The epidemic malarial fever of Assam, or kala-azar : a reply to criticisms / by Leonard Rogers.

THE EPIDEMIC MALARIAL FEVER OF ASSAM,

OR KALA-AZAR

Leonard ROGERS

INDIAN MEDICAL GAZETTE, 1898, 33.

3. Lesion of joint † — non-penetrating, penetrating.

4. Lesion of — Nerves; Arteries; Veins

Subdivision 2 would comprise fractures of humerus, radius, ulna, carpal and metacarpal bones and phalanges. Subdivision 3 would take the place of class XI, and would include all the joints from the shoulder downwards. Subdivision 4 would replace classes X and XII.

Similarly with class IX, which deals with the lower extremity. It would become class VIII and would have the same subdivisions.

IX.—Gunshot Wounds of the Lower Extremities—

1. Simple flesh contusions and wounds — slight, severe.
2. With contusion and partial fracture of long bones.
3. With simple fracture of long bones by contusion of round shot.
4. With compound fracture of — Femur. Tibia only. Fibula only. Tibia and Fibula. All three bones.
5. Penetrating, perforating, or lacerating the several structures of the tarsus and metatarsus.
6. Dividing or lacerating the structures of the toes.

It will be noticed that no place is given to the patella in this scheme; consequently fractures of the patella have to be entered in class XI if there was direct penetration or perforation of the knee-joint. On the other hand, a simple fracture of the patella must be relegated to the miscellaneous class XV, because there is no place for it in classes IX and XI.

In the new scheme class IX would become class VIII, with the following subdivisions:—

VIII.—Wounds of the Lower Extremity—

1. Contusions and simple flesh wounds — slight, severe.
2. Fracture of bone* — simple, compound.
3. Lesion of joint† — non-penetrating, penetrating.
4. Lesion of — Nerves; Arteries; Veins

Classes X, XI and XII would be wiped out, because they are structural in nature and would be shown as subdivisions of the eight new classes. They may, however, be fully mentioned here.

X.—Gunshot wounds with direct injury of the large arteries, not being at the same time cases of compound fracture.

XI.—Gunshot wounds with direct penetration or perforation of the larger joints — With fracture of bone, Without fracture.

XII.—Gunshot wounds with direct injury of the large nerves, not being at the same time cases of compound fracture.

One obvious defect is that the basis of these classes is now shifted from that of a region to that of a system or structure, *i.e.*, to structures such as nerves, arteries and veins, or to the nervous, arterial and venous systems of the whole body, instead of the portion of these systems or structures which are located in the different regions already defined.

The next two classes would also disappear, because gunshot, sword, sabre, lance and bayonet wounds, would be shown in vertical columns for each and all of the eight new classes above mentioned.

XIII.—Sword and Lance, Wounds of

XIV.—Bayonet, Wounds of ...

Here, once more, we have a change of the basis of classification, this time according to the nature of the weapon alone. Sword and lance wounds are mixed up; but their nature could only be similar if the sword is used as a *thrusting* in place of a *cutting* or slashing weapon.

Class XV.—Miscellaneous Wounds and Injuries received in Action.

This seems a useful group to fall back on in emergency, and it would certainly include wounds caused by splinters of stone or wood, crushing injuries caused by rocks rolled down, or by gun-carriages driven over men, and the thousand and one accidents that the soldier is liable to by flood and field while in action or on active service.

Summary.—Thus, this re-classification simplifies and reduces the number of classes from 15 to 9 on a regional basis, while it reduces and assimilates the subdivisions from 6 to 3 or 4 on a structural basis, and it allows of a wound of any and every structure in the human frame being mentioned and classed. Some alterations might advantageously be made in the second half of this form, *i.e.*, the classified return of operations performed; but these are slight and self obvious, and this paper is already too long, especially as it treats of such a dry and tedious subject.

* Here specify bone or bones.
† Here specify the joint.

THE EPIDEMIC MALARIAL FEVER OF ASSAM, OR *KALA-AZAR*.

A REPLY TO CRITICISMS.

BY LEONARD ROGERS, M.D., M.R.C.P., B.S., F.R.C.S.,
Indian Medical Service.

I HAD arranged to write an abstract of my report on *kala-azar* for the *Indian Medical*

his report, to claim that he has inadvertently

Gazette, but was prevented by my proceeding on field service, and subsequently being invalided home. Meanwhile, the review by Surgeon-Colonel Stephen appeared, and has been followed by various criticisms; a paper by me on the subject will shortly be published in the Medico-Chirurgical Society of London's Transactions, so it will now only be necessary for me to reply to these criticisms, as few of those who have read them, will have seen my full report, so many copies of which were destroyed in the Assam earthquake. Moreover, so much of the evidence that I have recorded has been ignored, or incorrectly quoted by some of my critics, that their papers convey an utterly false and inadequate impression of the evidence on which my conclusions are based.

In the first place, I may note that it is something gained that no one any longer thinks that *kala-azar* is caused by anchylostomiasis, pure and simple. Dr. Thornhill is wrong in saying that no one ever said it was, for on page 117 of my report, I quote Dr. Giles as writing: "All I wish to convey is that the increased mortality is due to anchylostomiasis and to no other cause," and again, "*kala-azar* is anchylostomiasis," with no qualification whatever.

The position now taken up by Dr. Giles is "that the increased mortality in Assam, which is known to the natives of that province as *kala-azar*, is due to the effects of anchylostomiasis acting on a population which is and has for generations been continuously poisoned with malaria." Dr. Thornhill argues at length that *kala-azar* is a cachexia produced by the combined action of malarial fever and anchylostomiasis, but he somewhat lamely concludes, "I am far from saying even if the anchylostomiasis element is admitted in *kala-azar*, that this factor will fully explain and account for its spread and apparent communicability," but he is of the opinion that this admittedly imperfect explanation is the most reasonable that has yet been offered.

Now Surgeon-Colonel Stephen, in reviewing my report, writes that he quite agrees with me that the facts I have recorded "prove incontestably that, whatever *kala-azar* may be, it is not anchylostomiasis, and that anchylostomiasis is not an essential factor in its production." Professor D. D. Cunningham, F.R.S., agrees with me that my determination of different blood changes in anchylostomiasis from that present in *kala-azar* is most important and conclusive against Giles' theory. Again, Dr. Rudduck, who had an intimate experience of *kala-azar* on tea gardens, and who was the first to study and discover anchylostomiasis in Assam, and would therefore be the last person in the world to minimise the *rôle* of this worm, wrote, previously to the publication of my report, that he believed "*Kala-azar* was distinctly a form of malarial poisoning which had, from some unknown cause, acquired an intensity fortunately rare, much as influenza occasionally does. I quote this opinion in fairness to Dr. Rudduck, as his views have been incorrently given in one place at least. Dr. Rudduck, after reading my report, very carefully has now written to me: "Finally I fully accept your conclusions as to the cause and pathology of *kala-azar*." Further, several eminent authorities in England and America, with whom I have conversed or corresponded, also agree that I have conclusively proved that anchylostomiasis plays no part in the causation of *kala-azar*. It behoves me, then, to examine closely the reasons given by Drs. Giles and Thornhill for continuing to hold the contrary.

Dr. Giles begins with the simple statement that he sees no reason to recede from his original position. I presume he means his present position, quoted above. He then proceeds to say that I have fallen "into the common fallacy of imagining that the number of anchylostomes found *post-mortem*, or, at any given stage of a case by expulsion by vermifuges, is any index of the number present at some previous period." This statement is utterly untrue, as will be seen by the following quotation, among many others, that could be given, from page 54 of my report. "In view, however, of the argument that when the worms are only found in small numbers during life or after death, they may have been present in much larger numbers at some previous stage of the disease, which is true in exceptional cases, it is difficult to absolutely exclude their action in this way. A means of doing so has, however, been found in the blood changes, as will be shown in the next section."

Now, how does Dr. Giles attempt to meet the fact, which has so widely been accepted as fatal to his views, namely, that the type of the anæmia of uncomplicated cases of *kala-azar* (ninety-three per cent. of all cases) is so totally different from that produced by anchylostomiasis, that the two diseases can be absolutely differentiated by means of a full examination of the blood alone. He admits the fact, for he writes: "Dr. Rogers has worked out a most ingenious and scientific method of differential diagnosis between the cachexiæ of achylostomiasis and malaria respectively." His only attempt at a reply is, "I fear that his method can have little beyond an academic interest to the practical physician of Assam." This is, of course, in no sense of the word an answer to the conclusive evidence against his view which is furnished by these blood changes, and until he is able to meet these facts, I shall hold that Dr. Giles has no scientific grounds for continuing to maintain his view that anchylostomiasis is an essential factor in the production of *kala-azar*.

In replying to my statement that the probable reason why Dr. Giles so under-estimated the malarial element in *kala-azar*, was because he

only worked in the plains in the cold weather, when the fever is at a minimum, and was engaged on microscopic work in the hill station, Shillong, throughout the rainy fever season. Dr. Giles writes: "I have little doubt that the fact that his study of the disease was mainly made at a time when the true cause of the enhanced mortality is marked by the seasonal prevalence of malaria, has much to do with the mistake into which he has fallen." This statement, again, has no basis in fact, for I spent the whole eleven months, from April 1896 to March 1897, with the exception of parts of September and October, in a continual study of the disease in the plains.

I shall return to Dr. Giles' other arguments presently, but a word may be said here with regard to his concluding paragraphs, in which he charges me with an ignorance of tropical diseases, on the ground that, before going to Assam, my experience was limited to two and-a-half years with a Native Regiment "where one's patients are all persons specially selected for good physique and stamina." This personal attack, which reflects more on the Government who selected me for the responsible post than on myself, would not require any answer from me if it had the bare merit of being true. As, however, apart from the question whether malarial diseases are so uncommon in India, that even a native regiment affords no field for their study, and apart from the fact that I was selected for the post mainly on account of the great attention I had paid to the subject of malarial fevers, my experience was not as limited as Dr. Giles is pleased to assume, for I had regularly attended Civil Dispensary work in the Panjab, North-West Provinces, and in Bengal, and had also officiated as Civil Surgeon in the last-named province, and that, too, in the height of the malarial season. In Dr. Thornhill's paper there are many incorrect or exaggerated statements, which it would take too long to point out in detail. In his first column, for example, he says that I claim to have proved that *kala-azar* has "the properties of infection as in the case of small-pox," and again, he denies that anyone ever said *kala-azar* was caused solely by anchylostomiasis. It will be sufficient to refer Dr. Thornhill to pages 117 and 125, in refutation of the above mistakes.

Passing on at once to the essential point at issue between Dr. Thornhill and myself, namely, the question whether I have proved that anchylostomiasis is not a factor in the production of *kala-azar*, Dr. Thornhill sums up his detailed argument on this head as follows: "I submit that it is futile for Dr. Rogers, on the evidence of these twelve clinical cases and of the twenty-five *post-mortems* he gives on page 104, and in face of Dr. Campbell's and Dr. Dobson's evidence which he himself records on pages 53 and 54 of his report, to claim that he has incontestibly established that anchylostomiasis was not a factor in producing *kala-azar*."

In the first place, what is the evidence of Drs. Campbell and Dobson? These observers found the worm present in a smaller percentage of *kala-azar* cases than they are in healthy people, while in the only cases in which the numbers found were recorded, the average worked out to be 10·4. This evidence, as far as it goes, is then entirely in my favour, not against me as Dr. Thornhill assumes. This is all the evidence on this point that Dr. Thornhill credits me with.

Now, on page 120, I wrote as follows under the heading—Is *kala-azar* anchylostomiasis?—"In reply it may be said that I have shown these parasites to be present in sufficient numbers to complicate the disease as proved by their effect on the type of the anæmia in under seven per cent. of *kala-azar* cases, while in eighty-three per cent. of a series of cases taken in as early a stage as possible, and which had not been previously treated with thymol, there were less than twenty worms, a number which I have proved to have no effect whatever on the blood of healthy persons in the Nowgong Jail, while Dr. Thornhill, of Ceylon, admits that fifty or less anchylostoma form a number altogether too small to have any deleterious effect. It is also worthy of note that Dr. Dobson found less than twenty worms, anchylostoma, to be present in eighty-two per cent. of 547 healthy coolies which he examined at Dubri, which agrees with my results in *kala-azar* cases. Again I find fewer anchylostoma on the average in a series of 25 *post-mortems* on *kala-azar* cases than I do in healthy men who had died of from accidental causes, and in only about a tenth of the number that I found to be the average of my cases of anchylostomiasis. Once more, in a series of 200 cases of *kala-azar* on a tea garden, 96 per cent. died, although the factor of anchylostomiasis was excluded from them by the use of thymol in an early stage of the disease. .... These facts prove incontestibly that whatever *kala-azar* may be, it is not anchylostomiasis, and what is more, the latter disease is not even an essential feature in the production of *kala-azar*, and only complicates it in six or seven per cent. of the cases, just as it occasionally complicates every disease in Assam, especially on tea gardens, and also, I have no doubt, in nearly every part of India." Further detailed evidence on this point is also given on pages 53, 54 of my report.

It scarcely seems necessary to add anything to the above quotations, in order to prove that Dr. Thornhill has argued from isolated facts arbitrarily selected by himself, and has entirely ignored much of the most important evidence that I have recorded on this head. I might just as fairly taken Dr. Thornhill's admission that fifty anchylostoma are harmless, and have argued

that he has not proved that they are ever harmful. It will be seen that the twelve cases on which Dr. Thornhill says I mainly base my argument are not even mentioned in the summary of the evidence on the point, for it would have been absurd to attempt to argue from twelve cases, however minutely observed. Moreover, in order to make use of them, he makes various assumptions as to the length of their stay in hospital, the number of them that had had thymol, and the amount of diarrhœa they had suffered from. Now, as a matter of fact, these assumptions are not correct, so that his argument, such as it is, falls to the ground. Moreover, it is not logical to argue from exceptions in order to prove a rule, for although it is doubtless true that in some cases there may be very few worms present in the later stages of anchylostomiasis, yet it is utterly illogical to argue from this that all or nearly all of my cases, in which a few worms were found, must have had large numbers in an earlier period, even if his assumptions were true. On the contrary, it must always be borne in mind that a large percentage of all diseases in Assam, as in other parts of India, must necessarily harbour these worms in small numbers. The same remark applies to the twenty-five *post-mortems* on *kala-azar* cases, in only two of which were more than fifty worms present, the average being twenty-one, which is less than that found in seven healthy men who died from accidental causes, in which they averaged thirty-five, two of them having over fifty worms. The slightly smaller number in *kala-azar* cases than in healthy persons I attributed to the fact that some, but by no means all, of the former had been given thymol. On the other hand, the average number of worms in four *post-mortems* on anchylostomiasis cases was 200, while in half of Sandwith's eighteen *post-mortems*, from 170 to 863 worms were found *post-mortems*.

With regard to the changes in the intestinal mucous membrane, I may say that I look on thickening and scarring as typical of anchylostomiasis, while atrophy and pigmentation I believe to be mainly due to malaria; but I am further studying this point microscopically. The above provisional opinion is strengthened by the fact that Dr. Sandwith has never seen such pigmentation in anchylostomiasis in Egypt, where malarial complications are rare.

I have shown, then, that Dr. Thornhill's arguments are based on a consideration of but a fraction of the evidence I have recorded, and that, too, aided by assumptions that are unwarranted. I will now proceed to show, chiefly from his own writings, that the facts which I have adduced, and which he has ignored, do completely disprove his contentions. The question is really a very simple one, if the main facts that are known are carefully borne in mind.

(To be continued.)

A Mirror of Hospital Practice.

CASE OF SUPPOSED CEREBRAL TUMOUR, TREPHINING.

BY SURGN.-LIEUT.-COL. W. K. HATCH, M.B., F.R.C.S.

THE patient, a man of 40 years, was admitted under the care of Surgeon-Major Lyons in January, 1897. He had been a painter and lately a butler by occupation, but owing to the occurrence of fits which caused him to fall suddenly and to break whatever he had in his hands, he sought admission into hospital. He had suffered from small-pox and malarial fever, and for the last nine or ten years from fits, which he said came on suddenly, on alternate days, and sometimes three or four times in the day; no syphilitic history could be ascertained. The fits were preceded by giddiness and headache for half an hour, after which he would fall suddenly to the ground and become unconscious. The family history was quite satisfactory. He was emaciated and had an anxious look, and appeared to be in pain, pain was complained of in the head and also in left shoulder. The left eyeball seemed rather more prominent than the right. His pulse was slow and incompressible, 56 per minute. There was a small wound in the scalp, four inches behind the left ear, another on left shoulder, and a scar over left orbit, all said to be due to falls. The heart and lungs were normal, also the abdominal viscera, and the urine shewed no signs of phosphates, albumen or sugar. As to the duration of fits, no statement could be obtained; but there was no foaming at the mouth or biting of the tongue. Vision was weak; no signs of anæsthesia or hyperæsthesia were present. The patient walked with a little difficulty owing to giddiness, but paralysis was not present. Further examination of the eyes showed doubts of his neuritis. The bowels were confined. On the 16th January, that is, on the 3rd day after admission, he had a fit which lasted a minute only, it began with shaking of the left upper extremity, twitchings of the facial muscles and convulsive movements of the whole body followed, the teeth chattered and saliva dribbled from the mouth, pain was felt in the occipital region. He became quite unconscious, the temperature was normal; next day he had another fit, and he appeared very dull afterwards and answered questions slowly. An enema was given, and the bowels were opened; but next day the patient did not know anything about it, and complained of feeling very giddy. After this he gradually improved and became more intelligent until the 27th, when his temperature rose to 102·6° in the evening; there were no fits observed in the interval, though it is possible he may have had one, at all events the headache was less, some blood was noticed in the stools, and two hæmorrhoids came down. On the 29th another fit came on when he was lying on

is not a better subject for lithotomy than the European I am unable to comprehend by what method of reasoning it is alleged—and I observed that this has been persistently alleged in the medical journals—that the former is a better subject for litholapaxy than the latter.

I am now, however, in a position to speak with authority in this matter, having had considerable experience of this operation both in India and in England. Last year I had 29 operations for stone in the bladder, and during the short period since I settled in practice in London I have performed 45 operations—*viz.*, 43 litholapaxies and 2 lithotomies, all of which were successful.

I find, however, that there are some interesting features of contrast between my practice in India and in England. In the first place I find that the average age of 40 adult Europeans operated on by me is fifty-six years, nearly eleven years greater than the average age in the adult natives of India. Mr. Cadge of Norwich, in his article on "Lithotrity," &c., in Heath's "Dictionary of Surgery," in contrasting the results obtained in my first series of 108 litholapaxies performed in India with a somewhat similar series of cases by Sir Henry Thompson in England, notices this marked difference in the average age. It is simply due to the fact that the "expectation of life" in India is probably about ten years less than in England, a native of India being at fifty years of age comparatively as old as a European at sixty years of age. Secondly, the average weight of the calculi removed by me from adults in London is 149gr. as compared with an average of 309gr. in India. This is a point to which Mr. Cadge also called attention in the article referred to, the average weight of the calculi removed by me in India in the series referred to having been nearly two and-a-half times greater than that of the average of Sir Henry Thompson's series. Thirdly, the very much larger proportion of prostatic patients met with in England than in India. Before I commenced practice in London my friends and present colleagues at St. Peter's Hospital, Mr. Reginald Harrison and Mr. Swinford Edwards, frequently said to me that they believed that in the surgery of stone one met the complication of hypertrophy of the prostate much more frequently in England than in India. In holding this view I find they were perfectly correct, for amongst 39 adult males on whom I performed litholapaxy in England there were 13 suffering from hypertrophy of the prostate, or one-third of the whole, a very much larger proportion than one meets with in India and which may to some extent account for the much larger number of recurrences of stone met with in England than in India. It will be noticed that in no instance in this series was the co-existence of hypertrophy of the prostate with stone a bar to the performance of litholapaxy, though in many instances there was very great enlargement of the gland present.

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BY LEONARD ROGERS, M.D., M.R.C.P., B.S., F.R.C.S.,
SURGN.-CAPTAIN, I. M. S.

(*Continued from page* 213.)

THUS, in the first place, Dr. Thornhill admits that "No advocate of the harmfulness of the parasite has ever written or hinted that *a few* parasites would cause symptoms, and it is quite time that this fiction was dropped." (I quite agree with him here) "All advocates of anchylostomiasis have always stated and written that the *continuous* presence of *a considerable number, probably not less than* 500, of the parasites in the intestines for a considerable period, probably six to twelve months at least *is, as a rule*, necessary to produce symptoms."—(*Indian Medical Gazette*, January and March 1896.) Again, in his congress paper, he quotes Lutz as writing: "It may be said that, in adults, in the absence of any complication, and in whom the disease runs a tolerably quick and uniform course, do not begin to show symptoms until the number of anchylostoma passes into hundreds, so that when pronounced general symptoms are present, 300 to 500 parasites may be set down as present in the duodenum." Dr. Thornhill continues: "I submit, therefore, that no inference as to the harmlessness of these parasites can be drawn from Dr. Dobson's cases, nor from any cases, even when hundreds, say even 500 or more, are proved to be present, *unless it be shown that they were present for a period of, I should say, six to twelve months at least.* I submit that it is manifest that small numbers may be present even for long periods, and large numbers for short periods, without causing serious or any symptoms, though, of course, the effects produced will vary according to the state of health of the host, *i. e.*, even a small number may produce serious symptoms, even in a short time, in an already debilitated host." Again, it should be remembered that, in the St. Gotthard Tunnel outbreak, as many as 2,000 and 3,000 worms were found in some of the cases. (The italics are all Dr. Thornhill's.)

Next, it must be borne in mind that Dr. Dobson's valuable researches have proved that upwards of eighty per cent. of healthy coolies, imported from a wide area of India, harbour this parasite, but in only three per cent. of them were over fifty of these worms found, while in

eighty-two per cent. of them, less than twenty were found. Among healthy Assamese, both Dr. Dobson and myself found these worms in from sixty-six to sixty-eight per cent. of those we examined, so that they appear to be slightly less common in Assam than in most other parts of India. It is evident, then, that a large percentage of cases of every disease in Assam (including *kala-azar*, must necessarily be accidentally complicated with small numbers of these worms, and that in order to show that they are a factor in the production of any given disease, it will be necessary to prove that they are present in actively harmful numbers, say 300 to 500, and that they remain present in such numbers for several months at least. If, on the other hand, it is found that, in *kala-azar*, these worms are not present in the early stages, or throughout the disease in actively harmful numbers, it will be evident that they are not a factor in the production of this terrible disease, while if it is also found that they are only present in about the same numbers that they are in both healthy people, and in those suffering from other diseases in Assam, then it will be evident that their presence is purely accidental.

The question largely turns on—What number of these worms may be safely taken as being too small to be capable of producing any symptoms? I will take Dr. Thornhill's own estimation. In his congress paper he wrote—"In the vast majority of Dr. Dobson's cases he gives the number of the parasites present as less than fifty, a number altogether too small to have any deleterious effect." This statement I have proved to be correct, by showing that such numbers have no effect whatever on the blood of Assamese people (page 85).

Now, I examined a series of cases of *kala-azar* in an early stage, before many of them showed any clinically evident anæmia, and who were not in the least degree "debilitated," which excludes the possibility of the action of large numbers of the anchylostomum for any length of time previously to their coming under observation. These cases were further re-examined from time to time during their comparatively short illness (for the great majority of them died within six months of the onset of symptoms), and yet I found less than twenty worms present in any stage of the disease in eighty-three per cent. of them. Moreover, this is almost the exact percentage in which such small numbers are found in healthy people. I have also proved that such a number has no effect whatever in producing anæmia, while Dr. Thornhill admits that two and-a-half times this number is "altogether too small to have any deleterious effect." Yet he would have us believe that it is owing to the accidental presence of these few minute worms that the malarial fever, which I have shown to be the constant and essential feature of *kala-azar*, gains the extraordinary power of being able to spread up 200 miles of the Brahmaputra valley in the course of the last twenty years and that, too, in the absence of any change in the districts traversed by it, which could account for its outbreak, and to attain such a virulence that it has wiped out about one-fifth of the inhabitants of the affected tract, and was attended by a mortality of upwards of ninety per cent., even when treated throughout by an experienced European doctor, thymol in an early stage being part of the treatment. The shorthand notes of the cases on which my statements are based would fill a volume larger than my report itself, yet Dr. T. Thornhill, totally ignoring the summary of evidence based on this laborious work, calmly picks out the notes of twelve cases, which were given, as stated in the place, as evidence on a totally different point, and the figures of twenty-five *post-mortems*, and argues from them that I have not proved my case. The most charitable explanation of his having done so, that I can think of, is to remember that he labours under the extreme disadvantage of never having seen a single case of the disease (*kala-azar*) on which he is writing, and that hence he must have read the details given in my report through the coloured spectacles of his experience of a totally different disease (anchylostomiasis) in Ceylon. This is the more to be regretted, as I should be one of the first to acknowledge the educative value of Dr. Thornhill's writings on anchylostomiasis, even though, I think, he has somewhat exaggerated their prevalence and importance.

As Dr. Thornhill has done me the honour of devoting so much attention to my report, he will doubtless be glad to hear that I returned him the compliment of examining the Ceylon Medical Reports for the years succeeding the appearance of Sir William Kynsey's valuable pamphlet on anchylostomiasis, in order to compare the "terrible ravages of anchylostomiasis in Ceylon" with those of *kala-azar* in Assam. I was much astonished to find very little reference to the disease in these reports, while the late Dr. Macdonnel of Ceylon, whose valuable researches on the life history of this worm are well known, has recorded his opinion that the worm does very little harm in a great number of instances, although capable of destroying life under certain conditions. Turning to the figures, and bearing in mind Dr. Thornhill's statement that previous to attention being directed to the presence of the worm in Ceylon, cases of disease produced by it were returned under the heads of anæmia and dropsy, I find that, during the years from 1886 to 1893 inclusive, there was a steady increase in the number of cases returned as treated in the Ceylon hospitals and dispensaries under the head of anchylostomiasis from 0 to 37·4 per 1,000; but during the same period, there was a decrease under the heads of anæmia and dropsy from 127·3 to 24·4 per 1,000, nearly three times as great a decrease

under the latter as there was increase under the former. It appears from this that anchylostomiasis has been steadily and markedly decreasing during the last few years, and I congratulate Dr. Thornhill on it. Again, in 1894, in Dr. Thornhill's own province of Uvea, among a population of over 160,000, there was actually a recorded death-rate in the hospital from anchylostomiasis of thirty-two. And these are the terrible ravages that he compares to *kala-azar* in Assam! Why, as many deaths from *kala-azar* occur in one year, in a single village, in parts of Assam!

As my views on the subject of the damage done by the anchylostomum have been mis-quoted, I will take this opportunity of saying that I believe that a large percentage of the natives of most parts of India harbour this worm in small numbers, but that this does not constitute anchylostomiasis, which may be defined, after Dr. Thornhill himself, as the morbid state produced by the past or present existence of a considerable number of anchylostoma. If, however, they are present in large numbers, such as 500, for six months to a year, they will certainly produce anæmia, which, when it has been proved to have been so caused, may rightly be called anchylostomiasis. Such uncomplicated cases are not very common among the natives of Assam or of Bengal and the North West provinces, etc., although they do occur. Intermediate between the above extremes, is a class of cases of anæmia which are partly caused by such factors as malaria, dysentery, venereal disease, or what I believe to be only too common, bad feeding, due not so much to deficiency of the total quantity, as of the proteid constituents; to one or more of which is added the presence of a considerable number of anchylostoma, say from 100 to 200, the drain caused by which might be withstood for a very long time by a healthy person, but when it is added to one or more of the other causes, produces anæmia, debility and other symptoms. These latter are more common than the former on tea gardens in Assam, and it is these cases that, I think, should be classed as anæmia of coolies in returns from tea gardens, for the reasons given in Appendix I of my Report. In such cases, a single administration of full doses of thymol is indicated, but repetition of this drastic measure will do more harm in debilitated people than the removal of the very few worms that may have escaped the first doses will do good.

I think I have written enough to prove that the anchylostomum is only accidentally present in very small and harmless numbers in *kala-azar*, but if anyone still doubts it, I reply that the differences in the type of the anæmia furnishes additional and conclusive evidence that this worm is not a factor in the production of the Assam epidemic fever. Dr. Thornhill devotes three pages to trying to throw doubt on the value of this evidence, much of which is occupied with unwarranted assumptions of possible errors on my part. He has, however, completely failed to grasp the primary significance of the figures recorded. For instance, he makes the extraordinary statement that I give a table to differentiate the blood changes in *kala-azar* and malaria, whereas, on pages 90-1, in explaining this table, I wrote: "these figures" (those of the anæmia of chronic malaria) "resemble those of *kala-azar* both in the great reduction of the white corpuscles, as compared with the red, in the high hæmoglobin value in the comparatively slight reduction of the specific gravity. It is evident, then, that the type of the anæmia in *kala-azar* is precisely similar, in all its details, to that met with in ordinary chronic malaria."

He goes on to suggest that the degree of the anæmia differs greatly in different stages of the two diseases. Precisely; yet, although the *degree* of the anæmia differs very widely indeed in both series of cases, yet *the type* of the anæmia is always so constant in each disease, that in no one case, out of about 100 in which these laborious estimations were made, did the type of the anæmia, met with in *kala-azar*, even approach that found in any one of the cases of anchylostomiasis, so that, by this means alone, the two diseases were absolutely differentiated. To give an example, in no case of anchylostomiasis was the hæmoglobin value, or colour index of the red corpuscles, as high as ·4, while in no single uncomplicated case of *kala-azar* (or of malarial anæmia) was it as low as ·5 and similarly with the ratio of the white to red corpuscles in the absence of leucocytosis due to fever, and with the specific gravity of the blood. Moreover, my observations find confirmation in those of Dr. Sandwith in the case of anchylostomiasis, and in those of numerous writers on malaria whom I have since consulted, as far as these changes had been previously worked out.

Again, Dr. Thornhill suggests that some of these cases may possibly have suffered from anchylostomiasis at an earlier period, and that the typical blood changes may have had time to disappear. Now I have found that, in cases of anchylostomiasis examined at intervals after their worms had been removed by thymol, the type of the anæmia persisted for many months after its degree has become much less. Further, in one case, a patient who had suffered from anchylostomiasis, but had been effectively treated by thymol, and was subsequently attacked by *kala-azar*, the anæmia showed the characteristic effect of the anchylostomiasis throughout her illness with the latter disease, so that it is obvious that if any of these cases had previously suffered from the worm disease, the blood-changes would have revealed it. Again, many of my cases were examined in the earliest stage of the disease, before they were in the least degree "debilitated," or anæmic, and before even a diagnosis of *kala-azar* could be made, but which subse-

quently died of the typical disease, yet throughout their illnesses, not only did they never harbour more than the smallest number of anchylostoma, but the blood changes were always typically malarial. Once more, I was able to correctly suspect the presence of a considerable number of anchylostoma (159 were passed after thymol) even before clinically evident anæmia had appeared in a case of *kala-azar*, solely from the type of the anæmia as revealed by the hæmoglobinometer differing somewhat from the regular malarial one, so that it is certain that the presence of any actively harmful numbers of these worms, at any stage of the disease would have been at once detected by the blood-examination. The evidence that is furnished by these blood changes is, then, by itself conclusive against the possibility of the anchylostomum being a factor in the production of the Assam epidemic malarial fever. Further details of these types of anæmia will shortly appear in the *Journal of Pathology and Bacteriology*, so need not be given here.

From the criticisms that have appeared, it might be thought that there is a close clinical resemblance between anchylostomiasis and *kala-azar*. The following table will show that this is very far from being so in the great majority of cases.

KALA-AZAR.	ANCHYLOSTOMIASIS.
Begins acutely with fever.	Begins insidiously with debility and indigestion.
Duration usually four to nine months.	Duration two to three years (Sandwith.)
Mortality under European medical treatment throughout, over ninety per cent.	Mortality three per cent., also 787 consecutive cases without a death. (Lutz).
Seasonal incidence: that of malaria.	Evenly distributed throughout the year.
Age: twenty-five per cent. below ten, fifty per cent. below twenty years.	Two per cent. under ten, fourteen per cent. only under twenty years old.
Great wasting of limbs, with large abdomen.	Fat or œdematous limbs, abdomen not large.
Intermittent or remittent fever throughout with occasional intermissions.	Fever never seen. (Sandwith). Subnormal temperature. (Giles).
Heart atrophied, muscle dark. Hæmic murmurs rare.	Heart large, muscle pale. Hæmic murmurs common.
Spleen always much enlarged, in fifty-six per cent. to or below navel. Average weight p.m., 2lbs. 5oz.	Spleen not enlarged. Average weight p. m., 10½oz.
Liver enlarged in ninety-three per cent. and dark. Average weight p.m., 3lbs. 13oz., against 2lbs. 5oz. in healthy Assamese.	Liver often smaller than normal. Average weight p. m., 1lb. 14 z.
Dropsy absent throughout in over half the cases, slight in feet only in thirty per cent. more. Very rare in face in last stage only.	Dropsy of face seen early. (Lutz). Always marked and often extreme in the later stages, in limbs, etc.
Tongue small and a dark.	Tongue large, pale and marked by the teeth.
Anæmia of variable degree, but of constant (malarial) type, increasing with fever, but improving in its absence.	Steadily progressive anæmia, of a totally different type to that of *kala-azar* and malaria.
Diarrhœa in the last stage of seventy-three per cent. slight constipation in three per cent.	Obstinate constipation in sixty per cent (Sandwith).
Marked pigmentation of liver, spleen, etc., with an increased amount of iron in the liver.	No pigmentation of the organs. Iron in liver reduced. (Bevan Rake).
Bone, marrow, dark red or purple.	Bone marrow, pale red.
Death from fever, or lung complications.	Death from dropsy and asthenia.

The above table shows at a glance that the symptoms and pathological change of *kala-azar* are those of malaria pure and simple, and that they differ so widely from those of anchylostomiasis, as in themselves to constitute a very strong argument against this parasite being, even in part, a cause of the epidemic fever. To take one point only. If the whole of the terrible mortality and the communicability of the disease is due to "anchylostomiasis and to no other cause," is it conceivable that in over fifty per cent. of the cases, from their commencement to their termination in death, should never suffer from the slightest œdema or dropsy, while, in thirty per cent. more, it should be limited to the feet only?

On pages 121—5 of my report will be found several other facts and arguments against *kala-azar* being a combination of malaria and anchylostomiasis, which have not been answered.

Thus the oft disproved fallacy that *kala-azar* was originated by the introduction of the anchylostomum into Assam, by the importation of coolies from India, is repeated by both Drs. Giles and Thornhill. The following facts will suffice to prove the absurdity of such a contention. *Kala-azar* began in, and was for at least the first eight years of its spread, entirely limited to parts of Assam into which no coolies from India were ever imported, while, on the other hand, it is still more than twenty years after its start, absolutely unknown in the upper part of the Assam Valley, which is precisely that into which the largest number of coolies have been imported from India. Again the disease *kala-azar* has never been known to spread from a tea garden to a neighbouring village, although the reverse has taken place. Once more I have recorded an instance of a certain tea garden in which both malarial fevers and anchylostomiasis were very prevalent, and yet not a single case of *kala-azar* had arisen at the time my report was written. In short, it may be said that, as far as the epidemic has yet spread, *kala-azar* has been prevalent in precisely the inverse ratio to the number of coolies that have been imported into the districts it has traversed.

On the other hand, I have shown that the symptoms and pathological changes of *kala-azar* are, from first to last, entirely malarial, while the plasmodium malaria is constantly present

in all stages of the disease. (The reason why it was only found in five of the twelve clinical cases, of which the notes were given, was simply because it only happened to have been looked for in those five out of that particular series.) It was also found in blood aspirated direct from the spleen during life. The forms seen were similar to three I had previously studied in Bengal. It has been argued that if I had found a new kind of malarial organism, that then it might have been accepted as explaining the epidemic fever, but that the intensification of an ordinary plasmodium malariæ cannot be accepted as the cause of the outbreak. To my mind, however, the latter is a more simple explanation of the origin of the epidemic than the former, for although we have some analogous cases of lowly pathogenic organisms becoming more virulent, owing to finding themselves under very favourable conditions, such as the succession of years of deficient rainfall in Rungpore, to which I have traced the origin of the Assam epidemic malarial fever; yet I know of no instance of a totally new and separate variety of such a widely prevalent disease as malaria, suddenly appearing and spreading in an epidemic form. Nevertheless, I may point out that, although I was not able to find any distinctive microscopical differences between the organisms met with in *kala-azar* and those of ordinary malarial fevers (except that the former seemed to be somewhat less frequently pigmented), yet it must be borne in mind that malarial organisms have been found in very various parts of the world which present very similar appearances under the microscope, but which cause probably distinct fevers; some of the African fevers, for example, just as there are many bacteria (with apologies to Dr. Giles for arguing "very nearly from a cow to a cowslip") of very similar shape, which have widely different properties. May it not be possible that when we are able to study the malarial organisms outside the body, and to cultivate them on different media, that those of *kala-azar* may prove to be specifically distinct from the ordinary Bengal and Assam plasmodium malariæ?

To pass on to my contention that the way in which *kala-azar* dies out of places and districts after a few years, proves that its spread cannot be due to anchylostomiasis. Dr. Giles admits the force of my argument, and he attempts to refute by making the following sweeping assertion. "Moreover, a little study of the subject should have taught Dr. Rogers that helminthiases wane and wax much in the same way as other diseases do." Precisely, what Dr. Giles had in his mind when he wrote the above very indefinite sentence is difficult to say, but this at least is certain, that I can find no reference in the writings of Lutz, Kynsey, Leucardt, Sandwith, etc., of any instance in which anchylostomiasis (which alone has any bearing on the question) waning or waxing to a degree that even remotely approaches the rapid rise and fall of the mortality caused by *kala-azar*. On the contrary, we have the evidence of Sandwith that this disease is as common in Egypt to-day as it was in the time of Griesinger (1851), and that from historical evidence "there is no reason for thinking that they were not equally common in the eighteenth dynasty (over three thousand years ago)." Again, Dr. Thornhill writes that in the absence of improved sanitation or migration of the inhabitants, anchylostomiasis would never die out of a place into which it had once been introduced, but would always be increasing in numbers it attacked and be slowly spreading to other places; and that if anchylostomiasis dies out of the places it has previously affected in the absence of such conditions, then it cannot be due to anchylostomiasis.

Now what are the facts with regard to the dying out of *kala-azar*. Dr. Thornhill states that I only give one instance of it having so died out of a place, namely, "that of Parakhasma at the foot of the Garo Hills." This is another instance of the way in which Dr. Thornhill has so misquoted and minimized the evidence that I have recorded in my report, as to convey an entirely false idea of the facts on which I rely. Anyone reading his sentence would imagine that the instance he quotes is that of a single village or place, whereas my sentence reads: "Between Parakhasma and Singimari (some seventy miles), it would be hard to find one single case of the real illness." As Dr. Thornhill quotes the very next sentence to the important and essential one which I have just given, he can have no excuse for this very unfair omission. Moreover, it is not even true that this is the only instance that I have recorded, for another will be found on page 4, and others might easily have been given. Now this tract of seventy miles was devastated by *kala-azar* between the years 1875 and 1881, to perhaps a greater degree than any other part of Assam, yet in 1887, the epidemic had so far died out that "scarcely a single case of the real illness" could be found by an official who had a long practical acquaintance with the disease in this very district. Moreover, in the ensuing ten years, the population of this district, namely, the Garo Hills, had increased considerably, and that at the very time that the next district of Goalpara, into which the epidemic had now passed, suffered so severely that there was a decrease of over eighteen per cent in the population during these ten years in the affected tract, while in the part of the same district north of the river, which was not attacked by the fever, there was an increase of nineteen per cent. in the population in the same period. Thus a difference in the population between the affected and unaffected parts of the Goalpara district in ten years amounted to over thirty-five per cent. due solely to the ravages of *kala-azar*. This district

has, for some years, been free of the epidemic, and I venture to prophesy that, in the next census returns, the population of the affected tract will have once more increased, as in the case of the Garo Hills. Again, in the Kamrup district, the estimated death-rate between the two censuses of 1881-91 from *kala-azar*, was 75,000, a very large percentage of which occurred in an area, the population of which in 1881 was but 172,125. I have also shown that, in certain areas, one-fifth of the land has fallen out of cultivation on account of the terrible death-rate from this epidemic. Before then Dr. Giles' assertion can be accepted as an answer to these facts, it will be necessary for him to furnish evidence to prove that anchylostomiasis can "wane and wax" to such a degree as to cause a decrease of nearly twenty per cent. in the population of a district within ten years, followed by an equally rapid decline of the disease, with a decided increase of the population of the same district in the very next decade.

Dr. Thornhill now falls back on an attempt to show that the large death-rate is equivalent to migration of the inhabitants, and in support, he quotes an instance in which the inhabitants of a small village are said to have died out. Here, again, he argues from an exception, for, in the seventy-mile tract, healthy people remained in the previously fever-stricken villages. He further suggests that thymol, as a potent sanitary measure, may partly account for the decrease of the disease. Unfortunately for this argument, the epidemic died out of the Garo Hills before thymol was introduced into Assam, while the amount used in the widely scattered dispensaries of the other districts has been far too small to have any such far-reaching results, as the slightest personal experience in Assam would have taught Dr. Thornhill. Similarly, with the water-supply, the improvement in which in Assam is practically limited to tea gardens. His remaining argument on this head is that the subsidence of the epidemic was due to the deaths of those who harboured most worms, so that "the deposition of their ova on the ground in their neighbourhood was either altogether stopped or very greatly diminished," is too absurd and illogical to require any refutation in the face of his frequently repeated argument that when a large proportion of the inhabitants of Assam harbour this worm in small numbers, some of them must have it in much larger and actively harmful numbers, and that a person who has only a few worms, nevertheless passes thousands of ova. According to these statements, when over eighty per cent. of the people have small numbers of the worms (less than twenty), then it is certain that some of them will have actively harmful numbers, namely, several hundreds; yet when a village is so badly affected that a large percentage of them are dying of anchylostomiasis, for which purpose they must harbour several hundreds of the worms for months, then the rest of the people living with them under the same conditions do not harbour even enough worms to continue the species, and that, too, when those dying of the disease must have been passing many thousands of ova every day for months at a time. To such straits is Dr. Thornhill reduced in trying to wriggle out of his admission that, in the absence of improved sanitation or migration of the inhabitants, the disease could not have died out in the way *kala-azar* has done if it were caused by anchylostomiasis. I am much indebted to Dr. Thornhill for this argument, which is so completely fatal to his case.

I pass on to the subject of the communicability of the disease, which it is now admitted I have furnished conclusive evidence of. I was careful to use the word communicability as far as possible, and I used it in its widest bacteriological meaning, namely, that the organism of the disease is capable of escaping from one person, and probably after an extra-corporeal existence in the soil, of getting into another person and reproducing the disease. In this sense all germ diseases and most helminthic ones are communicable in very various degrees. Exactly how the infection takes place in *kala-azar*, I do not profess to know, and my suggestion that possibly the plasmodium may escape through the lung of one person, by the same route that it probably entered, and, usually after having passed through the soil, it may be breathed in by another person living in the same house or village, was put forward merely as a theory which best explained the known facts, but which I expressly stated cannot be proved until the organism can be found outside the body. It has been contended by Drs. Giles and Thornhill that it is not logical to argue from bacterial diseases to malaria, because the former are caused by vegetable, and the latter by an animal organism. Now several eminent authorities in England, whom I consulted on this point, all agree with me that my argument is sound, because, in the first place, it is by no means certain that the above classification is correct and final; and secondly, that even if it is, the difference between these lowest species in the biological scale is so slight that there is no reason to believe that they would act differently under like conditions. It is, perhaps, worth noting that Dr. Thornhill, in the very next paragraph to that in which he objects to my using this argument, makes use of a similarly based one himself.

To sum up my position, which is a very simple one. I maintain that I have proved that the Assam epidemic fever, known under the name of *kala-azar*, is *per se* from first to last, solely malarial in the causation, symptoms and pathological changes, and there is not one symptom, or morbid change in it, which is not met with in malaria elsewhere. It is acknowledged that I have proved it to be communicable, while I have

also shown that it arose owing to the fever of the very malarious district of Rungpore, becoming more and more virulent and fatal through a series of five out of six successive years of very deficient rainfall, such has never been known to occur in that district before or since as far as records are obtainable, and I have traced its spread from here across the Brahmaputra river, by the two main lines of communication, to the foot of the Garo Hills, and from there throughout its course in Assam. I have also shown that the epidemic spreads up the valley in the form of a wave of greatly increased fever mortality, dying out of the first affected places as it proceeds, and further, that the anchylostomum is not a factor in the production of the disease, and will not account either for its origin or its spread.

One of two things follow: either there is some other factor in the disease which accounts for its communicability, as for example some bacterium which cannot be cultivated or stained by means at present known; or it must be acknowledged that, in this instance, a malarial fever has become intensified by the extraordinary conditions mentioned, until it has attained the power of spreading as *kala-azar* has done, in the absence of any local changes which could account for such a marked increase in the malarial fever in each of the districts affected. The first hypothesis cannot be absolutely excluded, for it is impossible to prove a negative, but it is so extremely improbable as to become a practically negligible quantity—for the following reasons: In the first place, if there is some such undiscovered factor to which the communicability and terrible mortality of the disease is due, then it must be constantly present, and yet not reveal itself by one single symptom different from those of malaria. Secondly, I know of no analogous example of a widely prevalent germ-produced disease, which is not communicable in itself, but which is occasionally rendered so by the addition of some other organism. On the other hand, several diseases, which are caused by the lowest forms of biological life, are liable to be intensified under exceptionally favourable circumstances until they become communicable and epidemic. Further, there is good reason for believing that malarial fevers have become epidemic in this way in other places, as for instance, in the case of the famous "Burdwan" and "Mauritius" epidemic malarial fevers.

Dr. Giles very ingeniously suggests that these also may have been due to anchylostomiasis, because, forsooth, the Assam epidemic is, in his opinion, so caused. Now, apart from the fact that he has not attempted to meet the evidence that I have adduced to prove that this worm is not a factor in the production of the Assam epidemic, and apart from his omission to give any evidence to show that anchylostomiasis can "wane and wax" in any way approaching that which I have shown *kala-azar* to do, I may point out that a very careful perusal of all the literature of the Burdwan and Mauritius epidemic fevers has convinced me that the clinical and pathological conditions, recorded as having been present in these two epidemics, do not afford the least evidence for in favour of such a novel view as Dr. Giles. On the contrary, they entirely negative it. To take one fact alone: one quarter of the inhabitants of Mauritius died of fever within the first four years of the first outbreak of the epidemic, and this occurred in a population which could not be said to be "malaria-stricken for generations" (whatever Dr. Giles may mean by that expression), for it is quite certain that there was extremely little, if any, malaria in Mauritius before this outbreak, as is proved by the following figures. The average number of admission cases for intermittent fever per 1,000 among the troops in the island averaged 13·9, and the death-rate 34 during the eight years before the outbreak, while the admission reached 1789·3 and the deaths 17 per 1,000 during its height in 1867-68. The figures declined in 1872 to 36·4 and 2·1 respectively, which are the lowest ever recorded since the epidemic began, the average during recent years having been about 600 admissions and 8 deaths.

Finally, Dr. Thornhill devotes over two pages to condemning my recommendations, including what he is pleased to call "banter," in which he seems to be more at home than in arguing logically, or in correctly, or even fairly, quoting the evidence recorded in the report he has taken upon himself to criticise, without having had any personal experience of the disease it relates to. As he does not agree with my conclusions, his disagreement with my recommendations only indicates that they are probably the logical outcome of those conclusions. It will then suffice to point out that every one of my proposals (with the exception of the use of the Epidemic Diseases Act, which is not considered necessary at present) have been accepted and carried out by the authorities in Assam, who, Dr. Thornhill will pardon me for saying, should be at least as competent to judge as my critic himself. After all, the proof of the pudding is in the eating, so the following instance, in which my plans have been fully carried out with the most complete success, will form the fittest conclusion to this paper. I am indebted, for the following facts, to Dr. Dodds Price of the Nowgong district, who has had a unique experience of *kala-azar* on tea gardens, and who is in entire agreement with my conclusions on the subject. During the four years ending with 1896, there were over 200 deaths on one of his gardens from *kala-azar*, out of a staff of coolies which was maintained at a between 800 and 900. One of the worst affected lines was dealt with thoroughly on the lines laid down in section 10 of my report early in

1897. I saw both the old and the new lines, and there was no appreciable difference between them from the sanitary point of view, including their height and relation to low-lying land or to ground water-level, etc. Nevertheless, during more than a year which has elapsed since all the healthy people were moved out of the infected lines, when the epidemic fever was at a minimum, there has not been one single case of *kala-azar* among them in their new lines. That this is not due to any accidental cause, is proved by the fact that cases have continued to occur, though to a less extent than previously, in two other infected lines, in which my recommendations have only been carried out to a very partial extent.

Unfortunately it is very difficult to carry out equally thorough measures in numerous infected villages, yet it is evident that my recommendations are based on right principles, and may, therefore, be confidently expected to be successful in proportion to the completeness with which they are carried out, while in the case of tea gardens, the prosperity of which in several districts has been so gravely imperilled by the spread of *kala-azar*, this epidemic fever is now robbed of most of its terrors.

A FATAL CASE OF PLAGUE CONTRACTED AT A *POST-MORTEM* EXAMINATION.

BY SURGEON-CAPTAIN S. E. PRALL, B.S., M.B.,
ACTING 2ND PHYSICIAN AND PATHOLOGIST, J. J. HOSPITAL, BOMBAY.

A. B., a Native Christian, was admitted in the J. J. Hospital under my care with fever and cough on the 18th May, at 12 noon. His respirations were rapid, and he complained of pain in the chest and constant cough. The temperature was 102°, and the tongue heavily furred; there was no bubo at this time. The physical signs were those of pneumonia in an early stage, which I accordingly prescribed for.

On arriving at the hospital the next day, I was informed that the patient had died suddenly about two or three hours after my visit to him, and I determined to inspect his body. The lungs were much engorged but did not at all present the usual patchy broncho-pneumonia that is the common microscopic pulmonary lesion in plague: there were no petechiæ under the pleuræ; the heart was large and soft, and there were several small extravasations of blood under the pericardium; spleen large and soft, kidneys extremely congested, and there was a remarkable disparity in their size, the right being less than half the size of the left, though both were equally congested and resembled each other closely in every other respect. There was a slight fulness in the right groin, and I found on dissection that the inguinal glands and the glands on the external iliac vein showed the characteristic plague lesions, the inner coat of the vein itself being markedly affected. A diagnosis of plague was made, and my assistant remarked quietly to me that he had earlier in the examination cut his finger, at the same time showing me a cut about a quarter of an inch long in his right index finger. It was bleeding freely, and I recommended him to wash it thoroughly under the tap, and then to apply strong carbolic lotion; this he did, and subsequently cauterised it. However, on the 21st, he was taken ill with fever and laid up in his quarters, whence on the 23rd he was removed to the European plague hospital. I saw him there on the afternoon of that day; he was sensible but breathing rapidly, complained of pain under his right arm, where he had a glandular enlargement. His eyes were suffused and watery; he had intense headache; pulse large, soft and rapid, and his tongue was heavily coated with brown fur. The original cut had healed and was not to be seen at all. His symptoms increased rapidly in gravity, the fever especially rising to 105 or 106° Fahr. and he was kept constantly in a wet pack. When I saw him again on the 25th he was delirious and evidently sinking, and finally, in spite of all that Surgeon-Colonel Boyd, under whose care he was, could do for him, he died early in the morning of the 26th May. This was a most melancholy case, as the lad was a promising and industrious student and a conscientious worker in the pathological room, where he had been of the greatest assistance to me. I publish the case as a warning of the real danger that awaits those who make examinations of plague bodies.

[Two similar cases of fatal plague, contracted by *post-mortem* wounds at autopsies have lately occurred, we believe, in Calcutta.—ED., *I. M. G.*]

PLAGUE CONVALESCENCE PERIOD: ANALYSIS OF 108 CASES.

BY SURGN.-CAPTAIN G. S. THOMPSON.

IN the following analysis of the convalescence period of 108 cases of plague at the Parel Hospital, between February and July 1897, no patient, except one who deserted on the twenty-third day, was discharged until the temperature had remained normal for at least four days.

Those discharged in less than ten days from the hospital had either been kept under observation for some time previous to actual entry of their names in the admission and discharge book, or had had plague outside and were practically in a convalescent state on their admission and were detained until their clothing had been disinfected and their dwellings made ready for re-occupation. The shortest stay in