SLEEPING SICKNESS, TSETSE, AND BIG GAME.

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I HAVE read with much interest the articles and correspondence in the issues of the BRITISH MEDICAL JOURNAL for June 21st and July 19th, 1913, regarding tsetse, big game, and sleeping sickness in Nyasaland and Rhodesia.

With the exception of three furloughs spent in Britain I have been at work in the territories referred to since 1894. For many years after my arrival on Lake Nyasa my practice extended over a very wide area, and it so happened that many of my journeys were made through districts which are now infested with tsetse fly. It has been my custom to use riding animals whenever it was possible to do so. Frequently I had opportunities of shooting game as we travelled. In the early days I frequently had dogs with me. The presence of riding animals and good dogs made me very careful about the possibility of encountering tsetse fly when on tour, and a close observation was made when game was shot as to whether or not tsetse happened to be about. Time and again I travelled through districts where in pre-rinderpest days Arab caravans used to travel by night in order to avoid their animals being bitten by tsetse. Yet in those early years—1894, 1895, 1896—both Nyasaland and Rhodesia were remarkably free from that fly. The explanation given me by many headmen and others was that since rinderpest had killed off most of the big game tsetse fly had disappeared. I can speak from personal experience and observation of many hundreds of miles of territory where in those early days not a single tsetse fly was to be encountered, and where to-day it swarms, and not a single domestic animal can live. I have watched the spread of tsetse fly for many years now, and long ago warned the Government of the risks they were incurring in allowing it to spread. I appealed to the authorities in Nyasaland and Rhodesia to arrest the spread of tsetse, both on the grounds that *Glossina morsitans* was killing off domestic animals and that it might yet prove to be a carrier of a trypanosome pathogenic to man.

Those of us who are working on the spot attribute the enormous increase of tsetse fly during recent years entirely to the great increase of big game. The increase of game we attribute entirely to the protective measures employed by the governments. About these we need not write in detail meantime. The laws were passed with a view to protecting the wild fauna of this part of Africa, and they have succeeded, I should think, beyond the wildest dreams of those who were responsible for bringing them into being. The wild animals have been preserved, but domestic animals have been sacrificed, and now we are paying a heavy penalty in human lives for having interfered with the balance of Nature.

In a district in which the natives owned 200 head of cattle an incursion of tsetse took place following upon the herds of buffalo that came into it. Within two years the natives owned only 80 head. Under normal circumstances there should have been an increase of 50 per cent. per annum—300 head in all, instead of 80.

In another district known to me where the people were rich in sheep, goats, and cattle, a recent investigation revealed the presence of only four dogs, and these recently imported. Besides having lost their stock, the people are now being killed off by *Glossina morsitans*. Yet with all this going on the people are not allowed to act in self-defence. Game laws which would turn any Government out of power if imposed at home are forced upon a primitive people here who have no means of redress.

To those of us who take an interest in politics, it is not a little amusing to find the Chancellor of the Exchequer posing in the matter of game as the friend of the down-trodden peasant at home, whereas a member of the same Government, in the person of the Colonial Secretary, carefully protects the huge animals that eat up the native's crops, kill off his cattle, and carry in their blood the disease germs of certain death to human beings, and that notwithstanding a promise we believe he made to a deputation representing all the missionary societies at work here—that on evidence being adduced that game

was responsible he would not fail to act. His replies to Dr. Chapple in the House of Commons have already been referred to in the JOURNAL. Not only do elephants destroy the meale crops, they this year (1913) fought the owners for possession of the gardens. In my hospital at Kasungu I had recently four patients at one time who had been mauled by wild animals. Three of them had been hurt by elephants in the gardens.

There is one aspect of this question to which I have sought to draw attention. The infant mortality in this country is appalling; so, too, is the mortality among adults from diseases of the alimentary tract. The native has no sick-room cookery worthy of the name. We have sought to teach him the value of milk as a diet for infants and during disease. But along comes tsetse, and this potential food supply in sickness is cut off. Thus indirectly by destroying the food supply and directly by carrying the deadly trypanosome does tsetse kill off the population. When we speak of the population being killed off we mean what we say. The disease, both in Rhodesia and Nyasaland, is much more serious than certain reports make it out to be. The first cases I had in my hospital here were from a district where sleeping sickness was not known to exist. Reports reached me of a very fatal disease having broken out among the natives, who spoke of it as a virulent form of measles. A lad in our employment entered the district and got badly bitten by tsetse. He became very ill, and was carried back to my hospital. I thought he had tick fever, and proceeded to examine his blood for spirochaetes; I found trypanosomes. So far as my knowledge goes, that was the first case for which *Glossina morsitans* was held responsible by a medical man. The Government sent a medical officer into the district and other cases were found. The virulent form of "measles" had carried off many victims before the real nature of the disease was diagnosed. It has carried off many more since.

How do those of us who are working on the spot believe that the disease should be eradicated? For we do believe that the disease should never have existed here, and that it can be, and ought to be, eradicated. We believe that for the time being a severe onslaught must be made upon the wild animals until these are driven back from human settlements and from the public highways. Until the present danger is past, all wild animals capable of acting as a reservoir of trypanosomes should be treated as vermin and destroyed or driven out just as rats are destroyed during an outbreak of plague. I, for one, would welcome an outbreak of rinderpest such as swept this country in 1893 and 1894. It would leave us with a clean country, and, having learnt our lesson in a hard school, we should be slow to impose laws protecting game. We cannot cure the disease when once it has taken hold of human beings. Only those who have seen the victims can realize the horror and the hopelessness of it. We can starve out tsetse by depriving it of its supply of warm blood, and at the same time we can remove the natural reservoir of the disease by driving out or destroying the game. It has been asserted that tsetse can and does exist where it cannot possibly depend upon game for its sustenance, game being absent. This does not hold good of *Glossina morsitans* in Nyasaland and Rhodesia. Instances cited by the Colonial Secretary do not stand the test of investigation.

Again, we are told that if game is driven out tsetse may attack man and his domestic animals more vigorously than at present. In that case, when rinderpest killed off the game tsetse ought to have betaken itself to the nearest villages to feed upon the people. But it did not. It disappeared or remained only in small patches among the foothills, where small herds of game escaped the rinderpest. Similarly, in South Africa, when the Boers shot off the wild animals tsetse ought to have remained behind to feed upon man and his domestic stock. But it did not. There is ample proof that wherever game is killed off by disease, or hunted out, *Glossina morsitans* disappears. There is no proof to the contrary. I hold that the time for experimenting has passed, and that we ought at once to adopt such measures as may be necessary to save the people here from further risk and loss. Traders, farmers, planters, transport riders, and missionaries ask for permission to act in self-defence, permission to save their interests from ruin and themselves and their people from danger.

If we make a mistake we suffer for it, and can blame no one but ourselves. But why should we be called upon to suffer for the fads and mistakes of people at home? We hold that further temporizing, in the light of our present knowledge, is nothing short of criminal folly.

With reference to Dr. Neave's suggestion that the game within a certain area should be impounded and destroyed, I may say that this suggestion was made fully two years ago. We asked, when the first epidemic broke out in Nyasaland, to have the game destroyed. This was refused, on the grounds, among other reasons, that the outbreak was distinctly localized, and that any interference with the game might cause the animals to spread out and carry disease into clean districts. On the strength of this statement I suggested to the Acting Governor that he should impound all the animals within the area mentioned, and once and for all stamp out the disease by exterminating the animals. To this proposal I got a reply that while His Excellency appreciated my good intentions he did not regard the proposal as practicable.

In conclusion, I should like to express my appreciation of and gratitude for the work of the Liverpool Commission, which has settled once and for all many hitherto hotly disputed points, and also my gratitude to the BRITISH MEDICAL JOURNAL for the publicity it is giving to the work of Dr. Warrington Yorke.

A STUDY OF EPIDEMIC DYSENTERY IN THE FIJI ISLANDS,

WITH SPECIAL REFERENCE TO ITS EPIDEMIOLOGY AND TREATMENT.

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THE observations recorded in this paper were made in the Fiji Islands during the year 1910. This group of islands, situated between lat. 17-19 degrees S. and long. 177-179 degrees E., was ceded to Great Britain in 1874, and now constitutes a Crown Colony.

According to Dr. B. G. Corney, I.S.O., a great authority on all matters appertaining to the Pacific, there is no evidence that dysentery was originally introduced by the early European settlers,* but it is known that since the early days epidemics of dysentery have been of annual occurrence, and have levied a heavy toll of life, not only among the European settlers and the imported indentured Indian and Solomon Island labourers, but also among the Fijian natives themselves, and have constituted the chief scourge of the colony, so healthy in other respects.

In former days these dysentery epidemics were of great virulence; the newly imported Solomon Island labourers were specially attacked; thus Daniels¹ reported one such epidemic in which the death-rate was 48 per cent.

At the present day, probably owing to better medical service and hygienic conditions, the case mortality from dysentery of all kinds is not nearly so high as formerly, and amongst the Fijians, according to the official records, is about 16 per cent.

During the year 1910 some 170 well-marked dysentery cases were studied in detail at the Colonial Hospital, Suva, the capital of the group; of these only 11 were found to be of amoebic origin—that is to say, vegetative amoebae bearing the characteristics of the *Amoeba histolytica* of Schaudinn were found in the stools. In the dysenteric discharges of the other 159 cases no amoebae were ever found.

The epidemic dysentery affected members of the various nationalities—Indian, Fijian, Solomon Islanders, Europeans, and half-castes—to an equal degree as far as could be ascertained.

In Fiji, as in more temperate zones, epidemic dysentery is an institutional disease; some 53 per cent. of the patients under observation were prisoners undergoing sentences in either of the prisons of Suva.

The Bacilli.

By plating out the mucus diluted in sterile broth on Conradi-Drigalski medium (litmus-lactose-nutrose-crystal-violet agar), dysentery bacilli were isolated from the stools in 35 cases and twice from the intestinal mucosa *post mortem*. The bacilli were recognized by their morphology, by their reactions with 1 per cent. solution of various sugars in litmus peptone water, by their agglutination reactions with high dilutions (1 in 50) of a polyvalent anti-dysenteric serum (Lister Institute), and further by their toxicity to guinea-pigs on intraperitoneal injection.

The dysentery bacilli isolated mainly conformed to the well-known types, the one most frequently encountered being the Shiga-Kruse bacillus, but a bacillus identical with the Hiss and Russel Y bacillus was also almost as common. In addition to these, other bacilli, morphologically and culturally similar, but giving atypical reactions with solutions of certain sugars such as saccharose and maltose, while agglutinating with the same polyvalent immune serum, were also encountered, and in these reactions agreeing with Strong's² original Philippine strain.

In the varieties of bacilli encountered the results, in the main, are in agreement with those of Shiga,³ who now recognizes five main types of dysentery bacilli as differentiated by their sugar reactions.

On three occasions bacilli giving identical reactions to those cited above, but in addition exhibiting a feeble lactose fermenting power, were encountered. Ohno,⁴ during his extensive study of 74 strains of the dysentery bacillus, also appears to have encountered this variety, to which he assigns 3 out of his 15 types.

Table of Reactions of Main Types of Dysentery Bacilli in Litmus Peptone Water containing 1 per Cent. of Sugars after Twenty-four Hours' Incubation at 37° C.

	Shiga-Kruse Type.	Hiss and Russel's Type Y.	Ohno Type M.
Mannite ...	—	+	+
Lactose ...	—	—	+
Saccharose ...	—	—	—
Maltose ...	—	=	+
Dulcite ...	—	—	—
Dextrose ...	+	+	+
Milk ...	Acid at first, returned to alkaline second day	Acid at first, returned to alkaline second day	Acid at first, returned to alkaline fourth day

The production of indol by these bacilli was very variable. In the true Shiga-Kruse type none was produced as a rule, even after lengthy incubation.

After long subculture over a period of nine months, and after the sugar reactions had been frequently tested, certain minor variations of certain strains were noted, especially with regard to the fermentation of maltose and saccharose; nevertheless, the reactions of the non-acid or Shiga-Kruse and the acid or Flexner type of bacillus to dextrose and mannite respectively remained constant throughout.

Clinical Observations.

Cases from which the dysentery bacilli were isolated varied greatly in their clinical manifestations—from a mild catarrhal condition, in which the stools were solid and coated with a small amount of blood and mucus, to a fulminating one, in which the call to stool was incessant and in which the patient, overwhelmed with pain, physical exhaustion, and toxic absorption, would succumb in from two to three days from the commencement of the attack. The temperature also varied within wide limits; cases were encountered in which it was subnormal throughout, others in which it reached 103° F. and over. No special dysentery bacillus was found to be associated with any clinical type of case.

No hepatic symptoms in life or suppurative lesions of this organ *post mortem* were ever encountered in this infection. Only one case of bacillary dysentery of a relapsing nature was encountered amongst the whole series. The serum of 74 per cent. of the bacillary cases agglutinated—in a dilution as high as 1 in 200 in some

* The Fiji Islands were discovered in 1643 by Tasman, who, however, did not land there. The first Europeans who had intercourse with the natives were the mutineers of the *Bounty*, who had crossed over from Tahiti in 1791.