of their morphology (absence of demonstrable nucleus and blepharoplast.) Doflein called them Proflagellata on account of their mode of division, transmission by ticks, reaction to drugs, chemicals and stains, and difficult cultivation. Fantham considers they are intermediate in character, showing morphological affinities with the Bacteria and physiological and therapeutical affinities with the Protozoa. He calls them Spirochoetacea. They may safely be called Protista. The generic name Spirochaudinnia, proposed by Sambon, has been adopted by Minchin.

We now turn from the study of the spirochoete to that of its transmitter, the tick. It would be difficult to name any biological discovery that has made greater advance in medical and veterinary science during recent years than that of the extraordinary part/
part which blood-sucking flies and ticks play in the dissemination of disease in man and his domestic animals. The association of ticks in the transmission of disease has engaged, and is still engaging the attention of the medical and veterinary profession, the bacteriologist and the entomologist with the result that questions of the greatest economic importance have been solved, and the findings turned to profitable account.

The epidemiology of tick fever rests upon the life history of the ticks, *O. monbata*, *O. savignyi* and as recently proved *O. tholozani*. 
TICK INFESTATION.

Short Classification of the Ixodoidea.

The Ticks or Ixodoidea are divided into two groups, known as (1) Argasidae, (2) Ixodidae. The former, known as "soft ticks", are distinguished from the latter by the following characters:-

a. The integument is leathery and chitinous plates, including a scutum are absent.

b. The capitulum is ventral and invisible from the dorsal surface (except in the case of the larva).

c. The spiracles are situated between the 3rd and 4th legs.

d. The sexes are difficult to distinguish from each other.

e. The palps are finger-shaped or leg-like, not club-like.

f. They have no pulvillus.

g. Their habits, especially as to egg-laying differ. They do not die as soon as they have produced their species, but they live and grow (as is evidenced by the frequent moulting of the cuticle) for years, and the female produces more than one batch of eggs.

Subfamily/
Subfamily Argasidae.  See page 27.


Genus Ornithodoros.  Margins of body rounded, not specially defined.  Skin has many irregular tubercles.  Eyes, when present, two pairs, situated on the supracoxal fold.  Rostrum even with anterior margin so that ends of palpi slightly project.

Subfamily Ixodidae.  See page 31.
General account of the diseases and causal parasites transmitted by Ixodoidea and of their evolution in the Tick and in the animal host.

From the medical standpoint the Argasidae or soft ticks are of greater interest than the Ixodidae or hard ticks, since they are more definitely human parasites. Their bite often causes severe and prolonged irritation, and even spreading inflammation and suppuration. Several species are known, others are suspected, to be the ordinary agents in spreading the infection of certain dangerous spirochoetal diseases of man and birds. According to Christy, O. moubata is capable of transmitting Filaria perstans. Ixodidae work havoc among domestic animals, (a) by simple abstraction of blood; (b) by spreading among them the infections of certain destructive diseases due to specific piroplasms and spirochoetae. As a given tick in any one stage of its existence feeds only on a single host, if it should become infected from that host it will not have the opportunity of passing on the infection directly, but the infection remains latent and becomes active in some subsequent stage of the tick's existence or in some subsequent generation. In some cases a female tick transmits the/
the infection to her eggs, so that in due time the larvae from these eggs are infective. In other cases, where the specific blood-parasite has a longer term of development, the larvae hatched from infected eggs are not infective, nor are the nymphs; though both larva and nymph are infected. Here, the development of the specific parasite is not complete until the generation inheriting the infection has become adult, and it is the adult that is infective. There are other possibilities, as where a larva or a nymph feeding on an infected animal incubates the infection and becomes infective in the stage immediately following - the larva when it becomes a nymph; the nymph when it becomes an adult. A tick once infected seems able to be infective for the rest of its life. Nuttall has found the piroplasma of malignant jaundice of dogs in a Haemaphysalis after seven months' starvation, and thinks it possible that an infected tick (presumably inheriting its infection) may transmit the infection to its offspring even though it itself should feed on a healthy animal.
The Parasites transmitted by Ticks, their evolution in tick and in the vertebrate host.

Family Argasidae.

The development of spirochoetes in the bodies of Argasidae, and the mode of their transmission to man and other animals has already been touched upon in the preceding pages. As an example one may describe the transmission of *Sp. duttoni* by *O. moubata*. It is transmitted by both adult and nymph. It is generally from 16 - 24 μ long, but may be from 12 - 36 μ long and ½ μ broad. It has a corkscrew-like and undulatory movement. It can be cultivated by Noguchi's method, in a medium of ascitic fluid containing a piece of fresh sterile tissue. When blood containing spirochoetes is ingested by a tick the spirochoetes can be demonstrated in the tick's gut, Malpighian tubes, salivary glands, genital organs and excrement at one time or another. Examination of coxal fluid usually proves negative, but the thick white excrement from the Malpighian tubes is positive. Ticks incubated at 21° to 24° C. show no spirochoetes, as such, in their organs except in the gut. When incubated at 37° C. for two or three days, spirochoetes, as such, reappear in the gut, organs and haemocoelic fluid./
fluid. Infection proceeds, not from the salivary gland, but from the thick white Malpighian excrement voided at the end of a meal. This material passes into the wound caused by the bite, aided by the clear coxal fluid which bathes the under surface of the tick's body. It has been proved that the spirochetes in the gut of infected ticks divide by a process of multiple transverse fission into granules, known as coccoid bodies or infective granules. Leishman first found them in clumps inside the cells of Malpighian tubules. By the spirochetes boring their way into the various organs and then breaking up into granules it can be explained how young ticks are infected when they bore their way into the ovaries; also it can be shown how the salivary glands may become infected by observing the cell development in the metamorphosis of the tick. The tissues within the larval cuticle forming a rough model of the nymph stage are made up largely of undifferentiated cells. The first part recognizable is the duct of the salivary gland, and if these undifferentiated cells are infected the salivary gland will be infected. Infective granules are also seen in the rudimentary Malpighian tubules of embryo ticks. Bosanquet and Fantham have shown that molluscan spirochetes also break/
break up into similar granules. Marchoux and Couvy, and Wolbach, who showed that certain spirochoetes can pass through a Berkefeldt filter as spirochoetes, consider the granules to be degeneration products, but Fantham considers otherwise, and with dark-ground illumination has seen the granules grow up into spirochoetes. This granule phase is infective to man, and the same multiplication of *S. recurrentis* by longitudinal and transverse division (including so-called "incurvation") and formation of small ovoid bodies has been shown in the louse. Nicolle and Blatt found that the organisms are infective in the louse just before they reappear as spirochoetes.

**Family Ixodidae.**

Many of the species of this family are known to attack man. They are chiefly important on account of their rôle in the transmission of diseases produced by a group of parasites belonging to the *Haemosporidia* known as *Piroplasmata*. These produce disease in oxen, sheep, horses, dogs, etc. The various genera may be listed as follows:

1. **Babesia.** This is one of the best known piroplasms and was first shown by Smith and Kilborne (1891) to be tick borne. Eight different species have been described, transmitting disease to different animals.
(2) *Theileria*. Rod-shaped and oval parasites. *T. parva* is the pathogenic agent of African East Coast fever in cattle.

(3) *Nuttallia*. Oval or pear-shaped parasites. *N. equi* is the cause of "piroplasmosis" in horses.

(4) *Nicollia*. Oval or pear-shaped with characteristic dimorphism. *N. quadrigemia* from the gondi.

(5) *Smithia*. Pear-shaped single forms stretching across the blood cell. *S. microti* and *S. talpae* from the mole.


**The Life History of the Piroplasma in the Tick.**

This can be briefly described with the aid of a diagram. This is interesting inasmuch as piroplasmata have been suspected of causing Spotted fever of the Rocky Mountains also blackwater fever; but there is no evidence of piroplasmosis in either of these affections.

Seidelin claimed that *Paraplasma flavigenaum* was the cause of Yellow Fever. *Oroya Fever* is caused by *Bartonella bacilliforms*.
Life History.

When the tick ingests the blood of its host the pear-shaped parasites are set free from the corpuscles in the gut of the tick. In from 12 to 18 hours they become amoeboïd sending out pseudopodia. These stellate forms are gametes and fuse in pairs. (Koch). 4 - 5 μ. A spherical stage follows which grows into a uninucleate globular mass. These then become club-shaped and motile and four times the size of the blood forms. 25 μ. These forms pass into the ovary and so get into the ova. There they become globular and later are found in the cells of the developing tick-embryo. They may then be transmitted hereditarily. The globular body becomes divided into a number of "sporoblasts" which are scattered through the tissues of the larval or nymphal tick. The sporoblasts divide into a large number of sporozoites which collect in the salivary glands of the tick and are inoculated into the vertebrate when the tick next feeds.
LIST OF TICS TRANSMITTING DISEASES.

Family Argasidae.

(a) Argas reflexus. The Pigeon Tick. It is known in Europe, North Africa, North and South America. It sometimes attacks man, and its bite causes much local inflammation.

(b) Argas persicus. The fowl tick. This so-called Persian tick, the "miana bug", is a scourge to travellers in Persia. The virulence of its bite is probably due to its transmitting fever germs to susceptible strangers from immune natives. In North America it is called A. miniatus.

(c) Argas brumpti. Attacks man in Somaliland and in the Sudan, and produces a painful swelling.

(d) Argas chinche. Very troublesome to man in Colombia.

Genus Ornithodoros.

(e) Ornithodoros moubata. This is the common tick which transmits the Relapsing fever of Tropical Africa, and it is with this parasite that the thesis is specially concerned: Its geographical distribution has been already mentioned, being usually found in localities on the trade-routes. In some rest houses 50% of the ticks may be infected. It hides in/
in the crevices and in the cracks of floors and walls and in the thatch of huts, coming out at night to bite the sleeping inmates. The bite may be very painful causing raised wheals on the skin. I have studied the habits of O. moubata which are more or less typical of the other species of the genus. It is chiefly nocturnal, but I have known natives to be bitten by them during the day on more than one occasion. When handled or touched they feign death, remaining motionless, with the legs bent close to the body, and thus when searched for in a dark place may be easily overlooked. They are very hardy, and can live for months without food. I myself kept three female O. moubata, fullgrown, nine months without food. They are said to feed on animals and birds as well as man. I can confirm the first part of this statement as I have taken the parasite from a cat which was kept in the native gaol at Abercorn, N.E. Rhodesia. I have also fed them experimentally on a monkey. When young it is oval yellowish, when mature it is greenish-brown and has always a very disgusting, bloated appearance. It feeds moderately, and after feeding it moults and repeats the process frequently. This allows for increased growth. Eggs are produced in batches of one or two hundred odd, and are laid in masses on the ground or in crevices. One tick may live several years and produce/
produce several batches of eggs. A three-legged larva is hatched in about one week. The larvae at first are sometimes seen to lie on their backs, making no attempt to feed, but in less than one week they moult and the active four-legged nymph appears and it is really in the nymph stage that the eggs from O. moubata hatch. By placing a gorged female O. moubata in a small pill-box I have seen the process of egg-laying take place through the glass lid of the box. On another occasion, previous to this, I pinned a gorged tick (O. moubata) to the bottom of a pill-box, the pin going right through the middle of the tick. Remembering it 10 days later I opened the box and found the mother tick still living and was able to count over two hundred very active nymphs. The absence of eyes distinguishes it from the closely allied species O. savignyi.

(f) O. savignyi. Very like O. moubata in general appearance. It has a wide distribution in Africa and occurs also in Aden and in South India. Drake-Brockman has studied its habits in Somaliland, and thinks it may take the place of O. moubata in spreading fever there. Brumpt has succeeded in transmitting tick fever by means of O. savignyi.

(g)/
(g) *O. pavimentosus*. Differs from *O. savignyi* in having the granules contiguous and flattened. Occurs in Namaqualand and hides in the sand, biting travellers when they lie on the ground.

(h) *O. coriaceus*. Attacks man in Mexico, Paraguay and California.

(i) *O. turicata*. "Adobe-tick". Suspected of carrying relapsing fever in Colombia.

(j) *O. talaje*. Causes a painful bite in man. May be found to transmit the spirochaete which causes relapsing fever in Panama. It is also found in Venice and on several islands in the Indian Ocean and South Atlantic.

(k) *O. tholozani*. The carrier of the spirochaete causing tick fever in Persia. (Marzinovsky).

(l) *O. megnini*. The spinose ear tick. So-called from presence of hairs on integument of nymph. They have been taken from the ears of child on several occasions.

(m) *O. canestrinni*. Described from 15 specimens taken at Mahaballeshwar, Satara District, India, at a height of 4200 feet.

(n) *O. lahorensis*. These have both been suspected of conveying disease to man in Persia by biting.
(o) O. pyriformis. Recently described, are small, notably pyriform in shape.

(p) O. asperus. Resembles O. talaje but integument more finely mammilate and tarsi distinctly humped.

Family Ixodidae.


(a) I. ricinus. Widely distributed in Europe, Asia, North Africa and North America. Chiefly attacks sheep but may attack man.

(b) I. holocyclus. Commonly attacks man where there is dense scrub and tropical jungle on the east coast of Australia. It may cause severe symptoms in children causing death. Taylor records it as attacking man in Queensland and New South Wales. It causes the disease known as tick-paralysis.

(c) I. hexagonus. Occasionally attacks man but is confused with I. holocyclus. Found on hounds, stoats, ferrets and hedgehogs.


Eyes present. Ornate. Long rostrum.

(d) A. cayennense. Common in the whole of Central/
Central America, attacking mammals, amphibious animals and man. The silver tick. Newstead records it as the worst pest to man in Jamaica, a vicious biter which produces an irritating wound followed by intense itching.

(e) A. americana. The so-called lone star tick from a silvery spot on the apex of scutum of female. Bites milkers and moss-gatherers in the Southern and Eastern states; also in Brazil and Guiana.

(f) A. maculatum. Found on the Gulf Coast and attacks birds, mammals and man, attacking especially the ears.

(g) A. variegateum. Found in French Guinea, French Sudan and Nossi-Be' Island, near Madagascar. It attacks man and other animals in these places.

(h) A. herbraeum. Found near Laurenco Marques, Natal and Cape Colony. They are a great scourge to man in cattle areas, setting up acute itching, larval ticks being found in little vesicles on the skin of the person attacked. General symptoms may be observed, - weakness, muscular pains, enlarged glands, stiffness of neck and headache. It causes heartwater in sheep and goats.
Genus *Hyalomma*. Characters. Palpi long, slender and of equal length.

(a) *H. aegyptium*. Abundant in Africa, also in S. Europe and Asia. It attains a large size, attacks camels, cattle and horses and may attack man, its bite producing morbid symptoms. (Ronsisvalle).

Genus *Haemaphysalis*. Characters. Very broad rostrum, triangular short palpi, and no eyes.

(a) *H. punctata*. Uncommon. Occurs on sheep, goats, horses and cattle and dogs. Theobald has seen one taken from man in Britain.

Genus *Dermacentor*. Characters. Has eyes, like *Amblyomma*. Most important characteristic is the large size of the coxae of the fourth pair of legs. No abdominal plastron in the male.

(a) *D. reticulatus*. S. Europe, Asia and America. Rare in England and Wales. It attacks sheep, cattle, deer, horses, goats, and sometimes man. It transmits Biliary Fever in horses.

(b) *D. occidentalis*. The Pacific Coast Tick or Wood Tick. Occurs on the Pacific coast region of U.S.A. and on domestic live stock. It commonly attacks man, the bite causing considerable local inflammation, itching persisting for weeks.

(c)
(c) *D. venustus* (Bankes). Occurs from British Columbia southward to Mexico, and from the foothills of the Rocky Mountains in Colorado to the base of the Cascade Range in Oregon and California. The larvae and nymphs are found on small mammals. The adults are found on larger domestic animals. Sometimes they bite man and can convey the disease known as Rocky Mountain Spotted Fever. Stiles described the tick which causes the disease under the name *D. andersoni* and considers *D. venusti* to be different. Some wild animal is supposed to be the reservoir of the disease. In Montana it causes the disease and is very fatal from March to July, the tick being most abundant then.

In British Columbia it causes tick-paralysis in man and sheep. When the tick is entirely removed the symptoms disappear. Hadwen and Nuttall produced paralytic symptoms experimentally in a dog at Cambridge.

(d) *D. variabilis*. This is a North American dog-tick which sometimes attacks human beings.

Genus *Margaropus*. Characters. No anal groove or festoons and circular stigmata.

(a) *M. annulatus australis* (*Boophilus bovis*). This tick has a wide distribution in Central and S. America and is the carrier of Texas Cattle Fever. Newstead/
Newstead states of this tick: "It is generally believed that the larval or 'grass lice' stage of this tick will attack any vertebrate animal coming its way. It is in this stage that it is such a great pest to man."

(b) *M. microplus*. Recorded by Aragao as occurring in larval stage on man in Brazil.

(c) *M. annulatus decoloratus*. Widely distributed in Africa. Said to attack man.


(a) *R. sanguineus*. The common dog-tick, transmitting Malignant jaundice. It sometimes attacks human beings. It is cosmopolitan in distribution.

Genus *Aponomma*. Characters. Like *Haemaphysalis* eyes absent, body subcircular. Distributed in Africa and the Orient, the parasitic almost exclusively on reptiles.
In the following pages I have given a short account of several cases of tick fever, accompanied by charts, which have come under my care at different periods from January 1912 to February 1917 in South Central Africa, chiefly in North Eastern Rhodesia. I also append an account of two experiments undertaken to prove the mode of the transmission of the disease, monkeys being used.

In order to better compare my own cases with the accepted standard accounts as published in the textbooks, I append below the account of the first case diagnosed and published by P. Ross in British East Africa, October, 1903. This seems to be a fairly typical case.

"The patient was a small poorly nourished man, thirty years old. His temperature was 103.4°. He was prostrate, anxious, vomiting matter of a greenish colour and complaining much of pain in the epigastrium and small of the back. He also had rather severe headache and pains in the limbs. There was some tenderness over the liver and spleen and slight enlargement of the latter. The urine was febrile/
"febrile and contained nothing abnormal. In the
afternoon the temperature was still over 103 and the
next morning it was normal, having fallen in the
small hours apparently by crisis. In blood films
taken next morning no spirochoetes could be found.
He complained then of nothing but weakness, and in
another day was at work again. On November 14th -
the 19th day - the patient had a relapse almost
exactly resembling in symptoms and duration the first
attack, and spirochoetes were found in both fresh
and stained specimens of blood."

He then goes on to describe the inoculation of a
monkey with blood from this case, the symptoms in the
monkey, and the finding of spirochoetes in its blood
in large numbers. The above patient was an Indian
who had been resident in Uganda for over a year.

"A blood film stained with Leishman's stain
showed one spirochoete to 15 or 20 fields, staining
bluish, and sharply pointed at both ends, about 4 \( \mu \)
in diameter and .36 \( \mu \) to 42 \( \mu \) long.

Symptoms generally present in the attacks as
described by the earliest workers, Ross and Milne,
Dutton and Todd and Breinl and Kinghorn are the
following:— The incubation period varies from 5 to
10 days. The attacks began with a feeling of malaise
and mental dulness. There was always present more or/
or less severe headache, pains in the back and limbs. Often there was vomiting. There was generally a certain degree of enlargement of the spleen accompanied by splenic tenderness. Often liver enlarged (Moffat). Respiration was quick, often cough and dulness over bases, with harsh, tubular breathing. The pulse, during the attacks varied between 90 and 120. The temperature from 100 to 105 or 106°F. About half the cases suffered from diarrhoea; less frequently there was constipation. In Dutton and Todd's cases. In Moffat's cases in Uganda, constipation was the rule, unlike the cases in the Zambesi basin, in which purging was more common. The skin was hot and dry, the conjunctivae congested, the tongue moist, white and furred. Sometimes there is a slight bronchitis. Jaundice is rare, and when present the prognosis is grave. Oedema of the eyelids has been noted. The urine is typically febrile, and not appreciably diminished. As in relapsing fever as observed in Europe and India, the initial pyrexia, called first paroxysm, ending in crisis characterised by profuse sweating, is followed by a first period of apyrexia during which the patient recovers so rapidly that after four or five days it may be difficult to keep him in hospital. This first paroxysm or initial fever generally terminates in crisis within three days, being not so prolonged as in the European and Indian/
Indian relapsing fevers. The apyretic intervals are of very irregular duration, being according to P. Ross, sometimes as short as one day, sometimes as long as three weeks; and instead of only one or two relapses, there may be as many as eleven, five or six relapses being the rule. The fever, though shorter, is as severe in the relapses as in the initial paroxysm, but the intervals tend to become longer. Sometimes the fever may assume a low chronic form, with headache and vomiting. The parasites are usually very scanty in the peripheral blood and may be hard to find. The only complication that seems to be mentioned in the textbooks is iritis, which is not uncommon.

The distinguishing features of *P. duttoni* and the clinical symptoms it evokes in man and animals differ from those of the other relapsing fevers as follows:

(a) Small rodents and many animals are susceptible.
(b) The course of the disease in animals is very severe.
(c) Subinoculations are positive for most animals.
(d) The course in man is severe, generally with four or five relapses, the duration of attack being only three days and periods of apyrexia irregular, generally seven or eight days.
(e)
The natural transmitter is *O. moubata*.

Immune serum is without any effect on *S. novyi* or *S. recurrentis*.

The spirochoete itself is long, generally about 13 μ, has open flexures flagella peritrichous, not terminal and very scanty in the peripheral blood as compared with European relapsing fever.

Philip Ross has divided the disease into two varieties, European and Native, the latter being a mild affection. He cites a case of a Native who suffered from an attack of tick fever, and who had no rise of temperature at the time when a relapse was due, his only symptom being severe headache. A microscopical examination of blood films taken at this time revealed the presence of spirochoetes in the peripheral blood. One of my own cases - the first native case which I have recorded - suffered in the same way without rise of temperature. I was successful in finding spirochoetes in blood films. In another case with similar symptoms I was not successful. There was a distinct degree of polymorphonuclear leucocytosis, however, present. Dutton and Todd have placed on record many severe and fatal cases of the disease occurring in natives in the Belgian Congo,
Congo, and Castellani, as a result of these two forms being met with in natives, puts forward the theory that there are two types of the disease, an East African or mild type and a West African or Conge type which may be fatal. As a confirmatory evidence, he quotes the biological tests carried out by Fränkel showing that the spirochoete of East African relapsing fever differs from Sp. duttoni as observed in West Africa. That this is the case is, however, very far from being proved, and I can cite my own cases which comprised all varieties of the disease from the mildest to one which was fatal, in natives. It is generally agreed that the mildness of the attacks in natives, sometimes without relapses, may be explained by a partial immunity conferred by previous attacks. There can be no doubt, I think, and in the light of my own cases, that the previous state of health of the patient and his condition at the time of his contracting the disease, is an important factor in determining the course of the disease.

MORTALITY.

According to the textbooks the death rate is 6% or below. In the feeble and old, death may take place at the height of the first paroxysm. Among my own cases the mortality was 2.5%.
This is usually made by the microscope, failing which, animal injection is relied on. Lowenthal’s agglutination method may also be tried. Dried films are best thin, the spirochaetes then being seen in the usual undulating form. In thicker films they appear more frequently coiled up, but I have found this method very useful when the spirochaetes were scanty in the peripheral blood.

As regards the symptoms of the disease, my own cases have shown practically all these mentioned in the textbooks. The only complication which is mentioned as of any importance by all the authors is iritis. No doubt this is a very important complication, but I have met with it in only one case in a European. Stiffness of the jaws, staggering gait, and signs of meningeal irritation were observed in three of my European cases, and neuralgia was a symptom in three. In all of them there was great restlessness during the paroxysms, accompanied by very distressing insomnia. Facial paralysis was observed in two of my cases, one of them being a very pronounced case. Photophobia was present in four of my European cases. Case No.1 did not show nerve symptoms like the other cases and he had on the average the highest temperatures during the attacks/
attacks and sweated most profusely. It is interest-
ing to note the mildness of the attack in the case of Case No. 3, and his history of a previous attack some years ago.

As regards the disease in natives, I think that one may meet with all degrees of virulence. Nearly all degrees were met with in my cases, at any rate. I do not think that sufficient data have as yet been collected to enable the disease as it appears in natives to be divided into two varieties. My cases were met with in natives infected in the Congo, N.E. Rhodesia and East Africa. I wish to emphasize the great importance of the nervous symptoms, which, in the absence of microscopy, and taken in con-
junction with the course of the disease, are quite diagnostic. The occurrence of severe headache, vomiting and photophobia in natives in an infected district is very suggestive of a mild attack of spirochoetosis, and even if the temperature is not raised. Neither in Europeans nor in natives did I see a very marked degree of inflammation arising from the bite of the tick, and this is in contradistinction to what others have observed. Natives whom I have talked with and treated are quite sure that bugs (Clinocoris hemipterus) can carry and transmit the disease, and they are continually being bitten by them/
them, not distinguishing between the bites of ticks
and bugs well.

Refford and Duke in Uganda describe a case of
spirillum fever which occurred in a European in
German East Afria near Muanza. What they describe
as remarkable features characterising the attack were
the occurrence of subtertian malarial parasites in
the blood, giving rise to pyrexia almost at the same
time as one of the pyrexial attacks due to the spiro-
choete, the prolonged course of the disease, the
seventh relapse occurring 195 days after initial in-
fecion, the patient not being exposed to any chance
of reinfection and the regularity and relative ease
with which spirochoetes could be found in the blood
during the attacks. Between the second and third
definite relapses there was a period of 46 days
apyrexia, between the third and fourth, 25 days,
between the fourth and fifth, 19 days, between the
fifth and sixth, 17 days, and between the sixth and
seventh, 9 days. The case was sent in to hospital
with a diagnosis of measles.

This case simply serves to emphasize the need
for the administration of quinine as a routine in
malarial regions along with measures to destroy the
spirochoete in the blood. Also, it may quite likely
have been an infection with more than one strain of
spirochoete/
spirochaete or a double infection. The relative ease with which spirochaetes were found in stained films points to nothing but improved technique. It must not be forgotten that one or more relapses may be partially or almost completely suppressed.

They did not resort to Salvarsan in treatment as in their experience at Muanza Hospital it proved unsatisfactory. Possibly their technique was at fault, for I know of no other workers with an experience of the disease who do not agree as to the efficacy of the drug.

As regards spirillosis in fowls, Levaditi and Manouelian believe that the destruction of the spirochaete and cure of the disease are brought about by phagocytosis carried out especially by macrophages of liver and spleen; some of the symptoms of the malady are attributed by them to thrombosis of blood vessels caused by impaction of masses of spirochaetes. Neufeld and Prowazek deny phagocytosis, and attribute recovery to the action of the serum, which is parasiticidal in vitro and also capable of protecting other birds. Atoxyl, useful in the treatment of trypanosomiasis, is also valuable in the treatment of fowl spirillosis, being both preventive and curative. Salvarsan is also very efficacious. The occurrence of spirochaetes in the blood in cases of Trench fever has been lately observed by Coles and others. These are very difficult to find, but/
but by using dark ground illumination Coles was able to demonstrate the spirochoetes more easily. Salvarsan, however, has not proved very efficacious in this complaint.

Salvarsan has also been employed with reported success in cases of chorea and the various forms of leucaemia, also in variola and scurvy: in the latter disease the beneficial effect being first seen in the mouth, no doubt due to the destructive action of the drug on the spirochoetes which cause the ulceration.

The tropical disease called yaws, framboesia, parangi, etc., due to Sp. tenue is the most readily acted on by Salvarsan of any disease known.

Summarising, we find that there is a group of spirochoetes associated with affections in which the symptoms are fever, a tendency to relapses, and enlargement of the spleen. In these diseases the spirochoetes give rise to a septicaemia without the formation of any local lesions.

METHODS OF STAINING.

(a) Breinl and Kinghorn's method of examining the blood for spirochoetes was the ordinary thickfilm one. Two or three drops of blood were placed on a perfectly clean slide and then spread out over a surface 2 by 3 cm. After drying in the air the films were fixed in the flame and the haemoglobin was removed by washing the films in distilled water. They were then stained with Romanovsky's stain made in accordance/
accordance with directions given by Stephens and Christophers, for half an hour.

(b) Giemsa's stain.

(c) " " followed by a watery solution of alum.

(d) Carbol-fuchsine diluted 1 to 3 of water which may be followed by Giemsa's or the film may be previously treated with 5% phosphoric acid.

(e) Carbol-gentian violet.

(f) Cresyl violet.

(g) Methylene blue and eosin.

(h) Various modifications of Levaditi's original silver stain.

(i) Indian ink method.

(j) Dark ground illumination for living spirochoetes.

(k) Ross's method, which was the one I generally employed myself.

Care must be taken not to confuse for spirochoetes objects resembling them, such as male gametes of some protozoa, flagella of trypanosomes, attenuated forms of these and of Herpetomonas or Leishmania; also Artefacts and Pseudospirochoetes.

Philip Ross's method of staining, which was the one I invariably used save in those cases when I resorted to the thick film method, is as follows:-

Two films were taken and one of them stained with aniline/
analine gentian violet or carbol-fuchsin, the other with Leishman's stain. Afterwards, if necessary, the oil was wiped off the film stained by Leishman's method, and without further fixing, restained with one of the other stains. Ross said that the parasite did not always stain well with Leishman's stain, but after I had several months' experience with this stain I found that it did very well by itself, if not used too fresh or too old - not older than ten days. The importance of a good method of staining and skill in using the stain cannot be too strongly insisted on when one considers how scarce the spirochoetes may be in the peripheral blood, even in severe cases of the disease, and there is no doubt that very many cases have been undiagnosed and ascribed to malaria in the past from the lack of facilities for microscopical examination, and also from the difficulty of finding the spirochoete in bloodfilms, - a difficulty greatly increased where the technique is faulty.

Stephens' method of staining may be used to demonstrate the "terminal flagellum", eviscerated forms and "linked" forms. He centrifugalised specimens of blood, washed the sediment in saline three or four times, and after mordanting, stained with gentian violet.
The chief diseases with which African tick fever may be confounded are malaria (chiefly), enteric, typhus and influenza. It is principally in the early stages that there is difficulty in diagnosing the disease as its relapsing character is not yet manifest. But later, the history of a fever relapsing about fourteen days from the commencement of the disease is suggestive, especially if there is the history of a bite of a tick or insect, though this is not always easy to obtain, especially in natives. Indeed, in natives, the disease is almost indistinguishable clinically, in mild cases, from ordinary attacks of malaria, and I am sure that I and others have overlooked cases of this disease when diagnosis by means of the microscope was not available. The mistake is all the more likely to have been made as, by the exhibition of quinine one may have thought that the fall in temperature was a therapeutic result and not a natural one.

Another factor mitigating against a correct diagnosis in natives is the difficulty of eliciting a history of a relapse. And again, there may be no relapse. I have had one or two cases of the disease in natives, of this kind. Moffat says that/
that it is quite common in Uganda, where he had a wide experience of the malady, and assumes that where the disease is so prevalent, such patients have had a previous attack. A history of a bite or bites from a tick is not always obtainable even in Europeans, as, in my experience, the bite is not always attended by much irritation, and if it were, bites from insects are so common and taken as a natural course of events, that very little, if any, reliance can be placed on this as a point of diagnostic importance.

MALARIA. For the above mentioned reasons it is important to make a correct diagnosis in a malarial district, and the general points of difference seen in tick fever are the following:-

(1) The occurrence of vomiting is infrequent in malaria in natives. It is fairly common among them in spirillum fever.

(2) There is much more complaint of pain in the back, head and limbs.

(3) The subsequent prostration is more marked relative to the duration of the attack.

(4) The pyrexia is less subject to remissions and tends to subside by crisis.

ENTERIC, TYPHUS, INFLUENZA. The history, course of/
of the disease and treatment ought soon to clear up any doubt as to the cause of the fever in each of these diseases. There should be no difficulty in recognizing the disease, from yellow fever, pneumonia, 7-day fever and Weil's disease, as mentioned in text-books.

Moffat states that in tick fever sometimes the liver is enlarged and tender and the spleen not. My cases did not show this clinical phenomenon or at any rate it was not observed, so that I cannot support him in bringing it forward as a diagnostic feature.

Plague must also be borne in mind.

PROGNOSIS.

This may be considered good in able-bodied men. It is not so good in the very young and in the old and debilitated. Pregnant women nearly always abort. Death is preceded by a rapid fall in the temperature and no improvement in the symptoms. (Moffat).

A prolonged irregular first attack ending by lysis and attacks showing marked jaundice are bad omens. (Ross). To this I may add that a case in which the temperature never comes down to normal during/
during the course of the disease, and in which spiro-
choetes are abundant in the peripheral blood, being
found even during the intervals between the paroxys-
ms, may be regarded as hopeless, unless it may be,
the administration of one of the arsenical prepara-
tions intravenously cuts short the disease. And
here, I may remark that in "606" or one or other of
the allied preparations we have a specific remedy
which, only recently used, has already vastly im-
proved the prognosis, recovery, in the great majority
of cases taking place after its administration,
without relapse.

MORBID ANATOMY.

The body usually shows signs of emaciation.

Spleen. This organ is usually enlarged, and according
to the text-books may be either firm or soft.

Liver. This may be enlarged and generally shows
signs of cloudy swelling. More often it
is not enlarged.

Kidneys. Usually show signs of cloudy swelling.

Heart. Shows cloudy swelling.

Bone-marrow is usually hyperaemic and dark in
colour, diffuscent.

The/
The skin and conjunctivae may be jaundiced, and there may be submucous petechiae. There is usually a marked polymorphonuclear leucocytosis. Spirochetes can be demonstrated post-mortem in the liver and spleen. From the post-mortem which I was able to perform on one fatal case and on a monkey I can corroborate the above statements. I would like to add that, in cases with marked nervous symptoms and signs of meningeal irritation, one would probably find some inflammatory changes in the meninges. Moffat mentions thrombosis of the vessels of the eyeball in cases with iritis. And this seems likely enough as in fowl spirochetosis this is noted in the case of Sp. gallinarum.

Pathological changes have also been noted by others in organs post-mortem, thus in lungs small areas of infarction are seen. Lymph glands are enlarged and some of them are haemorrhagic. Smears of organs show parasites mostly in liver, lungs and kidney.
NOTES OF CASES OF AFRICAN TICK FEVER.

Captain E. G. D., Northern Rhodesia Police.

This case was only attended by me during his fourth relapse and subsequently. This was the second of the only two cases in which I made the diagnosis of tick fever without finding the spirochaete in blood films. But he had been seen by Dr A. Kinghorn during his second relapse, who had also diagnosed the case as one of tick fever from finding one spirochaete in a series of blood films.

Patient was able to give me a few notes of his first attack as follows:- He had slept inside Abercorn goal for four nights, from August 11th to 15th. In spite of precautions he knew that he had been bitten on at least two occasions during these nights, but could not say whether he had been bitten by bugs or ticks exclusively. On the morning of the 19th he felt out of sorts and vomited once, shortly afterwards passing a very loose stool tinged with blood. Later, on the same day, he had a shivering fit and went to bed where his temperature rose to 104° F. Severe headache and backache then developed and a feeling as if he had been beaten all over which caused great restlessness and insomnia. These symptoms increased the next day and his temperature rose to 104.8° F. His temperature continued high/
high until the fourth day when it fell to just below normal. Eight days later he had a relapse which lasted four days, with a repetition of the foregoing symptoms. This was followed by another relapse in eight days. Between the attacks he felt quite well, but a little washed-out. He was seen by me on the 28th October 1914 during the crisis of his fourth relapse. His temperature then was 101.8°F. At this time he complained of thirst, pain at the back of his eyes, the conjunctives of which were injected, a dull aching pain in his thighs, and dry tongue. Mentally he appeared to be duller than usual. The crisis was not marked by much sweating. Examination of three blood films at this time showed nothing beyond a certain degree of anaemia. The spleen was palpable, a handsbreadth below the costal margin and slightly tender. The subsequent course of the disease is seen by a reference to the charts of the case. Treatment was symptomatic during the attacks and tonic during the intervals. He subsequently developed a neuralgia of the 5th nerve on the left side. Under continued tonic treatment, he made a good recovery. His illness lasted fifteen weeks and he had eight relapses.
Notes of Cases of African Tick Fever.

Rfn. L. P., N.E.R.

History.

This patient was admitted to Abercorn hospital on the 20th March 1915, during his first relapse. He had been treated for malaria for ten days previous to my seeing him. He gave a history of having been seized on the 10th of March with violent headache, pains in back and legs, especially severe at night, and vomiting. The headache and body pains increased in intensity during the next three days, his temperature at the height of the fever rising to 105.6° F. On the fourth day his temperature fell, patient having sweated very profusely. For the next six days he felt almost himself again, but on the tenth day headache and body pains returned and he was sent in to Abercorn for treatment in hospital with a diagnosis of malaria. On his arrival at Abercorn after a journey of sixteen miles in a machilla, I found him in a delirious condition, with a septic wound of the thumb and a temperature of 104.2° F. A blood smear showed two spirochaetae after two hours search. Patient was very restless, veronal and bromides having little effect. At 4 a.m. on 21st he became violently delirious, requiring five attendants to hold/
hold him in bed and was given an injection of morphine
grs. ½. By continuous sponging the temperature was
kept down and did not rise subsequently beyond
100.8° F. Extreme restlessness and insomnia per­s­
stained, iritis developed in the right eye and he
complained of some stiffness in his lower jaw for
several days. A second relapse occurred on the
30th, and a third on the 10th April, spirochoetae
being found in blood films during the height of the
fever but not during the apyrexial periods.
Polymorphonuclears were increased. On the 11th April
I gave an intravenous injection of Neo Salvarsan
0.6 grms. The temperature fell rapidly and improve­
ment was almost immediate. The iritis disappeared
in a few days, he began to sleep well at night and
appetite returned. He made an uninterrupted re­
covery and was discharged on the fiftieth day of
the disease. He gave a history of having slept in
a native hut in which ticks "O. moubata"were found,
six nights previous to the commencement of his
first attack. He had previously suffered from
malignant tertian malaria. Spleen and liver were
both slightly enlarged.
Notes of Cases of African Tick Fever in European.

Surgeon-Major A. E., M.O., Belgian Forces, Abercorn, N.E. Rhodesia.

Patient first seen during first relapse at Abercorn.

History.

While on his way to join his Unit at Abercorn, he slept in a grass hut near Chiengi, which had previously been slept in by some askari. On waking during the night, he felt a pain in his leg, and observed a tick crawling away, gorged with blood. The irritation caused by the bite was not very severe, but caused a small red lump which remained for a few days. Four days later, at Chiengi, N.E. Rhodesia, he was suddenly seized with severe pains in the back and legs, preceded by very severe headache. On taking his temperature he found it registered 104.8° F. at 4 p.m. He went to bed, where these symptoms increased in severity, accompanied by great restlessness. Vomiting occurred whenever he attempted to take food, such as chicken broth, or other light nourishment, for which he had no appetite. The pains continued and temperature remained high for the next four days, coming down to normal on the fifth day. The temperature reached its height on the second day of the disease, 104 to 105.4° F. During this time he suffered/
suffered greatly from insomnia, only gaining relief by injections of morphia hypodermically. The crisis was marked by very profuse sweating, and a gradual cessation of the symptoms, with return of appetite, and though weak, he felt able to continue his journey in a machilla. Seven days later, - the day after his arrival at Abercorn, - I was called to see him, and found him suffering from his first relapse. Temperature 104.6° at 11 a.m. 105° at 6 p.m. Pulse 120 per minute. Respirations 20. Blood smear showed scanty spirochoetae on staining by Leishman's method, and a slight increase of polymorphonuclear cells. From this day on, I was able to keep an exact record of his clinical features, which are shown in the accompanying charts. I would especially like to draw attention to the nervous symptoms, which were very severe. Patient was able to identify the tick which bit him as Ornithodorus moubata.

During my temporary absence, patient attempted one day to carry on with his work. He was seen staggering like a drunken man, and when asked questions could answer with difficulty owing to stiffness of lower jaw. This was during the height of fever of second relapse.

The full notes and charts of this Case are still in Dr B's possession.)
Notes of Cases of African Tick Fever.

Captain F. A. O. O., N.E.P.

History.

This patient slept in the same cell in the Abercorn goal as Case No. 2. He gave a history of being bitten by bugs at night on several occasions, but could not say whether he had been bitten by a tick or not. He gave a history of having already suffered from tick fever some three years previously, whilst in the civil administration of Northern Rhodesia. Like Case No. 2, he had slept in the infected goal for one week previously to the first symptoms of the disease, which were similar to those of Case No. 2, but not quite so severe. He was admitted to hospital on the 19th September 1934 and left on the 3rd October, having suffered from one relapse. I since heard that he had had another relapse of fever fourteen days after he left, being attended by Dr Allan Kinghorn (of S.S. fame), who also diagnosed the case as one of tick fever. The chief symptoms were intense headache, prostration, pains in back and legs and insomnia, the latter symptom being no doubt due to the former. The diagnosis was differentiated from that of malaria by/
by the course of the fever, the history, the fact of the administration of quinine in large doses having no effect on the course of the fever, and by the finding in blood films of a polymorphonuclear leucocytosis. Patient had also suffered from malaria of the malignant type, for many years previously, and, like the three former cases, had a slightly enlarged spleen, a clinical feature which is found in many old malarial subjects.

Beyond a certain degree of anaemia, this patient made a very good recovery in a short time. It is possible that he had acquired a certain amount of immunity.
This patient was admitted to hospital two days after Case No.1 with a very similar history of infection. He, also, had slept in the native goal for eight days previous to the commencement of his symptoms. He had caught several bugs which had bitten him on four successive nights, but was positive that he had not been bitten by a tick. However, he might have been bitten without knowing it, as these ticks do not always cause pain or much irritation when they bite.

The outstanding features of his case were the severe symptoms arising from meningeal irritation, delirium, facial paralysis, headache and photophobia. These symptoms were present during each rise of temperature. He also suffered greatly from insomnia. On the fifty-second day of the disease he refused to stay in hospital, and, though in a very weak state, started to walk to Kasama, 100 miles south. I heard that he had another rise of temperature on the road, with severe symptoms. He returned to Abercorn on the 25th, and on the 26th was readmitted to hospital with a temperature of 102°F. His pains, especially his headache, was so severe that I had to/
to administer morphia hypodermically, veronal and bromide not acting sufficiently. When I last saw the patient he had only partially recovered from the facial paralysis. This man was in very poor health from the very start, being an old elephant hunter, and having suffered from malignant malaria for years. The spleen was enlarged, palpable two finger-breadth below costal margin. He was troubled with constipation alternating with diarrhoea, but it is difficult to say how his malarious condition had to do with this symptom. He did not sweat much during the height of the fever. This was the only case that showed a rash, which was evanescent and mealy, disappearing after the fifth day.

The photophobia was so bad in this case that patient had to be kept in a darkened room for several days.
Notes of Cases of African Tick Fever.


History.

On the morning of September 16th, 1914, patient was found in bed, suffering from a high degree of fever, accompanied by very severe pains in back and limbs and frontal headache. These pains, he informed me, had commenced the previous evening, and he had vomited twice during the night. During the previous week he had slept for defence purposes inside the native goal at Abercorn, where he had been bit by numerous bugs. This place swarmed with ticks and also various sizes of bugs which, during the day, dwelt in cracks in the walls and floors of the cells, but which came out at night to feed. Patient was not able to tell me whether he had been bitten by a tick or a bug in particular. He had suffered previously in the Congo from severe malignant tertian malaria, and also on his arrival in Abercorn in August 1914. His temperature was 102.8° F. His native servant kept sponging him down with tepid water from time to time, and six hours later the thermometer recorded his temperature 102.2° F. A blood smear, stained by Leishman's stain showed numerous spirochoetes. I removed him to hospital and/
and kept him under observation. There was a slight degree of constipation present. He was put on a mixture of iron and arsenic, and given 5 grs. of quinine daily. His case was a very typical one with five relapses. This patient had very few nervous symptoms, sweated very profusely during the height of the fever, and towards the end showed great emaciation, but ultimately seemed to recover completely. When I saw him one month after his last relapse he had grown enormously stout. Patient suffered from a certain amount of photophobia when the fever was at its height.
Case No. 1.

History.

This was a store boy working at Kawambwa, N.E. Rhodesia. His chief symptoms were intense headache, pain in back and legs, especially down the back of thighs, photophobia and vomiting of stomach contents during first day of fever. There was some headache and renewal of body pains on the eighth day of apyrexial period, but no rise of temperature. Spirochoetae were found in the blood on the first day of the fever but not thereafter.

Case No. 2.

This was my own personal boy. He complained on the 5th January, one day after Case No. 1, of severe and continual headache and body pains. A blood smear showed the presence of numerous spirochaetae in films stained with Leishman's stain, on the first day of the fever. On questioning him I learnt that he had slept in the same hut as Case No. 1 seven days previously, and on making an examination of this I collected over thirty specimens of Ornithodorus moubata. Like Case No. 1, he made a good recovery without having a relapse.

Case No. 3./
Case No. 3.

This askari was infected at the same time, and from the same source as four of my European cases. Therefore we may infer that he acted as host to the same variety of spirochaete. His chief symptom was very severe headache. I determined to try the effect of Salvarsan and injected 0.6 grms. Neo Salvarsan intravenously. There was an immediate marked improvement in all his symptoms and he had no relapses.

Case No. 4.

This case was similar to No. 3 and infected from the same source. He had a relapse seven days after crisis of his attack, when I injected 0.6 grms. Neo Salvarsan. His temperature immediately fell and symptoms improved rapidly. He had no further relapse.

Case No. 5. (Fatal Case).

This was a very severe infection, affecting a woman during her puerperium. She was in a very anaemic enfeebled state of health. From the 13th day onwards she became very drowsy and finally on the 18th comatose. There were spirochaetae demonstrable in her blood during the whole course of the disease, and her temperature never reached normal. Herpes formed
on her upper lip. During the height of her fever she was delirious and her body was continually bathed in perspiration and she became greatly emaciated, refusing food. She refused Salvarsan. Liver enlarged and spirochaetae abundant there post-mortem.

Case No. 6.

Wife of store boy, Chiengi, N.E. Rhodesia.
Age about twenty-five years. Under partial observa-
tion during course of illness. Examined at Chiengi
Boma village on January 18th, 1914. "There is no
"history of a tick bite, but patient has just come
"from a journey from the north of Lake Bangweulu and
"is known to have slept at least at two rest huts
"which are known to be very badly infected with
"nkhufu", ten days to a fortnight previously.

On January 18th and 19th the temperature varied be-
tween 104.5° at night and 101° F. in the morning.
On the 20th the temperature was normal and patient
felt well though weak. During the attack she had
severe headache, diarrhoea and complained of great
pain in the back and thighs. Numerous spirochaetae
were found in several films examined on the evening
of the 18th. Profuse perspiration occurred at the

危机。The spleen was felt well below the costal
margin."
margin. I had to leave Chiengi on the 21st but heard from the N.C. that she had a second attack on the 26th with the same symptoms, and a third attack on the 8th February after which she remained well.

Case No. 7.

This was one of my own carriers. He gave a history of sleeping at the same rest huts as Case No. 6 on January 17th. These huts are well known to be infective, full of ticks. They are built after the Swahili pattern, square and with brick floors. On the 23rd he complained of great pain in back and limbs and severe headache, and vomited once. On the 24th the temperature was 104° in the evening. I took a blood smear and stained it. This I examined three days later and found three spirochoetes. On the 3rd of February patient had another attack at Kawambwa and was admitted to hospital, to which he had refused to go after recovering from the first attack. The course of the disease and results of blood examinations are seen on accompanying chart. This patient was also suffering from a septic wound of the hand.

Case No. 8.

Female aged twenty-seven years, wife of a policeman (N.R.P.) at Kawambwa. No history of tick bite/
bite, but many O. moubata found in huts she had slept in some days previously. Complains of great headache, and weakness and anorexia. Spleen enlarged and tender. Three blood films made, and one spirochoete found. Refused to come to hospital.

.4 grm. Neo Salvarsan injected intravenously. This patient gave a history of a previous attack of headache and bone ache eight days previously. No further rise of temperature. No other symptoms.

Case No.9.

"The wife of an askari travelling from Chiengi to Kawambwa. Age twenty-eight years. Gives a history of being bitten on the night of the 7th March while sleeping at the same rest huts as Case No.8. She looks distressed and complains of great frontal headache and body pains. Spleen not felt below costal margin. Came to dispensary to-day (14th). Examination of blood films reveals spirochaetes. Temperature on 17th. Patient left for her home while still under observation.

Case No. 10.

Patient was an askari who contracted the disease on the road between Chiengi and Abercorn to join his battalion (Congolese). He slept on the night of the/
the 23rd March at a village, recently deserted about fifteen miles from Chiengi. He arrived at Abercorn on the 31st and came straight to the dispensary to see me, complaining of great pain in head and eyes and a feeling as if he had been beaten severely all over his body. I found his temperature to register 104°F, and put him to hospital where he vomited twice. Spirochoetes were observed in blood smears taken that night and during the height of the fever of a subsequent relapse. On the 16th of April after the first relapse I injected 0.6 grms. intramuscularly. Patient then made an uninterrupted recovery.

Case No. 11.

This case ran a very similar course to Case No.10. The symptoms and history were the same in both, save that the illness in this case commenced a day later. They both belonged to the same detachment of soldiers. The temperature charts of these two cases are very similar and quite typical.

Case No. 12.

A young Cercopithecus monkey. Ticks caught in the Abercorn gaol were fed on this monkey which had been trapped the preceding day. Five days later spirochoetes were detected in its blood. They increased in number and were present in all smears examined up to the time of death. The temperature kept/
kept very high until the day before death when it fell to well below normal. The animal was greatly emaciated and refused food. Post mortem. The spleen was enlarged. The heart, liver and kidneys were pale and cloudy. The bone-marrow was dark-red and diffluent.
Case No. 13.

Tamba Tamba, native medical orderly at Taniramali, German East Africa, was seized with sudden severe pain in lumbar region and very severe pains in the head on February 5th, 1917. He had just arrived at my camp, having delivered a message to our Supply Base four days further south. He said that he had slept there at the neighbouring village in a hut which had been deserted for some time, and on the two nights of January 31st and February 1st had been bitten by "bugs". He vomited that evening twice, and on taking his temperature I found the thermometer registered 104.4° F. I made four blood films which I stained next morning, but did not find any parasites, only a certain degree of leucocytosis. The pain in head and back remained all next day and I noticed that the conjunctivae of both eyes were greatly congested. His temperature that evening was 97.8° F. Next morning he was distinctly better, and on the 8th was performing his usual duties. Suspecting the possibility of the symptoms being due to a spirochaete, I made a note of the case and kept a watch for a relapse. On the 16th, ten days later, he complained of headache and nothing else. His temperature was normal. He was given a dose of salts/
salts and later, aspirin grs. 5. I made a provisional diagnosis of tick fever, as the case was so suggestive of it. I was in charge of a field hospital at this time and met with no other.

Case No. 14.

sanswe, a Yao messenger, in my employ whilst acting as M.O. to the Anglo-Belgian Ecundary Commission at Moliwe, Belgian Congo, was attacked on the 10th January, 1912, with profound sickness, vomiting, and severe headache. On inquiry I found out that he had been sleeping in a Swahili hut in a neighbouring village. There was marked tenderness on deep pressure over the loins and thighs. The conjunctivae were injected. The tongue furred and moist. His temperature that evening was subnormal. I had as a routine, however, taken a couple of blood films and next day having stained them with Leishman's stain, discovered to my surprise several spirilla in one of them. Unfortunately I did not mount this slide. This man had a continuance of headache next day, but was able to resume work on the 13th. On the 21st he had a recrudescence of headache, its character being occipital and extending along the trapezius. This only lasted the one day. The treat-
treatment on both occasions was calomel grs. 4, followed by a dose of salts next morning. He had no further symptoms. I did not take full notes of the case at the time, having only been a short time in the country, but the fact of there being no fever, and finding the spirochoetes in a blood film stamped the case indelibly on my memory. I did not discover if any of the huts in the Village were tick infested.
An attempt to transmit Sp. duttoni by the bites of Clinocoris hemipterus, the bug of warm countries.

Having managed to trap and secure a young Cercopithecus monkey from the magistrate's garden, Abercorn, N.E. Rhodesia, I took several blood smears from it and found them free from parasites of any kind. Two days later I secured forty-six bugs (Clinocoris hemipterus), all of them being taken from the native gaol. Half of these bugs were applied direct to this monkey, and sixteen of them fed. The next day, (March 27th, 1915), I made twenty of the bugs which had not already fed on this monkey, take an "interrupted" feed on the monkey upon which I had already fed the ticks and which was suffering from Spirochetosis. The bugs - (seventeen fed) - having half filled themselves with blood, were immediately transferred to this monkey and most of them finished feeding. The monkey's blood was tested daily, several smears being stained, but no parasites were ever found, and the animal continued in apparent good health. Temperature varied from 98.8° to 99° F. On April 21st it was unfortunately killed by a dog just on the very morning I intended that it should be a host for ticks.

Post mortem. I found nothing remarkable in the various organs. I was led to make the experiment from/
from the account of Breinl's attempts to transmit
Sp. duttoni to monkeys by Clinocoris (Cimex) lectularius. He obtained a positive result in one
instance, but concluded that the technique was at fault because he had no more successes.

I think that further experiments are required before we can say with certainty that bugs
(C. lectularius or C. hemipterus) do not occasionally convey the infection.
Tick fever in Africa is pre-eminently a disease where 'causa sublata, tollitur effectus'.

**CURATIVE.**

Curative treatment may be discussed under three headings for convenience, as follows:-

(a) Symptomatic, (b) Tonic, and (c) Specific.

(a) **Symptomatic.**

Before Ehrlich's great discovery of the value of "606" in Syphilis and the subsequent use of it and other arylarsenates in the spirochaetal infections we had to rely on remedies which allayed the clinical manifestations of the disease without striking at the root of the trouble. The hope that atoxyl, which had been so useful against trypanosomiasis, would be of benefit in the spirochaetal and relapsing fevers, was proved to be without foundation. In many experiments no effect on either the disease or the parasites was observed.

Quinine has also proved of little value, but, considering that most of those who suffer from tick fever are malarial subjects, and the symptoms of the two conditions are very similar in the initial stage of the affection, a variable time being taken for diagnosis, I always give it to commence with, usually five grains in solution of the hydrobromide. As a routine/
routine I generally give a small dose of calomel, 2 to 3 grs. at the outset, followed in a few hours by a saline purgative. This may prevent some of the sickness and vomiting. During the paroxysm, to combat the severe headache and body pains I have generally found much relief has been obtained by the administration of aspirin in 10 gr. dose. Sometimes, however, the pain is so severe that nothing seems to relieve it except an injection of morphia hypodermically, $\frac{1}{2}$ grain usually being sufficient. The morphia always helps to allay vomiting, if present. Failing morphia, two or three drops of Tr. Opii, repeated, if necessary, is useful. The high temperature is best dealt with by sponging or the use of baths with the chill removed, under supervision. Delirium may be prevented by these measures, but should the patient become very restless and delirious, I have found morphia and bromide of service. The bromide is best given in a mixture containing a dose of fifteen grains along with a similar amount of spir. ammon. aromat. flavoured with orange. Heroin is most useful in cases where there are symptoms of chest trouble and gives relief at night in troublesome insomnia.

One must always be on the look-out for signs of iritis, and on the first indication of it, an injection/
injection of the deeper vessels of the sclera round
the cornea—even though the iris looks quite clear,
and a cursory examination reveals no immobility—
pain, lachrymation and photophobia, — a drop of
atropine should be instilled into the eye. This will
reveal slight adhesions. On ophthalmoscopic exam­
ination one may not be able to see the fundus on
account of turbidity of the vitreous or a deposit
of pigment on the lens. Salicylates have not been
found of any value in any of these symptoms. For
thirst and vomiting sips of cold water or a small
piece of ice to suck would be useful, if available.
If epigastric pain is very severe the application of
a mustard plaster or fomentation sprinkled with
Tr. opii gives relief.

During the febrile periods the diet should con­
sist of fluids only. The best are milk and soda
water, milk and barley water or milk and lime water.
If there are no complications during the apyrexial
period solid food may be allowed, and, indeed the
patients usually eat well. Cold applications to
the head may enhance the action of aspirin, and
chloral may be added with advantage to the bromide
mixture for insomnia, and for vomiting and malaise
I have found sips of champagne very useful, also an
effervescing ammon. carb. mixture. Collapse at
the/
the crisis is not common in the African form of relapsing fever, but, should it occur, it should be met by prompt stimulation with alcohol and strychnine, and the use of hot drinks, hot water bottles and blankets. The patient should be kept in bed in a well ventilated room, and his strength maintained, as in all specific fevers, by careful feeding.

High Temperature, per se, does not call for special treatment, and the other symptoms and complications are treated on general principles.

(b) TONIC.

Tonic treatment is carried out during the aphyrexic periods and convalescence. As in other debilitating diseases our greatest assets are fresh air, good feeding and cheerful surroundings. The patients usually feel so well during the periods of aphyrexia that there is no difficulty in getting them to go out of hospital and take exercise, - rather the other way about. Care should be taken that the patient does not over-exert himself then, for should the heart be affected, - (not a common complication, fortunately, as it is in the Indian form of relapsing fever) - syncope might follow. If the patient is at a low altitude it would be advisable to move him, if feasible, to one several hundred or a thousand feet higher. The drugs which we chiefly rely on are iron, arsenic, quinine and strychnine.
in judicious combination. In malarial districts quinine should be administered as a routine, since the chances are, especially in natives, or Europeans who have for long been resident, that they harbour the malarial germ in their blood. My native cases were always given the sulphate in five grain doses in solution daily. To Europeans I generally gave a mixture of the hydrobromide with hydrobromic acid. I only met with one case where quinine was badly tolerated and in that case euquinine, given in double the dose, as a powder, proved useful. When vomiting is a marked feature in the case the bihydrochloride is injected intramuscularly. Iron and arsenic are given in combination and the old Army pill No. 13 was very useful during the campaign, but these drugs are best given in solution and under supervision in a hospital, for it is difficult to get the patient to co-operate in drug-taking when he thinks he has recovered from the first attack. It is after the last relapse, many weeks after the commencement, that he really sees the need for tonics. Quinine and strychnine are also useful adjuncts to iron and arsenic and should be included in the prescription, especially if there is any tendency to bronchitic cough or praecordial distress.

The/
The electric current in the form of faradism or galvanism (weak currents) for facial and other paralysis, and massage have merits, employed during the period of convalescence.

I have found the following prescriptions beneficial:

**Tonic.**

R

- Ferri et Ammon. Cit.

Sig. \( \frac{3}{7} \) t.d.s.

**Palliative for Neuralgia.**

R

- Tinct. Gelsem.
- Aspirin
- Ammon. Brom.
- Caffein. Cit.
- Ac. Chloroform. ad

Sig. \( \frac{3}{7} \) s as directed. a.c.

**For injection into nerve sheath.**

R

- Beta-eucaine
- Alc. absol.
- Ac. dest. ad

Palliative/
Palliative measures in neuralgia include the administration of counter-irritation (e.g. mustard leaf behind the ear), or the local application of chloroform by cataphoresis. Externally, warmth, the most convenient method, in my opinion, being the flannel-covered hot water bottle. For iritis, besides heat, the application of atropinedrops (1%) along with *dicnin* (5%) should be tried.

I have also found benefit in the use of Donovan's solution, mainly, I think, because of the arsenic content.

Finally the tonic influence of the mineral and organic arsenical compounds must be mentioned, as it is very marked, in addition to their specific influence.

(c) SPECIFIC TREATMENT.

Ehrlich's discovery of the value of *Salvarsan* in the treatment of syphilis naturally suggested that this drug would prove of use in the other spirochete-caused diseases, among them the relapsing fevers. Fortunately this surmise has proved to be well founded. It was not so in the case of *atoxyl* and *soamin* which have no effect on African relapsing fever, though potent against trypanosomiasis. Nor has any benefit accrued from the employment of *mercury*.
mercury in any form save, perhaps, when combined with arsenic, as already mentioned, in Donovan's solution.

Serum-therapy, in the absence of a powerful specific remedy, might be, and has been used as a preventive inoculation, but chiefly in animals. Our knowledge of serum-therapy has chiefly been derived from the experiences of Novy and Knapp and Breinl and Kinghorn, who say:-

"Immune serum, whether derived from horses, monkeys or rats has no appreciable value in preventing the occurrence of the attacks in susceptible animals, or in curing the disease once contracted. The incubation period may be prolonged. Hyper-immune serum occasionally cuts short an attack, but does not prevent the occurrence of relapses."

We thus arrive at the discussion of the remedy which now constitutes our sheet-anchor in the treatment of this affection, namely, Salvarsan and its derivatives. In relapsing fever after the use of Salvarsan a patient can now leave hospital within a week, and the mortality has been reduced to nil. The drug nearly always aborts the attack in a few hours and cures the disease, subsequent relapses being very rare. According to Levaditi the most favourable time/
time for the administration of salvarsan is during the "precritical period" of the spirochaetal infection, when it causes a precocious crisis, thus enabling the organism to combat the infection by the usual processes - spirochaeticidal bodies and phagocytosis. The newer products, being less toxic and more soluble than "606" are preferable.

My own experience of the arylarsenocates has been gained in the intravenous injection of Neo-Salvarsan ("9014") and galyl. With these drugs I had great success and consider that, having them, the medical man practising in the tropics to-day, has a remedy the value of which cannot be over-estimated. But they must be given with caution and skill, after due examination of the patient's internal organs.

Practically all the newer preparations of arsenic act specifically on the spirochaete of tick fever. The following are some of the most powerful: Luargol, Kharsivan and Neo-kharsivan, Novarsenobillon, besides these already mentioned. They all are derived from the original dioxydiaminoarsenobenzol dihydrochloride, and may be given either intramuscularly or intravenously. Although in syphilis many observers believe that it is better to give them intramuscularly, because easier, and also not excreted so quickly, yet, I think, that in the case of/
of tick fever most medical men would prefer to give the preparation by the intravenous route. Moreover, the simplest method is to be recommended, for tick fever cases are usually met with a long way from hospitals and laboratories, and if the drug and apparatus for administering it can be made into a really portable form so much the better.

Hence the method advocated by Prof. Thibierge, of using concentrated solutions of ampoules (.6 grm. Neo-Salvarsan) is to be recommended. Because such a small amount of fluid is injected, ordinary sterile water, free from gross impurities can be used, and simplicity of administration is gained without loss of effect. As a knowledge of a simple method of administration is of so great importance and should be thoroughly understood, I do not think it out of place to describe in detail a method which appeals to me, - that of Major Lockhart and Captain Atkinson of the C.A.M. Corps. They use a 4 ccm. all-glass syringe with an accurately fitting plunger, no shoulder, and long barrel of moderate diameter so that it can be introduced at an acute angle with the skin. The needle is slightly larger and has a shorter point than the ordinary hypodermic needle. A burette (30 ccm.) is at hand in/
in a stand and contains sterile water. It is continued at the bottom by a short piece of glass tubing ending in a glass nozzle and closed by a pinchcock. An ampoule is flamed, filed, and 2 cc. water introduced. Then a piece of small glass tubing with rubber connection at one end and a few filaments of wool at a constriction near the centre, is inserted. The ampoule is then shaken gently and solution is complete in a few seconds. A syringe is picked out of a small basin of alcohol with dressing forceps, washed in sterile saline solution and the contents of the ampoule aspirated through the rubber connection to the glass tube; then a needle which has been boiled in a container is adjusted to the syringe. Meanwhile an orderly can be preparing the patient by fastening a light rubber tube round the upper arm and painting the bend of the elbow with iodine. The patient seats himself at a narrow table at the opposite side from the medical officer, resting his arm cut on the table. The medical officer steadies the skin with the left hand, introduces the needle into the vein and slightly withdraws the plunger to make sure that the needle is in the lumen, then, removing the tourniquet, completes the injection. The needle puncture is so small that no bleeding takes place usually, and only a few minutes are required for/
for each injection. Owing to the small size of the needle, pain is less than when the large needle required by the gravity apparatus is used and smaller veins may be successfully entered. In addition to the foregoing advantages of this method it is generally now believed that the administration in concentrated solution is more effective than when given diluted. One must not forget to test the urine beforehand.

Another valuable drug which has been used in protozoal diseases generally, with great effect, especially in leishmaniasis, bilharzia, espundia, oriental sore and kala azar, also sleeping sickness, is antimony, usually as the tartrate. McDonagh has used antimony in bilharzia since 1912. He gave it as tartar emetic (1 to 1 ½ grs. thrice weekly), antiluetin and colloidal antimony. Christopherson considers antimony tartrate a specific in all the above mentioned diseases, but warns us that great care must be taken in its administration. The maximum dose for an adult is 12 grs.; for a boy of ten years, 1 gr. He commences with half a grain to twenty minims distilled water, diluted with two volumes distilled water at time of use. The injection is repeated and the dose increased by ½ grain every other day until 2 grs. are reached and this is continued/
Castellani has also found this drug of great use in protozoal diseases. He used it specially in cases of relapsing fever in Macedonia and Corfu, both intramuscularly and intravenously, preferring the latter method. He injects a 1/2 solution of the tetrathionate in sterile saline solution, giving 2 to 10 cc. daily for five to ten days, then every other day or twice weekly. In the absence of a Salvarsan preparation and a medical men to administer it he recommends the following preparation:

R
Antim. Tartar.  gr. viij
Acid. Carbol.  m. x
Glycerin.  f. ivi
Sod. Bicarb.  gr. x
Ac. dest. ad 7
He injects 1/2 to 1 cc. every other day into the gluteal region intramuscularly.

One of our latest and apparently most valuable drugs is "J02" or luargol. Dr Danyez of the Pasteur Institute says: "The mineral or organic compounds of arsenic seem to have a common defect; the organism and parasites soon become accustomed to their presence so that the sterilization of the organism becomes difficult and sometimes impossible, and the apparent cure is sooner or later followed by a new outbreak. To overcome this difficulty the idea occurred to me to/
to combine the arsenical compounds with elements known to possess powerful action upon parasites to which the system does not show a tendency to become accustomed, notably the metallic salts."

Luargol is a composition of bromo-argentinc-antimony of arsenobenzol. To one molecule of arsenobenzol he added one of silver salt and one of antimony.

Experiments on animals have shown that luargol is more stable, more active, and relatively less toxic than arsenobenzol. It is ten times more active than arsenobenzol and 75 times more active than atcxyl in sleeping sickness. There is therefore not the same contraindication to its use as in "606" and the other preparations.

Although according to Murray and Row, disodoluargol has not proved very effective in the treatment of malaria, I have not yet heard of this drug being used in the treatment of the spirochaetal infections, but I intend to try it myself on the first opportunity.

In "Luargol" the antiseptic properties of Salvarsan are increased by the co-ordinated silver bromide, and these are reinforced by the specific action of the antimony. It is insoluble in water and rendered soluble by the addition of caustic soda and in some cases, in consequence of too high a content/
content of caustic soda in the solvent, it has given rise to indurations of the veins of the arm.

Neo Salvarsan has also been used with success, intravenously in Anthrax. Among the drugs containing quinquivalent arsenic, formerly used in sleeping sickness, syphilis and relapsing fever may be mentioned Arsacetin, Orsudan and Pectine. As the result of exhaustive trials on animals and numerous clinical tests, medical practice has now concentrated on the use of Salvarsan and its modern substitutes.

Treatment of Bites.

Prolonged bathing in hot water is recommended, followed by the application of a strong solution of bicarbonate of soda, which is allowed to dry upon the skin. For severe itching, smearing the bites with vaseline, which is slightly impregnated with camphor or menthol is advised. Should the bites become septic medical advice should be sought.

Continuing the work carried out in Ehrlich's laboratories, Kolle used various arsenical compounds in the treatment of spirochaetal diseases in rabbits. The new preparations were experimented with in inoculated rabbits from the viewpoint both of their toxicity and their therapeutic action. The chemo-
therapeutic quotient allows one to estimate the value of each medicament, abstraction being made of the difficulties in applying to man the experimental results obtained in the rabbit. The quotient is represented by a numerator: the smallest curative dose, and a denominator: the largest dose tolerated. It is all the smaller the greater the utilisability of the given preparation. The various products studied were Salvarsan, Neosalvarsan, Galyl (French), and finally Salvarsan-silver-sodium, which he thought excellent and which possesses the lowest quotient (1/25 - 1/30) and is readily soluble. It also offers a neutral reaction. He is to publish later the therapeutic results obtained in man with this product. He likewise announces a new product which he mysteriously designates Salvarsan "1495", but says its composition "cannot be revealed on account of the enemy". !

This product, "1495" is a Sulfoxylated product of Salvarsan-silver-sodium and has been found to be more active in action than the Salvarsan products on the spirochetes of recurrent fever. As it does not undergo any change in the state of solution, does not decompose when exposed to the air with the same rapidity, e.g. as Neo Salvarsan and no accidents from the product have been observed, it may render the/
the greatest service to armies and in the field generally.

As regards dosage, Kolle proposes 25 centigrams as a maximum dose of Salvarsan-silver as compared with 40 centigrams for Ehrlich's salvarsan. Gennerich has also obtained good results from the exhibition of Salvarsan-silver, and finds that it is less toxic and more efficacious than the other Salvarsan products. In secondary syphilis many cases became sero-negative in from $3\frac{1}{2}$ to 4 weeks.
PREVENTIVE TREATMENT.

This may be considered under two headings,
(A) Personal prophylaxis, and (B) General prophylaxis.

(A) Personal Prophylaxis.

The fact that the spirochaete (Sp. duttoni) has been proved to be conveyed to man by the bite of the tick, and possibly by bugs and lice, - (Sp. recurrentis, Sp. berbera and Sp. Carteri certainly) indicates that personal and domestic cleanliness and the avoidance of people and places infested with such vermin must form the basis of successful prophylaxis. The following rules should be observed: -

(a) Personal and domestic cleanliness. This goes without saying.

(b) Native huts must be avoided and not slept in. Specially anathema to the European are the Arab and Swahili houses. This was noted and remarked on by Livingstone in 1874. Dutton and Todd's observations confirmed his assertions. They said that the principal reason why ticks are found more often in Arab than in native houses is that the Arabs make better and drier buildings and live in permanent villages. Native villages are temporary affairs and slight causes may make a community leave their homes and build a village elsewhere.

(c)/
(c) The old caravan routes must be avoided as far as possible, and if one has to travel, neither European nor native rest-huts should be slept in; nor should the porters' loads be placed in them. I have found that in the rest-huts built by the District Commissioners in N.E. Rhodesia along the lines where Arabised villages are dotted, in a short time these huts became infested with *O. móbata*. This is no doubt because, when the D.C. or N.C. are not travelling, the huts are used constantly by natives returning to and from work in the Congo, for sleeping in. This is mainly noted along the old Arab routes.

(d) A mosquito net should be used always, and carefully searched for ticks, bugs, etc. last thing at night. These points were all noted by Livingstone long ago.

(e) The bed should be well off the ground and the legs of it should be smooth as the tick is said not to be able to crawl up a smooth surface. It should also be away from the wall, or a hammock should be used.

(f) A night-light for use at the bedside is recommended, as the nocturnal habits of *O. móbata* render the hours of sleep especially dangerous.

(g) Pyrethrum powder should be dusted between the sheets of the bed in localities where ticks abound. It/
It must be fresh. Iodoform powder, which is very useful for ridding one of fleas might also be tried.

(h) Natives should not be allowed to sleep in or near the quarters of Europeans, and in badly infested places should be made to wash their feet and change their clothes before entering a European dwelling.

B. General Prophylaxis.

The tick in question should be regularly destroyed.

(a) By searching for it, collecting them in bottles, the smooth sides of which they cannot climb. At Abercorn, N.E.R. I employed a small gang consisting of some of the native prisoners themselves, and in one morning was presented with two quart bottles full of the loathsome Argasidae of all sizes. It is best to make use of the accustomed native eye for searching, as the tick when disturbed simulates death, curling up its legs and remaining motionless, sometimes for hours, and is easily overlooked.

(b) Permanent buildings, e.g. European quarters, barracks, gaols should be disinfected by any or several of the following means:— (1) Scattering about as a powder, Sodium Fluoride which is very lethal to cockroaches and ants. (2) Scattering powdered napthalene on the floors—some pounds of it must be used. Then the room is closed up for 24 hours. Thus/
Thus one deals with the adults and larvae of fleas and there is no reason why one should not try this method in the case of ticks. (3) Like fleas, ticks do not like wet, therefore floors and walls should be thoroughly wetted, and contact made, with hot soapy solutions. (4) The walls may be sprayed with the following solutions: kerosene, crude oil emulsion, solution of vermijelli or cresol. (5) Cracks should be swept in the direction of the cracks and the sweepings burnt. (6) Kerosene or benzine may be put into the holes and cracks in walls etc. with a pipette. (7) As the tick, like the bug, lives mostly in cracks in walls and furniture, especially in the dust near the hearth, (and on bed-platforms, cracks in door-sills, crevices in mud walls or in grass walls and in the thatched roofs in native houses), various fumigation methods, which are also applicable to other vermin may be used. These are sulphur dioxide, one pound per 1000 cubic feet, camphor and phenol, equal parts - four ounces per 1000 cubic feet, fresh pyrethrum powder - five ounces per 1000 cubic feet. When any of these substances is used, it is necessary that the exposure be as long as possible, and it is further desirable that the vermin — many of which are merely stunned — should be promptly swept up and burnt.

Hydrocyanic acid is lethal, but is too dangerous for practice.
An iron roof is to be preferred in a tick infested locality to a thatch.

(c) Old camping-grounds, rest-houses and native huts, when infested, should be burnt. Natives frequently smoke their huts to drive the ticks from their lodgement in the thatch, but this is not eradicating them.

(d) Use of cow-dung and mud. The Boers and several cow-keeping native tribes plaster the walls and floors of their houses with this mixture, but in my experience this is also merely a temporary measure, as cracks soon form again when the plaster dries. I may mention that I tried the same plan, using a mixture of native salt and mud, equal parts, but found only transient benefit from it.

(e) Native soldiers, porters, servants, labourers and other controllable bodies of natives should be compelled to observe regulations regarding regular bathing and washing of clothes, and made to keep their houses clean.

Dutton and Todd write as follows:— "Ticks seem to have come into the Free State by two routes; from the East Coast with the Arabs, and with traders from Portugese territory to the south. The rivers are the present highways. Old and present caravan routes used by Europeans, along which they are known to occur, are/
are indicated by dotted lines on the map. A glance shows that ticks are found, particularly, along much travelled roads. They are carried commonly in porters' loads, but how easily they may be carried in even a European's luggage is well shown by our experience on leaving Nyangwe, where we were well lodged in well-built houses. The rest-houses are always the most infested." With the foregoing statements I cordially agree in the light of my own experience in N. Rhodesia and East Africa.

A word as to O. savignyi, which is more diurnal in its habits than O. moubata, and seems to have a predilection for market-places, cattle-stands, wells, etc., and by means of which Erumpent has succeeded in transmitting tick fever. It may commonly transmit African relapsing fever as the natives dread its bite. Drake-Brockman says of it, "That tick is found in the soil in or around the huts on outskirts of coastal towns in the more squalid areas. It frequents most camps of long-standing which are inhabited by human beings and their domestic animals. They are extremely common in the dusty soil surrounding the wells and water-holes. They are often found under large trees, especially when these are in isolated positions and afford the only shade for miles to the shepherd and his/
"his flock during the heat of the day.  O. savignyi is capable of living for months without a fill of blood in the soil, into which it burrows for a depth of \( \frac{1}{2} \) to 1 inch, lying dormant there until the ground is disturbed by the foot of man or beast. It will attack human beings, camels, cattle, mules, donkeys, sheep and goats with equal vigour. It seldom climbs much higher than the ankles in human beings and the hocks in animals."

He says that the best method of destroying these ticks when they swarm in confined areas is to cover the whole of the infested area with dry grass and brushwood, after harrowing the surface, and then set fire to the grass all round simultaneously, so that the fire will gradually burn its way towards the centre. Native soldiers should rub their feet and ankles with turpentine when entering localities where this tick is known to occur and to be infected with the spirochete of relapsing fever.

In Persia similar prophylactic measures will have to be taken against O. tholozani.

Fantham, writing regarding Sp. recurrentis (var. berbera) says, "Infection by way of the eye is quite probable in Africa, remembering the constant trouble due to sand, dust, insects, etc., resulting in frequent touching of the eyes." This is just as likely, in my opinion, in the case of Sp. duttoni, and/
and emphasizes the importance of early diagnosis and treatment of all eye affections.

A case of tick bite of the conjunctiva is recorded by Kulerich. A young girl complained of a foreign body which had settled on the right eye the day before. A subconjunctival effusion of blood was found on the right side of the eyeball. In the centre of this effusion a red, round body of the size of a pin's head, was perched. With considerable difficulty it was detached by forceps, and proved to be the six-footed larval stage of a tick. Probably the tick had settled on the patient's right hand and had been brushed off on to her eye in the act of rubbing it.

Sargent, working in Algeria, has shown that the spirochoete of relapsing fever is well able to pass through the unbroken moist membranes of the body such as the eye and the inside of the nose.

The dust of dried droppings of lice kept for four months has produced trench fever by being introduced into skin wounds and equally well by being blown on to the eyeball.

The shaking of a blanket used by a lousy patient may therefore be dangerous.

What applies to the droppings of the louse may also apply to the droppings of the Argasine ticks which are the cause of African tick fever.
Mallavan advocates the use of tobacco as a pulicide, and has carried out very successful experiments in the City of Hyderabad.

His reasons for doing so are as follows:

1. Tobacco kills fleas almost instantaneously.
2. Being a diffuxible drug its action is effective even at a distance of six inches.
3. Its action is permanent and continuous, acting as a disinfectant day and night when kept spread out on floors.
4. The same tobacco can be used up over and over again, its substance not being used up in destroying fleas.
5. It does not damp or mess the floors of houses.
6. Being used in a dry state, no caste can object to its use.
7. It is common and cheap.
8. Reinfection of houses can be prevented as long as tobacco is kept in the house.

I mention this method of disinfection as being very clean and easy of application and suggest that it might be given a trial in the case of tick-infested native gaols, barracks, houses, etc.

The most satisfactory method of tobacccing houses is that of stitching the leaves on to a piece of cloth/
cloth like a strip of matting, which is then laid on the floor. Powdered tobacco should be introduced into holes.

Blacklock at Runcorn has tested various sheep-dips and found that they frequently failed to prevent *O. mcubata* from feeding on animals, and in vitro its resistance to these substances is marked. More lethal effects might be produced by gases.

Philip Ross says that in his experiments with ticks he failed to produce infection in British East Africa and thinks the explanation lies in the lowness of the temperature. "If the spirochaete does not develop in the tick at 18 to 20°C., it is probable that the tick brought from the moist warmth of Uganda to the cool days and cold nights of Nairobi ceases to be infective". My cases in Abercorn were infected during the end of the cold weather at an altitude of 5,500 feet, where the days are cool and the nights as cold, and perhaps colder than in Nairobi. It would therefore appear that under natural conditions the tick can quite well convey the disease at fairly low temperatures as exampled by my own cases occurring on the plateau of Northern Rhodesia. No prophylaxis can therefore be expected to follow removal to a high altitude.

Of natural enemies, like other insects (sensu magna)
magna), the tick has not a few. They are devoured by chickens, rats and mice, and ants carry off young ticks and eggs.

Wellman in 1905 wrote that he had found a bug, the "Ochindundu" which Austen identified as *Phonergates bicoloripes* belonging to the family Reduviidae, devouring an *Ornithodoros moubata*. He writes: "It may prove to be an unpaid assistant to the tropical sanitarian, helping him in the laudable task of combating the worst of all African vermin, which infects not only native lines, but is sometimes even found in white quarters." But Austen pointed out that the Reduviidae prey on insects of all kinds. We cannot include the "unpaid assistant", then, in our scheme of tropical sanitation, as it is extremely unlikely it especially preys on *O. moubata*.

Wellman also states that the natives think a marked degree of local inflammation arising from the bite of the tick has a certain protective influence against a general infection. I made many inquiries from time to time among the natives of the various tribes of N.E. Rhodesia, Nyasaland and German East Africa, but could not get this confirmed.

Moffat records the case of a European who suffered from an attack of tick fever with five relapses. After an interval of 2½ months he was exposed to a fresh/
fresh infection. A month later he had a sharp attack of fever lasting three days and with a scanty show of spirochaetes in the blood. After 19 days he had a relapse with spirochaetes in the peripheral blood, and no further relapse. The irregular course and long period of immunity was no doubt due to partial immunity conferred by the first infection. The mild course of one of my own European cases (No.3) bears out this theory of immunity. Among natives it is common to have only one attack and no relapse, and the disease is so prevalent that we may assume they have had it before.
SUMMARY AND CONCLUSIONS.

From the foregoing facts as set forth in this thesis we conclude that:

(1) African "Tick Fever" is conveyed by an Argasine tick, Ornithodoros moubata, naturally, with certainty; probably by O. savignyi; possibly by bugs; and that temperature has little effect on the powers of the tick to convey the infection.

(2) The disease is commoner in Central Africa than has hitherto been supposed and in consequence of our increased knowledge of the disease and its conveyance, many of the cases which were formerly diagnosed as malaria are now rightly ascribed to infection with the spirochaete. This knowledge has been, and will be gained by frequent use of the microscope and the cultivation of greater skill in the use of stains and dark ground illumination.

(3) A considerable degree of immunity can be acquired. This is made manifest by accounts of cases in the literature, by some of my own native cases, and also European Case No. 3.

(4)/
(4) The disease presents symptoms of all degrees of severity which may depend on one or more modified forms of Sp. duttoni; the differentiation of a spirochoete showing different cultural and morphological characteristics from Sp. duttoni tends to confirm the opinion that there is a severe and a mild form due to a different variety of spirochoete.

(5) Among the symptoms of the disease, those which are manifestations of disease affecting the nervous system are prominent, viz., severity of the headache, eye symptoms, inco-ordination, neuralgias and paralyses, especially facial paralysis, tending to persist as a sequela in some cases, long after the disease has run its course.

(6) It is essentially a preventable disease, and much can be done to prevent its occurrence by enforced sanitary measures. The benefit of personal prophylaxis, especially the care of the eyes should be borne in mind.

(7) In Salvarsan and the other compounds of arsenic, and, perhaps to a lesser extent, in the antimony compounds, we have a true specific remedy for the/
the disease. Of particular importance is the recent product \textit{Salvarsan-Silver Sodium}, and more especially "1495".

(6) By simplicity and portability of apparatus, and use of these drugs in greater concentration, the remedy is now available for all, and in the most remote areas.
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APPENDIX.

MAP, CHARTS AND DRAWINGS.
THE MEDICAL SUPPLY ASSOCIATION.

NOTES OF CASE.

NAME: Dickinson
Age: 33 years

Case Book No.

R. Aspirin: gr. vi
Caffeine gtt.

Resins, etc.

Nov. 28th

Date of admission: Oct. 1944

Result

Temperature (Fahrenheit)

97° 98° 99° 100° 101° 102° 103° 104° 105° 106° 107° 108°

97° 98° 99° 100° 101° 102° 103° 104° 105° 106° 107° 108°

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Resp. 25 29 30 31 23 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38

Date. 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17

Entered at Stationers' Hall

Printed and published by Wodderspoon & Co. 6, Gate Street, Kingsway, W.C. 2

Gould's Clinical Chart.
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Notes of Case:
- Initial symptoms: Abdominal pain
- Treatment: Bed rest
- Progress: Improved after treatment

Case Book No.
- Mr. Smith
- Mrs.Jones
- Dr. Brown

Entered at St Thomas Hall
Notes of Case.

Name: Arthur C. 
Age: 41 yrs

Casebook No.

Date of admission: Sept. 18, 1914

Result

Entered at Stationer's Hall

Printed and Published by Wodderspoon & Co. 6, Gatre Street, King'sway, W.C. 2

Gould's Clinical Chart
DISEASE: African Typhus Fever

Notes of Case:

Name: Samuel
Age: 44 yrs

Case Book No.

Temperature (Fahrenheit)

Normal Temperature of Body

Result

Date of admission: 18th Sept. 1914

Entered at Stationers Hall

Printed and Published by Woodruff & Co. 66 Gate Street Kingsway W.C. 2.

Gould's Clinical Chart.
**DISEASE:**

African Tick Fever

**Notes of Case:**

**Name:** Dandano

**Age:** 44 yr

**Sex:** M

**Case Book No.:**

**Result:**

**Date:** Sept. 9, 1914

**Temperature:**

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<td><strong>Normal Temperature of Body:</strong> 98°</td>
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THE MEDICAL SUPPLY ASSOCIATION.

DISEASE

Notes of Case.

Name: C. K. Kimbrough
Age: 44 years
Diet
Case Book No.

Miss A. W. McLean 3 years

Result

Date of admission: 26 Nov. 1914

Entered at Stationers Hall.
**Notes of Case.**

**Name:** John Thompson  
**Age:** 31 years  
**Sex:** Male

*Patient admitted to hospital on commencement of joint pains on March 20th, 1915.*  
**Also suffering from acute wound of thumb.*

**R. A. Browne.**  
**Penultimate of note:**  
**24th - Joint above was incised to cure the ulceration of the skin.**

**Date of admission:** March 20, 1915

**Result**

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**Temperature Chart:**

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**Notes of Case.**

*Name:* James Poulston  
*Age:* 3 1/2 years  
*Case No.*  

*Diagnosis:* African Tick Fever  
*Temperature:* 
- Normal body temperature: 98°F  
- Day of illness: 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50

**Results:**  
- Pulse: 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27

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*Entered at Stationers Hall*  
Printed and Published by Wodderspoon & Co. 6, Gate Street, Kingsway, W.C. 2  
Gould's Clinical Chart.
**Notes of Case**

**Name:** Alex Thomson  
**Age:** 38 yrs. 

- **Disease:** Chronic Liver Disease
- **Result:**
  - 99.6°F
  - 99.7°F
  - 99.8°F
  - 99.9°F
  - 100°F
  - 100.1°F
  - 100.2°F
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**Date of Admission:** 16 Dec. 14

**Result:**
- **Day of Dis:** 22, 23, 24, 25, 26, 27, 28, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107.
- **Pulse:** 110, 108, 106, 104, 102, 100, 98, 96, 94, 92, 90, 88, 86, 84, 82, 80, 78, 76, 74, 72, 70, 68, 66, 64, 62, 60, 58, 56, 54, 52, 50, 48, 46, 44, 42, 40, 38, 36, 34, 32, 30, 28, 26, 24, 22, 20, 18, 16, 14, 12, 10, 8, 6, 4, 2, 0.
- **Date:** 16 Dec. 14
<table>
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<tr>
<th>Date</th>
<th>Time</th>
<th>Temperature (Fahrenheit)</th>
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</table>

**Notes of Case:**

- Blood exam - no spirochaete found before treatment.
DISEASE: Cholera.

Notes of Case:

Name: E. McGregor
Age: 38 yr.

Diet: [Blank]
Case Book No. [Blank]

Date of admission: 16 Sept. 14
Result: Case

Temperature (Fahrenheit):
97°, 98°, 99°, 100°, 101°, 102°, 103°, 104°, 105°, 106°, 107°

Time:

Bowels: M M M M M M M M M M M M M M M M M M
Urine: M M M M M M M M M M M M M M M M M M

Result of admission: Died.
THE MEDICAL SUPPLY ASSOCIATION.

**Disease.**
Tick Fever

**Notes of Case.**
Typhus Fever

**Name.**

**Age.** 30 yr

**Diet.**

**Case Book No.**

**Date of admission.**
24 Jan., 14

**Result.**
Care

**Temperature (Fahrenheit).**

<table>
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<th>Day of Dis.</th>
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</table>

Entered at Stephens Hall.

Printed and Published by Wodderspoon & Co., 66, Gower Street, Kingsway, W.C. 2.

Gould's Clinical Chart.
**Disease:** African tilt fever

**Notes of Case.**

- **Name:** Manuelga
- **Age:** 30
- **Diet:**
- **Case Book No.:**

**Wife of Manuelga, hospital order:**

- **Dr. Smyth, 69, m.s. gynecologically.
- **Dr. West, D.C.H., Ch.
- **Dr. Green, V. K.

**Date of admission:** 21st May 1915

**Result:** Death
<table>
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<th>Time</th>
<th>Temperature (Fahrenheit)</th>
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</table>

**Notes of Case**

Name: Lucy
Age: 24

**Date of admission:** 1st Oct. 1914

**Result:**
Notes of Case

Name
Age
Sex

Case Book No.

Disease

Date of admission
19th Oct 1914

Result

Entered at Stationers Hall

Gould's Clinical Chart.
Notes of Case.
Name: John Brown
Age: 17 years
Diet:
Case Book No.

Date of admission: 1st Jan. 1914
Result

Entered at Stationers Hall.
Printed and Published by Wodderspoon & Co., 6 Gate Street, Highbury, W.C. 2.
Gould's Clinical Chart.
Complications

Occasional Notes and Prescriptions (To be copied into the General Report)

After being bed on Monday on 20th March
Fourteen " " " " 21st"

Shivered first seen in that film on 23rd March thereafter all through the infection.
Died early morning, 3rd April.

P.M.
All organs examined.
Spleen slightly enlarged back, liver & kidney show cloudy swelling. Bone marrow dark & stiffer.