onovan will be able shortly to elucidate the nature of these very interesting objects. Their size and shape and the arrangement of their chromatin are so constant and characteristic that it is scarcely possible to doubt that they are parasites of some kind, probably protozoa. For my own part, I find some difficulty in adopting Leishman's view that they represent involution stages of trypanosomes in the *post-mortem* spleen. He describes accurately such forms in the rat; but there, it should be carefully noted, the stained flagella are always present (with a successful Romanowsky technique) in addition to the macronucleus and micronucleus, and are, indeed, the last of the elements to refuse colouration. If, then, these bodies are nothing but disintegrating trypanosomes, we should certainly find a few of the flagella among them. Then, again, neither the large nor the small nucleus in the preparations seems to me to recall those of trypanosomes; and they are, moreover, approximated in a much more regular manner than, I think, we observe in dead trypanosomes.

Lastly, as Donovan observes, his two preparations made *intra vitam* exclude *post-mortem* changes altogether, and should, therefore, contain some unaltered trypanosomes. *Prima facie*, then, I am strongly inclined to think that we have to do with some quite novel organism. As it has already been found in eight cases of fever and cachexia, it promises to be a common and important one. The charts of two of Donovan's cases sent by him to me certainly recall the chronic pyrexia with enlarged spleen so frequently observed in India, and are, indeed, not a little suggestive of kala-azar.

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FILARIASIS IN MAN CURED BY REMOVAL OF THE ADULT WORMS IN AN OPERATION FOR LYMPH SCROTUM.*

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FILARIASIS is very rarely met with in Canada; in fact, I am unable to find the record of a single instance in which *filaria sanguinis hominis* has been reported. In the case at present under consideration the patient had for many years been a resident at Barbadoes, and had contracted the disease whilst living there.

The pathogenesis of filariasis is well known, and we may sum up the historical record of our knowledge of filariae as follows:

The embryo filariae were first discovered in the fluid of chylous hydrocele by Demarquay in 1863. Lewis showed that the embryos had their normal habitat in the blood of a man in 1872. Bancroft found an adult worm in an abscess in a lymphatic gland in the arm in 1876, and later four others in a hydrocele of the spermatic cord. Manson,¹ who studied the disease in China, was the first to observe a periodicity in the occurrence of the embryos in the peripheral blood; his paper was read before the Pathological Society of London in 1881. As early as 1877 he had suspected the mosquito (¹, p. 291) as playing the part of the intermediary host, and of transmitting the infection to man. In 1879 he demonstrated the life history of the parasite in the body of the mosquito *Culex ciliaris*. Low in 1900 observed and described the filariae lying in the proboscis of the mosquito. In 1901 Annett, Dutton, and Elliott,² whilst studying the disease on the West Coast of Africa, determined still more accurately the manner in which the mosquito parted with the parasite; they found that the filariae passed into the labium of the mosquito, and made its way from thence into the blood of man by rupture of a thin chitinous membrane on the upper surface of the labium. It would thus appear that infection is communicated to man by the bite of the mosquito. There is another possibility, though it is considered a remote one—namely, that drinking water may become contaminated by the dead bodies of mosquitos containing filariae, and thus infection of man may occur.

The victim of filarial infection does not necessarily suffer any inconvenience from the presence of the worm and the embryos; in fact it would appear to be the exception for pathological lesions to manifest themselves in persons thus infected. Annett, Dutton, and Elliott² found in some locali-

ties of the West Coast of Africa as many as 50 per cent. of the inhabitants infected; it was extremely rare to find these individuals suffering any inconvenience whatever; seldom was any diseased condition manifested. Nevertheless filariae are capable of producing disease; this may be brought about by the adult worms blocking the lymphatics and thus interfering with the circulation of the lymph. Occasionally, too, parent worms may die and in process of disintegration may produce local inflammatory trouble. Then, again, the embryo forms are, under certain conditions, responsible for disease. The normal embryonic filariae are apparently incapable of producing any pathological lesion whatever; they circulate freely in the blood and cause no disturbance of function, but immature ova have been shown by Manson to be a cause of trouble. Under certain conditions, probably in consequence of injury to the parent worm, immature ova are discharged in large numbers into the blood, and as a result pathological conditions subsequently manifest themselves. This is due to the fact that the immature ova cannot move as smoothly in the circulation; they are apt more particularly to block the finer lymph vessels, and then trouble ensues.

The filarial diseases which may thus be produced have been enumerated by Manson³ as follows: Abscess, lymphangitis, varicose groin glands, varicose axillary glands, lymph scrotum, cutaneous and deep lymphatic varix, orchitis, chyluria, elephantiasis of the leg, scrotum, vulva, arm, mamma, and elsewhere, chylous dropsy of the tunica vaginalis, chylous ascites, chylous diarrhoea, and probably other forms of disease depending on obstruction or varicosity of the lymphatics or on the death of the parent filaria.

The patient whose history I now record suffered from elephantoid fever, lymph scrotum, lymphadenitis, and abscess with varicose groin glands. The details of the case are as follows:

J. W. W., aged 47 years, a native of Barbadoes, West Indies, came under my care in the Toronto General Hospital on December 18th, 1900, in order to get advice concerning a swelling of the scrotum.

History.

He had lived in Barbadoes all his life with the exception of six or eight short trips which he had made to Canada and Europe. He gave the following history:

In 1881, when he was 28 years of age, he suffered from what he believed to be hydrocele, for the treatment of which both sides of the scrotum were tapped and fluid drawn off, after which iodine was injected. This apparently effected a cure. With the exception of the hydrocele he enjoyed perfect health and never had a serious illness of any kind until 1884, when on one occasion he became suddenly ill; an initial chill was followed by high fever and drowsiness. The chill was not repeated, but he was in bed for a week, the fever continuing, and there developed a painful swelling of the right leg below the knee; this gradually subsided and he fully recovered. He remained well for two years, when a similar attack occurred with exactly the same symptoms, but in addition, coincident with the occurrence of the chill, the glands in the right groin became swollen and tender. In subsequent attacks pain in the groin glands formed the first symptom and warned him of the approach of trouble. These attacks occurred at long intervals for the next few years: an interval of a year or eighteen months would pass without one. During an attack in 1891 an abscess developed in the calf of the right leg; this was opened and subsequently healed up without much trouble. At this date the scrotum first began to be affected and to swell up during the attack, and this became a feature of all subsequent seizures. At first the scrotal swelling would go down after the attack, but soon permanent thickening resulted. From October, 1899, until March, 1900, he had a series of successive attacks in Barbadoes: they were more severe than he had previously had. In all, seven attacks occurred during the six months mentioned. In one of them he had a severe rigor which lasted half an hour.

The patient never suffered an attack whilst in a cold climate, and hence during his visits to Northern Europe and Canada he has been free from the trouble altogether. A curious fact is that, in spite of the series of attacks such as I have described, filariasis had not been suspected, and he had never had his blood examined until he came under my care in Toronto. The history, too, would indicate that he had been a victim of the infection for sixteen, possibly for twenty, years.

Condition on Admission.

The whole scrotum was greatly thickened. There was sufficient enlargement to form a heavy, somewhat flabby mass about three times the normal size. He supported the scrotum in a flannel bag when he walked about. The enlargement diminished slightly after he had been lying down for some time, but there always remained a considerable amount of permanent thickening. The tissues of the scrotum had a thick, indurated, leathery feel on manipulation, and there was some additional enlargement in the region of each testicle. On the right side there was a nodule about the size of an almond apparently in the epididymis. The condition about the testicles might have been due to the hydrocele from which the patient had suffered in 1881.

Examination of Blood.

On my first examination of the patient I suspected that I had to deal

* Read before the Canadian Medical Association at Montreal.

with a case of lymph scrotum, probably of filarial origin. I stated my suspicions to Dr. J. H. Elliott, who happened to be in Toronto at the time. Dr. Elliott had just returned from the West Coast of Africa, where, whilst a member of the staff of the Malarial Expedition to Nigeria from the Liverpool School of Tropical Medicine, he had studied filariasis in man and in birds. He kindly undertook the examination of the blood, and subsequently succeeded in isolating the parent worm from the tissues which I excised at the operation. On the night of January 4th, 1901, Dr. Elliott drew some blood from the finger tip, and made three cover-glass preparations. In each of these he found the embryo filariae in large numbers. Thus under one cover-slip he counted 20 embryos. They were of a typical form, and were readily recognized as *filaria sanguinis hominis nocturna*. In all instances the embryo was seen to possess a distinct case, such as is characteristic of the nocturna variety of filaria.

Operation.

I operated on January 5th, 1901. Chloroform was administered, and the greater part of the indurated scrotum was removed by an elliptical incision, the long axis of the ellipse running from before backwards, beginning in front at the base of the penis, and ending behind at the most posterior part of the scrotum in the middle line. Having excised this portion of indurated skin, there were left two lateral flaps of scrotal tissue just sufficient to cover the testicles. The operation was simple and presented no difficulties whatever. There was very little bleeding, and no ligatures were required. The edges of the wound were brought together by interrupted silkworm-gut and continuous horsehair sutures. An antiseptic dressing was applied. The wound healed kindly by first intention, and the stitches were removed on the eighth day.

Examination of Specimen.

At the time of operation Dr. Elliott took charge of the scrotal tissue at the moment I removed it. He placed it in normal salt solution, and made a search for the parent filariae, teasing the tissue with a couple of needles. A number of fragments, apparently of an adult filaria, were floated off, but at first no complete worm. Then a small cyst-like body was observed on the surface, on opening which a drop of serous fluid escaped, while in it was found an adult filaria. On placing this in a dish of salt solution its movements became very active, coiling and uncoiling itself with extreme rapidity; it looked like a piece of animated white thread, about 6 cm. long. If momentarily its movements ceased it would seem to be in an inextricable coil. Further examination of the tissue failed to reveal any other adult worms. In the salt solution the adult filaria remained alive for some hours.

Observations on Periodicity.

In order to determine the periodicity of the appearance of the embryos in the peripheral blood a careful count was made every three hours from noon on January 28th to noon of the next day. Dr. Coutts, who was then assistant in the Pathological Department, made the counts. A glass pipette was used, and each drop of blood for examination was carefully measured so as to obtain the same quantity of blood under each cover slip; the embryos under each were then counted. Four cover-slips were prepared for each count, and the number of embryos in each cubic centimetre of the blood was calculated from the results obtained.

Date.	Time.	Embryos per c.cm. of the Blood.
January 28th	3.10 p.m.	126
January 28th	6.10 p.m.	840
January 28th	9.10 p.m.	1,175
January 29th	12.10 a.m.	1,680
January 29th	3.10 a.m.	1,540
January 29th	6.10 a.m.	1,764
January 29th	9.10 a.m.	273
January 29th	12.10 p.m.	0

After-history.

On February 19th, forty-six days after the operation, the patient suffered an attack of elephantoid fever. This was the first occasion on which he had experienced an attack of this nature in a cold climate. Immediately after lunch, in the middle of the day, he was seized with a chilly sensation, the skin became hot and dry, and he realized that he had what previously in the Barbadoes he had been the habit of calling an attack of "ague." When I saw him at 5 p.m. his temperature was 104.4° F., respirations 32 per minute, and pulse 120. A tender swelling about the size of a hen's egg existed in the posterior triangle at the root of the neck on the right side. The superficial tissues over this region were congested and oedematous. He tells me that in the early afternoon when the attack first manifested itself, he suffered severe pains in the back and limbs, with characteristic stretching and yawning, a feeling of lassitude and drowsiness such as he had always experienced in the tropics when an attack of this kind supervened. The local inflammatory condition subsided but recurred, and ten days after the onset of the attack the oedema and tenderness made me suspect pus, and I incised the cervical swelling. I found a mass of sloughy-looking material, composed of greyish, oedematous, broken-down glandular tissue. There was no pus formation. Careful search was made for parent worms, but none were found. The wound slowly healed.

On March 13th at midnight, and at 7 o'clock the next morning, a sample of blood was taken for the purpose of demonstrating the embryos to my class. I was surprised to learn that no embryos were to be found, and none could be discovered on subsequent occasions, although many attempts were made to secure a specimen. The following week Dr. Coutts made a systematic search for the embryos, proceeding in the same manner as he had done when making the counts for periodicity

in January. The same quantities of blood drawn at similar hours were submitted to careful search. He succeeded in finding a single embryo in the blood drawn at 9.10 p.m. All the other samples were free from embryos; they had entirely disappeared. Subsequent to this, samples of blood taken at irregular intervals at night failed to discover any embryos.

It would therefore appear that not only had the patient got rid of his lymph scrotum, but the operation had resulted in freeing him of his filariae. The only possible explanation appeared to be that the filariae which Dr. Elliott found in the excised tissue were the parent worms, which were responsible for the production of the embryos in the blood, and that on their removal the embryos ceased to be produced, whilst those already in existence gradually disappeared, as, of course, they never come to maturity in the human blood, and must perish unless they are carried to an intermediate host.

My patient subsequently returned to the West Indies, where he at once adopted precautions to prevent reinfection, from the very first night sleeping under netting in order to keep the mosquitos from him. He writes to me under date July 25th, 1901, to the effect that he had had his blood examined. He was fortunate enough to have the examination made (at the request of Dr. Phillips, the medical attendant) by Dr. G. C. Low, of the London School of Tropical Medicine, who happened to be in the locality at the time. Dr. Low pronounced him still free from filaria embryos. This examination was made six and a-half months after my operation on the scrotum when the parent worms were isolated. One may therefore conclude that a permanent cure had been effected by the removal of the parent worms from the scrotum.

DR. J. H. ELLIOTT'S DESCRIPTION OF THE ADULT WORMS.

Dr. Elliott was good enough to make a careful study of the adult worms found. He reports as follows: Microscopic examination of the fragments found showed them to be apparently from a fully-developed female. The one perfect filaria proves on examination to be a female not fully mature.

Fig. 1.—Anterior end $\times 60$. A. Oral aperture. B. Nerve ring. C. Oesophagus. D. Vulva.

Fig. 2.—Posterior end $\times 60$. A. Alimentary canal. B. Posterior horn of uterus. C. Cloaca. D. Anal orifice. E. Cuticular striations.

The length is 55 mm., and width 0.19 mm. (When mature the length of the filaria bancrofti varies from 88 to 155 mm. and the width 0.6 to 0.7 mm.) Both ends are tapered. The head end is slightly bulbous, measuring 0.052 mm. at its greatest diameter. The neck at its narrowest point has a diameter of 0.045 mm. The tail end tapers until it reaches a diameter of 0.044 mm., and then is abruptly rounded. The cuticle shows

Fig. 3.—Region of vulva X 270. A. Alimentary canal.

Fig. 4.—A point 3.48 mm. from head end X 90. A. Alimentary canal. B. Muscular walls of vagina.

Fig. 5.—Near middle of the worm X 90. A. Alimentary canal. B. The two horns of uterus.

transverse striations, especially near the ends. The buccal orifice is centrally situated at the anterior end. The first part of the alimentary canal is thick-walled and muscular, but throughout the greater part of its length the walls are thin. The posterior opening is lateral, the anal orifice being marked by a slight depression 0.28 mm. from the end. What appears to be the nerve collar crosses the oesophagus 0.195 mm. from the anterior end. The vaginal orifice is situated at a distance of 0.67 mm. from the anterior end, being marked by a depression surrounded by a slightly-raised papilla. The width of the worm at the vulva is 0.125 mm. The vagina, which possesses thick muscular walls, extends backwards 3.45 mm., where it receives the two horns of the uterus. Only a small number of ova are present in the vagina, but the uterine horns are completely packed with them. The uterine walls are thin. The two horns make several turns about one another and the gut as they pass backwards to the posterior end. The gut has a constant diameter of 0.0347 mm. The rest of the coelomic cavity is taken up by the distended uterus. The uterus terminates blindly about 1.2 mm. from the tail.

Cobb's formula,⁴ which was used extensively by the malarial expedition to Nigeria (², p. 23), was devised so as to indicate the measurements of different parts of nematode worms as percentages of the whole length of the body. Reduced to Cobb's formula the size of the parent worm in this case was as follows:

— 0.36	3.64 ?	1.22	99.5
— 0.145	0.327 ?	0.254	0.147

Measurements for specimens of *filaria Bancrofti* do not appear to have been previously published. They could not be obtained for the work on *Filaria* issued by the Malaria Expedition to Nigeria (², p. 44).

COMMENTARY.

The attack of lymphadenitis in the neck with the characteristic symptoms of elephantoid fever, which came on forty-six days after my operation, might be accounted for by supposing that immature ova had been discharged into the circulation by the parent worms during the manipulations at the time of operation. The immature ova, as has already been pointed out, are considered by Manson to be a frequent cause of trouble in individuals suffering from filariasis. In my case, it might be presumed, they brought about a lymph stasis in the cervical region, with the sequel of events described. This was the only attack of elephantoid fever

suffered by my patient in a cold climate, and I think the observation is one of considerable interest. With this single exception he enjoyed complete immunity from such attacks whilst visiting Canada and Northern Europe; and it must be admitted that the single instance referred to occurred under very exceptional circumstances, directly traceable, no doubt, to the operation. The general observation might therefore be made, if we might conclude as much from the evidence here presented, that an individual the subject of filarial infection gets complete relief from attacks of elephantoid fever when he migrates from the tropics and takes up his abode in a cold climate. Immunity from such attacks was enjoyed, although, as examination showed, the embryos continued to circulate in his blood. The truth of this fact was not deduced from a single experience, but was proved repeatedly by my patient, who on many occasions sought and found relief from the frequent attacks which he suffered in the tropics by visiting a cold climate.

Many interesting observations have been made regarding the occurrence of the embryos in the peripheral blood. Manson⁵ has determined by observations conducted *post mortem* on a man who committed suicide, and who had been a victim of filarial infection, that when the embryos are absent from the peripheral circulation they find shelter in the larger blood vessels, particularly the arteries, the majority being found in the vessels of the lung. Manson calls attention to the analogy which exists between filarial periodicity and that of the malarial parasite, "the two parasites of man subverted by the mosquito." In 1882 Stephen Mackenzie⁶ reported a case of haemato-chyluria of filarial origin in the person of a private in the Royal Artillery who had come from India, and was admitted to the London Hospital. Mackenzie reversed the patient's usual hours of movement and rest, his meal times being correspondingly altered. The effect was a complete inversion of the filarial migration. The embryos were now found in large numbers during the daytime, and were absent during the night. On resuming his usual habits the nocturnal periodicity returned as before. No reasonable explanation of this is forthcoming.

In tropical countries it is not uncommon to find a type of filarial infection in which the embryos are only found in the peripheral blood in the daytime in individuals of ordinary habits of sleep, etc. In the case of this parasite known as the *filaria diurna* the periodicity is therefore the reverse of that described in my case. No marked pathological lesions have been encountered in connexion with this parasite, and this circumstance, as Annett, Dutton, and Elliott (², p. 53) point out, accounts for the fact that their geographical distribution has not been made out with accuracy. No difference can be detected, in the embryonic form, between *filaria nocturna* and *filaria diurna*, and according to Annett, Dutton, and Elliott (², p. 92) "the weight of evidence is on the side of the identity" of these two forms. If they are identical, then, for some unknown reason, there must be a reversal of the periodicity comparable to that established experimentally by Mackenzie. The parent form of *filaria diurna* has not been discovered. Manson suggested the possibility of *filaria loa* being the worm in question. This worm (*filaria loa*) is from 16 to 70 mm. in length (², p. 50), it inhabits the connective tissues, and has frequently been observed moving about under the conjunctiva or under the thin skin of the eyelids. It moves about in such localities with marvellous rapidity. Two specimens of this worm have been obtained to my knowledge in Toronto, in both cases removed from the eyelids of individuals who had returned to Canada from the tropics. One case was reported by Dr. Frederick Fenton,⁷ the other occurred in the practice of Dr. F. N. G. Starr. Unfortunately in neither case was the blood examined for embryos, as the suggestion regarding *filaria diurna* and its possible relationship to *filaria loa* had not then been made, and there was no reason to suspect the presence of parasites in the blood.

There are some forms of filarial infection in which no periodicity whatever occurs. For example, in *filaria perstans*, which is very common on the West Coast of Africa, embryos in small numbers are found in the peripheral blood at all hours of the day and night. The embryonic form of *filaria perstans* is very different from *filaria diurna* or *filaria nocturna*. It is much smaller than either of these, and differs markedly from them also by possessing no sheath.

Occasionally an individual may be the victim of filarial infection, and may present evidence of extensive lesions produced thereby without at any time showing filarial embryos in the peripheral circulation. This can only be explained on the hypothesis that the lymphatic channel has been so

blocked that the embryos are unable to find their way to the blood stream. Thus Strube in the *Deutsche medicinische Wochenschrift*⁹ describes five such cases, from Natal and the Transvaal, where filarial embryos were found in the urine but none were present in the blood, which was carefully examined during the day and night. Lothrop and Pratt¹⁰ (p. 284) cite this paper in support of the statement that embryo filariae had been found in the blood of the negroes of the Transvaal. As a fact, however, the blood was free from embryos in Strube's cases.

Manson¹ asserts his belief that before embryos can be found in the blood by ordinary microscopic examination large numbers of parent worms must be present in the lymphatics. Annett, Dutton, and Elliott² (p. 67) were led to believe from their investigations that Manson's conclusions were not justifiable. The case which I now place on record would seem to have a direct bearing on the question, and would go to show that very few parent worms may be responsible for a large number of embryos in the peripheral circulation. Lothrop and Pratt,¹⁰ of Boston, report two cases of filariasis observed in that city, in one of which operation was performed for lymphatic varices and chylous hydrocele, with the removal of adult worms. In all nine worms were removed, of which two were males. There was, however, in their case no subsequent disappearance of the embryos from the blood; they were found present six months after operation. In their paper, which was published in 1900, they had collected 11 cases from the literature in which adult filaria bancrofti have been found, and, as far as they were able to ascertain, the adult worms had never before been found in the United States. I can find no case on record of a cure being effected by removal of the adult worms such as I now report. Manson³ (p. 505) states that the disappearance of filarial embryos from the blood which sometimes occurs in cases of lymphatic varix of long standing is doubtless due to the death of the parent parasites, but there is no suggestion that such a result may be brought about by operation.

The interference with the lymph circulation is often very great in these cases, and those who have had much experience in operating state that there is danger in attempting to excise affected tissues, lest in so doing the anastomotic circulation of the lymph may be interfered with to such an extent that matters may be made worse rather than better. Godlee¹¹ has quite recently, on the suggestion of Manson, operated with the view of producing an anastomosis between one of the dilated lymphatic vessels and a tributary of the internal saphenous vein. Subsequently another lymphatic had been introduced into the spermatic vein. The result had been extremely successful.

Apart from the presence of embryos the blood has exhibited other peculiarities in infected individuals. Gulland¹² has shown that there is a distinct eosinophilia observable when the embryos are present in the peripheral circulation. This, Gulland observes, brings filariasis into line with other parasitic infections—for example, ascariis, oxyuris, ankyllostoma, taenia, and trichina, in all of which eosinophilia exists. This, it is asserted, goes to show that the eosinophiles are concerned in protecting the body from the toxins of the parasites. Coles¹³ has confirmed Gulland's observations and has found eosinophilia in bilharzia disease also. Calvert¹⁴ made somewhat similar observations among Filipino prisoners at Manila, but he was of opinion that the eosinophilia was only characteristic of the early stages of filariasis, and that as the disease progressed the leucocytosis and increase in eosinophiles gradually decreased.

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BUBONIC PLAGUE ("KAUMPULI") IN CENTRAL EAST AFRICA.

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In the writings of several of the earlier explorers and missionaries in Uganda and in that part of German East Africa to the west of the Victoria Nyanza, frequent references are made to the occurrence of local outbreaks of a rapidly-fatal disease known in Uganda as "kaumpuli," and in German East Africa as "tubunga."

Their accounts of the disease are vague, but all seem to agree as to its being a bubonic affection, attended with a very heavy mortality. Some state definitely that it is plague. Thus Ashe, a missionary who saw much of the Baganda during the reign of M'tesa and M'wanga, says:¹

This frightful malady [small-pox] and the plague, a kind of black death, called kaumpuli, from time to time decimated the population of Buganda. The kaumpuli is recognized by the swellings in the armpits and in the groin, and by a terrible rise in the temperature of the patient. The fever runs its course in about twenty-four hours, and usually proves fatal. Small-pox perhaps kills more people, yet it is not so much feared by the natives as kaumpuli, of which they have the greatest terror, as it is supposed to be frightfully infectious.

Stuhlmann, referring to diseases prevalent amongst the Baganda, remarks² that the most feared is the so-called kaumpuli, which is in the highest degree contagious and deadly, and often wipes out whole villages. Where it appears the lymphatic glands begin to swell and suppurate, accompanied by symptoms of severe fever. Probably, he says, it is a kind of bubonic plague. The French missionaries use as a remedy plasters made by themselves, which they place upon the chest and back of the patient; but whether with good results or not he does not state.

Dr. Milne, acting Principal Medical Officer for Uganda, reports³ that a chief near Kampala informed him that "in Lugard's time it [kaumpuli] carried off very many people; in fact, it was worse than war." He also states that the Collector of the Budu district, Mr. Prendergast, had in 1900 under his own observation at Masaka seventeen men of the country police force who died in forty-eight hours with all the manifestations of a bubonic disease, and that many more cases were reported to him. The result of Sir Harry Johnston's inquiries⁴ was that

from accounts of English and French missionaries and the German authorities to the south of the Uganda border there is little doubt that in Budu, and perhaps also in Busoga, the bubonic plague, or some disease related to that malady, exists in an endemic or chronic form.

Also that

the bubonic plague, which is always simmering in these countries near the Victoria Nyanza, has visited Unyoro repeatedly, having largely brought about the depopulation of the Bwenli subdivision.

During 1900 and 1901 several letters were received by the administration authorities at Entebbe from the White Fathers of Bikira, a large missionary station in Budu, two marches south of Masaka, relating the occurrence of severe outbreaks of kaumpuli at their station and in its neighbourhood. One letter told of the death of one of the Fathers after only four days' illness; another stated that many rats had died in the huts of the natives and in the mission buildings. A medical officer was sent to them, but at the time of his visit there was no outbreak, nor did he discover any dead rats. I believe he came to the conclusion that the disease, whatever it was, was not plague. Shortly afterwards, early in 1902, the mission station at Bikira was closed by an order of the Bishop, and the Fathers were dispersed to other districts.

At the time of my recent visit to Uganda (November, 1902), a good deal of speculation as to the nature of kaumpuli was indulged in; but no medical officer of the Protectorate had succeeded in observing an authentic case of the disease. The Baganda are apt to apply their word for "plague" to any disease which carries people off suddenly; and, to add to the confusion, sleeping sickness was frequently called "kaumpuli" in the Sese Islands. It was certain, however, that sudden localized epidemics of some very fatal disease of a bubonic character, and of which the natives stood in great dread, did occur from time to time in the province of Budu, and had occurred in the past on a large scale in other parts of Uganda.

Having served for three years under the Indian Government as Special Medical Officer for Plague in the Bombay Presidency, I had been able to study the disease in the dark, ill-ventilated, low, mud houses, and filthy overcrowded