ought or ought not to be removed. The microscope there and then showed that only the large glands upon the capsule of the tumour contained growth, and that those nearer the crural arch contained none, so that it was deemed unnecessary to open the abdomen.

Mr. Shaw will now demonstrate how quickly sections of growth can be cut with a freezing microtome, and how clear and good the sections are. In my opinion the apparatus he is using is an essential part of the equipment of an operating theatre.

DEMONSTRATION BY ERNEST H. SHAW.

Method of Cutting Frozen Sections of Fresh Tissues for Immediate Microscopic Diagnosis During Operations.

The method may be divided into two parts:

1. *The Arranging and Fixing-up of the Apparatus Required.*

The microtome must be fixed on a firm table, and all the instruments arranged in a convenient manner. A mental survey of the cutting, mounting, and staining of a section is then made, in order to make sure that everything is present and in its proper place. This ensures that no time will be wasted when once the process is begun.

2. *Preparation of the Microscopic Section.*

(a) The selected piece of tissue received from the surgeon is placed directly on to the brass disc of an ether-freezing microtome, and is surrounded by gum solution.

(b) The tissue and gum are frozen, and sections made by a razor on a carrier.

(c) The sections are transferred to a dish of cold water, and, after separating them with a glass rod, a suitable section is lifted out.

(d) It is dipped for a moment into pure methylated spirit, and

(e) Then placed in another larger dish of cold water; the currents set up by the spirit in the water cause the section to spread out flat.

(f) A glass slide is dipped in the water under the section, and the latter is lifted out as the slide is slowly drawn up out of the water again.

(g) The water is drained off the slide, and a drop or two of stain (Löffler's methylene blue) is allowed to fall directly on to the section.

(h) A thin cover-glass is placed on the stain and section after three to five seconds; it is lightly pressed down so as to drive out excess of stain; this is then blotted off, and the specimen is ready for examination under the microscope.

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THE ETIOLOGY AND PROPHYLAXIS OF DRACONTIASIS.

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DRACONTIASIS is very common and widely disseminated throughout the native races of our West African possessions. During the last five years it has twice assumed epidemic proportions amongst the black troops stationed there, and on the more recent occasion severely hampered military operations in Nigeria.¹ These outbreaks indicate how, by aggravating the difficulty of maintaining the necessary supplies of labour, the disease may materially retard the commercial development of these colonies in the future. The authorities are at present deterred from initiating any active prophylactic measures by the varied and conflicting nature of the theories now in vogue as to the life-history of the parasite, and the manner in which it gains access to the human body.

In order to investigate these points in more detail, and, if possible, to determine the means by which the disease might be mitigated or controlled, I was sent to West Africa in August, 1905, by the Committee of the London School of Tropical Medicine.

The aim of the present paper is, first, to record the

results of experiments undertaken to test the truth of previous theories; secondly, to define the conditions essential to the complete development of the parasite; and, thirdly, to base upon the facts ascertained some suggestions for the prevention of the spread of the disease.

The hypotheses of infection that have been advocated from time to time may be classified as follows:

I. Those in which the development of the embryo is supposed to occur without the intervention of any intermediate host, human infection being caused by:

a. The embryo, as discharged from the parent worm; or

b. The mature larva, evolved from the embryo in water or marshy soil; or

c. The young adult, the product of the continued growth of the larva in water.

II. Those in which an intermediate host is considered essential for the development of the larva in order that it may become fitted to reinfect man:

d. The only, and in itself sufficient, host being cyclops.

e. A second, and at present unknown, intermediate host being necessary to continue and complete the changes begun in cyclops.

Further, there remains to be considered under each heading a still more controversial and important question: By what route does the parasite enter the human body? Does it do so passively with the ingestion of fluid or actively by penetrating the skin?

The theories comprised in Section I have been strongly advocated by many writers on dracontiasis in the past. They are still to be found in some of our modern textbooks of pathology, though they have become discredited. Plehn² is the most recent investigator to support the theory (a) of direct infection by the embryo. In a tumour of the right thigh of a monkey which had been fed eight months previously on bananas containing freshly discharged embryos he found a filaria "indistinguishable from the Medina-worm." This experiment I repeated in Accra, but though the monkey swallowed thousands of living embryos, not a sign of infection could be discovered *post mortem* six months afterwards. Busk³ has maintained on clinical evidence that the embryos directly penetrate the skin, but I am unable to support this view. The application of living embryos in water to the dorsum of my own hand always failed to produce the erythema and itching noted by Looss to accompany skin infection by nematode larvae, and there have as yet been no other indications of infection.

The remaining hypotheses (b and c) of this section may be rejected from a critical examination of their literature. It is obvious both from the descriptions and illustrations that the larvae and adults of certain free-living nematodes have been very generally mistaken for the developing Guinea-worm. The same explanation disposes of the statement (Carter) that *Urolabes palustris* is the free form of the *Filaria medinensis*.⁴

It has frequently been shown that the embryos discharged from the parent can only live in water for quite a short time, and that they undergo no metamorphosis. In my observations on the vitality of these embryos in water the vast majority died on the third day, though some survived until the sixth. In mud they lived a day or two longer, probably because in this they move more slowly, and are consequently less quickly exhausted. Although provided with a mouth and digestive tract they are still unable to obtain food for themselves.

This inability on the part of the embryo to infect man by the skin or by the mouth, to undergo any further development, or even to remain alive in water, points to an intermediate host as an essential factor in its life-cycle.

This conclusion brings us to the consideration of the theories in Section II. The conditions under which the embryo becomes a larva were discovered by Fedchenko about 1870.⁵ He observed that the embryo made its way into the body cavity of a small crustacean of the genus Cyclops, and there, after a period of twelve days, underwent metamorphosis. Having followed the growth of the larva until the fourth week, he then endeavoured to complete the life-cycle of the parasite by feeding cats and dogs upon the infected cyclops. These experiments were unsuccessful. Nevertheless, Fedchenko concluded that

cyclops was undoubtedly the efficient intermediate host of the Guinea-worm, and that probably the larvae were taken into the alimentary canal of man whilst still within this host, and there set free. His view was that in the intestine they then attained sexual maturity, and the females when fertilized made their way into the connective tissues, whilst the males died and were discharged in the faeces.²

The failure of Fedschenko's efforts to produce infection artificially led many to doubt the accuracy of his conclusions. It has been urged that "possibly cyclops is not the real host of the *Filaria medinensis*, notwithstanding that the nematode might attain a certain stage of development in its body" (Sambon³). Others maintain, upon the analogy of the behaviour of the ankylostome larva, that the larvae by becoming free-living later, may infect man through the skin. They are thus able to offer an explanation for the remarkable fact that the parasite occurs in the lower limbs in 90 per cent. of all the cases of dracontiasis. While my own experience does not confirm Fedschenko's observations and conclusions in their entirety it brings no support to these later views.

I obtained the materials for experimental purposes most satisfactorily by the following means. Cases of dracontiasis were selected in which the vesicle caused by the worm was still whole or had only recently burst, and a damp compress was bound over the sore. When this was taken off, a few hours later, there would be found upon it an almost solid mass of embryos. With material obtained in this way one had complete control over the degree of infection of water containing the cyclops. The worm discharged its young by a prolapse of the uterus, as described by Manson,¹⁰ though this extrusion never occurred through the mouth but by an opening just outside the circumoral ring of papillae.¹²

Cases in which there was considerable suppuration or abscess formation were useless for our purpose, the accessible part of the worm being filled by white cells, which had destroyed the embryos. To secure a supply of cyclops, likely pools were swept with a fine silk net (60 meshes to the linear inch) into the apex of which had been fitted a wide-mouthed bottle of about 10 oz. capacity. When this bottle was filled with creatures of various sorts it was emptied into large jars, which were then corked and transferred to the temporary laboratory. After a time, possibly because the oxygen became exhausted, the cyclops collected near the surface. The upper layer was then decanted into a large Petri capsule and gently "swirled" round until the solid contents gathered in a vortex in the centre. By means of a large syringe with a fine nozzle all the dirt and the innumerable small creatures could now be got rid of—except the cyclops, which always darted away from the current into the clean water at the sides. In this way it was easy to get a large and fairly pure supply from the most miscellaneous catch.

Although most of the animals that prey upon cyclops were excluded by this method, there was some difficulty still owing to the evaporation and concentration of the water, and the growth in it of paramecia which destroyed large numbers. The bulk of the water had consequently to be drawn off and replaced by fresh every third day. Moreover, the cyclops themselves are carnivorous, and in absence of their usual food supply consume their fellows, so that it became necessary to add some organism that was suitable as food and yet harmless. The best results followed the use of rain water, which contained numerous nauplii and cladocera, to refill the tanks.

Of all the other organisms found in the ponds none was capable of infection with Guinea-worm embryos. In a few instances these were swallowed, especially by mosquito larvae, but they were invariably digested and their cuticles evacuated. Cyclops, on the other hand, was very susceptible, no resistance being met with even in adult forms carrying brood capsules. This fact provides a strong argument against the suggestion of Graham⁷ that the greater incidence of the disease in May and June is due to the prevalence in these months of young forms of cyclops, which he supposes are much more liable to infection.

I do not propose to give any account in this paper of the changes undergone by the larva in becoming mature. Such embryological details may be more fittingly considered later in connexion with the anatomy of the adult worm.

There are, however, two episodes in the embryo's life that are of sufficient practical interest to be dealt with here, and regarding which I find myself at variance with previous observers, namely, the mode of its entry into cyclops, and the time required for the completion of metamorphosis. The teaching of the present day is that the embryo bores its way through the integument at the intervals between the "ventral plates," and so directly reaches the body cavity. I was never able, in spite of repeated endeavours, to observe this process, and there are several considerations which make it somewhat improbable. The cuticle of cyclops is continuous and almost uniform. There are no distinct "ventral" plates or intervals between them. In fact, as may be seen after the soft parts of a dead cyclops have been completely cleared away by paramecia, the cuticle is a rigid and uninterrupted exoskeleton. The Guinea-worm embryo possesses no boring apparatus with which to pierce this resistant layer. Its movements, moreover, intermit regularly, and are insufficiently powerful to maintain its own weight against gravity. For it, the presence of a cyclops in its neighbourhood is a matter of indifference—a strong contrast to the swarming of the miracidia of trematodes around their special hosts. If by chance an embryo does come in contact with one of the cyclops's appendages it loops round it in the form of a Greek α, but it has no means of firmly affixing itself and quickly relaxes again. The coiling appears to me to be essentially spasmodic. It may be produced by salt solution and other chemical irritants. This peculiar flexion followed by sudden straightening, while useless for penetrating a resisting object from without, seems very well calculated to help the embryo to explode one or other of its extremities through the walls of a soft containing tube like the cyclops's intestine. Further, where more than half a dozen embryos gained access to the body cavity, the infection generally resulted in the death of the host. In these cases it could be seen that the intestinal wall at one part was severely lacerated. Lastly, we have the analogy of *Cucullanus elegans*, the similarity of the embryos of which to those of the Guinea-worm suggested to Leuckart⁸ the possibility that the cyclops was an efficient host for both. Although the *Cucullanus* embryo is provided with a special "perforating tooth" (Blanchard⁹), it is an accepted fact that it infects cyclops through the intestine. The question can only be settled finally by serial sections of recently-infected specimens, and these I have hitherto had no opportunity of making. Meanwhile it is, I venture to submit, at least an open one.

As regards the time necessary for the completion of metamorphosis, Fedschenko, who continued his observations into the fourth week after infection, states that it occurs on the twelfth day. Manson,¹⁰ working in London with English cyclops, found that ecdysis was postponed until the sixth week, owing, doubtless, to the cold. In Africa I found that the striated cuticle of the embryo was cast generally on the eighth day. The larva which emerged lost, two days later, a very delicate enveloping pellicle, and from that time onwards underwent no further ecdysis. The subsequent changes were confined to the differentiation of internal structures, the larva apparently becoming mature in the fifth week.

The next point to be considered is the way in which the mature larvae quit the cyclops. If infection of man is to occur by the skin, it is clear that they must leave the cyclops and become free-swimming. If, on the other hand, infection occurs by the mouth, there is no necessity for them to do so till they arrive in the human stomach. Now, in the six weeks during which I was able to observe them developing, the larvae showed no inclination to leave the cyclops. On the contrary, as time went on they became more quiescent, and even when, as happened only too often in the later stages of the experiments, the cyclops died, they did not escape, but were found dead in its interior.

The manner in which they can be set free I have already described in a previous communication.¹¹ When an infected cyclops was transferred to a 0.2 per cent. solution of hydrochloric acid it was immediately killed, but the larvae, far from being destroyed, were awakened to great activity, and eventually escaped into the fluid, in which they swam about freely. This experiment pointed clearly to the probable way in which the larvae are released in nature, for

if such a cyclops were swallowed the action of the gastric juice would give the same result.

In order to clinch the argument, I fed a monkey on bananas containing cyclops which had been infected for five weeks and which had in them larvae which appeared mature. Six months later a careful *post-mortem* examination was made for me by Dr. Daniels, and in the connective tissues five filariae were found which, as I have stated elsewhere, possessed the anatomical characteristics of *Filaria medinensis*.¹² There were three unimpregnated and obviously immature females about 30 cm. long, and two remarkable small males (22 mm.), which were obtained one from the psoas muscle and the other from the connective tissue behind the oesophagus.

These results point strongly to the truth of the theory that infection of man takes place from the drinking of water containing infected cyclops.

The suggestion⁸ that a second intermediary host is necessary for the complete development of the Guinea-worm larva is disposed of by the fact that this is actually attained in cyclops.

The finding of both male and immature female forms in the connective tissues relieves us of the more improbable alternatives previously open to us,⁹ namely, that either the filaria was hermaphrodite or that the larvae became sexually mature in the intestines, and the fertilized females only migrated into the body. The view that the larvae at once make their way through the gut wall and become sexually differentiated later in the tissues of their host, brings the after-development of the parasite much more into line with that of other filariae.

We have already referred to the extraordinary frequency of the parasite in the lower limbs in human cases, and discussed the possibility of the larva directly reaching its final position in the connective tissue by piercing the overlying skin. Those who reject this explanation suppose that the pregnant worm is attracted to these parts by a maternal "instinct" which bids her seek to discharge her young into water.¹ In the case of our monkey, however, the three female worms, although immature and unfertilized, had already made their way into the limbs, being found *post mortem* in the forearm, the axilla, and the popliteal space. I venture to suggest that in these wanderings of the Guinea-worm we have an example of one of those "tropisms" that, as Loeb has recently shown,¹⁴ dominate the movements of many lowly-organized creatures. Geotropism¹³ seems to me to afford a rational explanation of the remarkable distribution of the parasite in man.

Two further statements must be briefly dealt with before we can proceed to summarize the circumstances controlling the complete development of the parasite. It has been repeatedly affirmed that the embryos as they come from the patient are able to withstand desiccation,¹⁰ and thus be spread widecast by the wind, or in the dry season await a favourable rain to carry them to a cyclops-infested pool. Were this true it would present an almost insuperable difficulty to the prevention of the spread of dracontiasis. The embryos are, however, immediately killed if dried by natural evaporation, and they cannot be revived by the readdition of water.

The presence of numerous large lagoons along the Guinea coast has been thought to explain the great prevalence of the disease in that part of Africa. At Accra there are two lagoons, but neither contains cyclops. The water was distinctly saltish. This excludes them as a source of infection, for on the one hand the species of Cyclopidae are, with one or two rare exceptions, freshwater forms, and, on the other, water taken from these sources was found to kill the Guinea-worm embryos in half an hour. Sea water had the same effect.

We are now in a position to define with some accuracy the conditions essential for the completion of the life cycle of the parasite.

The young must be discharged directly into fresh water soon after the parent worm has succeeded in creating a break in the overlying skin and before the wound has become markedly septic. The embryos must find a cyclops within a few days. They must, moreover, succeed in entering its body cavity. Five weeks later they will have developed into mature larvae. These must, thereafter, be taken into the human stomach, and having been set free from its host by the gastric juice, reach the connective tissues by penetrating the gut wall.

The life-cycle of the parasite will necessarily be broken: (1) By the death of the embryos, either from sepsis while still within the parent worm, or after their discharge, by saltish water or drying. (2) If cyclops are not present in the water, or if the infected cyclops die or are not taken into the human stomach. (3) If the larvae, ingested by the final host, are immature or fail to escape from the chitinous sheath of the cyclops. Though they do gain their final habitat, the cycle will still be incomplete if (4) there are not both males and females among the matured adults, and if in their wanderings the females are not impregnated.

It will at once be seen from the above summary that the isolation of infective man from healthy cyclops and of infected cyclops from man must be the object of any organized effort to stamp out dracontiasis.

It is with diffidence that I offer any suggestions on the practical application of this conclusion. We must look rather to those who are intimately acquainted with the customs of the people and the physical conditions in the affected areas for proper guidance as to the means by which it can be most successfully achieved. My remarks are based on what I saw of the water supplies of the villages in the eastern half of the Gold Coast Colony during a journey from the coast at Accra to Kpong on the Volta river. The observations, necessarily somewhat limited, were made at a time of severe drought, and it may well be that measures which then seemed best adapted to the requirements may prove unsuitable or impracticable elsewhere and under other climatic conditions.

From the nature of the disease it is evident that dracontiasis will disappear from the Coast towns when the provision of a properly-controlled water supply, obtained either from artesian wells or through pipes from some rapidly-flowing stream, permits the filling-in of the surface collections of rain water and the shallow wells upon which the natives now depend for their supplies. Care would have to be taken that any artificial reservoirs constructed were efficiently protected by their isolated situation from the possibilities of infection.

We have to adapt our prophylactic measures in the meanwhile, however, to the conditions as they now are in the towns, and as they will probably always remain in the inland villages. The simpler and less costly these are the likelier they are to be put into practice.

The native, more concerned with quantity than purity, takes his drinking water wherever it is most accessible—from rivers or rain tanks, from shallow wells, or the large ponds usually found at each village.

The question of the infectivity of these sources depends primarily on the presence of cyclops. It is said that cyclops does not occur in swiftly running waters, and this I found true of the Volta and the Sekum, the only rivers I had an opportunity of examining owing to the drought. It is noteworthy that in the village Oblogo, which depends solely on the latter stream for supplies, I could get no information of the occurrence of dracontiasis amongst the inhabitants. If, therefore, the scheme for providing Accra with this water becomes a reality it is to be expected that dracontiasis will disappear from the town. An examination of the rain tanks and the wells in and around Accra, Christiansborg, and Labadie was likewise negative. The reverse was the case with the village pools. Those at Akuse and Christiansborg were found to contain vast numbers of cyclops and other forms of crustacean life. In the latter village the evidence seemed irrefutable that the disease is endemic, and a small pond situated centrally, which supplied a proportion of the small population at least during the dry season, appeared the likely centre of infection. In cyclops obtained there I frequently found a nematode larva differing only from that of the Guinea-worm in its greater size and the sharp chitinous tips of its triradiate tail. Probably it belongs to the genus *Cucullanus*, but it deserves mention as a possible source of error in the examination of suspected water.

At Akuse, numerous cyclops were found where the natives drew their drinking water, and where at another spot they could be seen washing.

In most of the villages of the plains I was able, while passing through, to procure cases of dracontiasis, and the histories were suggestive that the infection had been locally acquired. Unfortunately the ponds at most of

these places had become dry, and consequently the presence of cyclops could not be determined.

The village pond seems to me undoubtedly the most likely source of the vast majority of the infections. Attention must, therefore, be mainly directed, on the Gold Coast at least, to their protection. My negative results seem to show that the wells are relatively less important. The wells should be surrounded by a stone parapet of about 2 ft. in height and the village pools stoutly fenced around in order to prevent the natives from bathing there or from wading into the water when filling their storage vessels. Each pool would have to be provided with a sufficient number of "drawing" wells dug at a short distance from the pool and connected with it by underground pipes in such a way as to ensure a constant supply of water. The openings of these would be protected by a stone or cement wall. In this way by preventing water in contact with the legs of the drawers returning into the pool the chances of infection would be reduced nearly 90 per cent. The "drawing" wells would most likely be used freely by the natives, as less labour is involved in obtaining water by draw-bucket or pump than by collecting it with a calabash. Nor would the prevention of the use of the pools for washing prove as great a difficulty as might be supposed, for at several places the natives have already learnt to do this at a place apart from where the drinking water is drawn. At Accra, the filling of the large reservoir by a thunderstorm a few days before I left gave me the opportunity of observing that there those using the water for washing purposes carried it over the embankment to the lower ground. These precautions were, however, rendered unavailing, as far as dracontiasis was concerned, for those drawing the water for home use frequently entered it for some distance in order to secure a more copious supply than that obtainable at the margin.

If by these or other and more practical methods the infection of the open ponds in the villages could be prevented there is little doubt that a considerable diminution in the disease would soon result.

There still remain for careful investigation in relation to the prophylaxis of dracontiasis many details regarding the life of the intermediate host. We have still to determine more accurately the conditions under which it lives and multiplies in these tropical countries, what are its natural enemies, and upon what other forms of life it depends for its food supply; whether it can survive the drought of the summer, buried beneath the sun-caked mud, or if, when once a pool has dried, it must be restocked from another source; lastly, if, by the addition of chemicals, we can destroy the cyclops in suspected waters without rendering these useless or dangerous to man.

No account of my investigations on the Gold Coast would be complete were I to leave unacknowledged the kindly interest with which Sir John P. Rodger and Major Bryan encouraged my work, the generous way in which the Principal Medical Officer and his deputy aided me with every facility the medical department could have provided, and the daily help and forbearance of Dr. Buée, who had charge of the Government hospital, and Dr. C. H. D. Ralph, the Medical Officer of Health for Accra.

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THE MEDICAL PROFESSION IN AUSTRIA.—According to official sources, the total number of medical practitioners in Austria at the end of 1906 was 11,937, being an increase of 173 as compared with the previous years. These are distributed according to provinces as follows: Austria, 3,613; Lower Austria, 409; Salzburg, 136; Styria, 696; Carinthia, 147; Carniola, 106; Coastland, 382; Tyrol and Vorarlberg, 622; Bohemia, 2,845; Moravia, 958; Silesia, 249; Galicia, 1,480; Bukowina, 155; Dalmatia, 138. In Bosnia and Herzegovina there are 118 practitioners. The following are the figures for the larger cities: Vienna, 2,902; Prague, 447; Graz, 263; Trieste, 199; Innsbruck, 97; Lemberg, 347; and Czernowitz, 68.

ATOXYL IN THE TREATMENT OF
TRYPANOSOMIASIS.

BY

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SINCE a number of statements concerning the use of atoxyl in human trypanosomiasis have recently appeared in various journals, lay and medical, it seems opportune briefly to recapitulate what is known concerning the action of this drug on trypanosomes and to indicate what seems to be the best method of administering it.

"Atoxyl," a preparation of arsenic (meta-arsenic-anilid; $C_6H_5NH As O_2$) which contains about 37.6 per cent. of arsenic, was first used in the treatment of various skin diseases and of anaemia.^{1,2,3} The beneficial action of arsenic in trypanosomiasis (nagana) was first reported by Livingstone. Succeeding experimenters (Bruce, Lingard, Laveran and Mesnil, Thomas and Breinl, and others) also noted the marked action of arsenic, administered in various forms, on the parasites in the blood stream of animals infected with trypanosomes, of several different species. All these observers recognized, however, that, as a rule, the protective effect of the usual forms of arsenic was only temporary, and that, in the end, the parasites reappeared in spite of steadily increasing doses of arsenic, until a point was reached at which the animal died, either of its disease or through poisoning by the arsenic given to cure it.

Thomas and Breinl^{4,5,6} thought that it might be possible to find in some of the compounds of arsenic with organic bases a form of the drug which would still retain its deleterious effect on trypanosomes, but would be less toxic to the animal treated. A number of preparations were tried without result, until atoxyl was found to possess both of the desired qualities in a marked degree. A large series of experiments on animals infected by various trypanosomes was carried out at the Runcorn Laboratory,⁷ and it was found that when very severely infected animals were thoroughly and persistently treated with atoxyl a very marked improvement immediately followed. The parasites obviously degenerated, and totally disappeared from the blood in a very few hours. The animals gained weight, lost their symptoms, and seemed in apparent health. In some of them the amelioration was permanent. In others, if the treatment were stopped, the parasites reappeared in the peripheral circulation after a varying period, or, if the drug were given continuously, the parasites eventually reappeared in spite of treatment, and increased in numbers until the animal died, as in the ordinary course of an untreated trypanosome infection. Laveran, Mesnil and Nicolle⁸ have had a similar experience. Thomas and Breinl were much impressed with the effect of atoxyl, and advised that the drug should be given in as high doses and as continuously as possible in cases of human trypanosomiasis. They stated that,⁹ "at the same time, all aids in building up the physical condition of the individual should be used." In their opinion, "if such a régime be carried out and treatment be begun at an early period, the prognosis (based on the experience of animals treated) will be good." As a result of the work of these authors, the Liverpool School of Tropical Medicine has ever since consistently advised that this drug should be given a most careful trial in the treatment of sleeping sickness.⁹

Since October, 1905, the Liverpool School of Tropical Medicine has sent large quantities of atoxyl (2,800 grams), with careful instructions for use, to various medical men and missionaries in West Africa, who have signified their readiness to give the drug a careful trial. Reports have as yet been received from none of them.

In the meantime, atoxyl has been used in Europe in the treatment of several Europeans and negroes, from both the east and west coasts of Africa, who were infected with *Trypanosoma gambiense*. In some of them the results have, so far, been most encouraging; in others, unfortunately, the drug has had but little action.

Kopke is quoted¹⁷ as having treated cases by atoxyl with results encouraging at first, but later followed by