A discussion on ankylostomiasis / G.M. Giles [and others].

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A DISCUSSION ON ANKYLOSTOMIASIS

G.M.Giles et al.

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with a case of filarial disease escape infection? The presumption is that the whole community would become affected sooner or later. Yet many escape.

THE HOT WEATHER DIARRHŒA OF INDIA,

By Major W. J. BUCHANAN, I.M.S.,
Superintendent, Central Prison, Bhagalpur.

MY object in writing this paper is to call attention to a very severe form of diarrhœa with watery stools, which is certainly not cholera, but in many cases somewhat resembles it. It is not difficult to understand that as hot weather in Europe is an important, if not the chief, factor in the production of "summer" diarrhœa the extreme heat of India should be *a priori* even more so.

Bowel complaints of all kinds are common in Bengal, but it is here intended to refer only to one particular form. It might be called thermic diarrhœa, and it has been called "choleraic diarrhœa." It bears, however, the same relation to cholera Asiatica as does English summer diarrhœa or "cholera nostras." Clinically though it is very common in adults it has a strong resemblance to what is called "cholera infantum."

Before going further it may be well to give a short account of the clinical features of an acute case: The onset of the attack is always sudden, the patient having been apparently in his usual health up till a few hours before, then suddenly at night or in the early hours of the morning he is attacked with violent purging and vomiting, at first the stools are loose and fæculent, and the vomit contains the remains of the last meal; soon, however, the stools become abundant more or less colourless, and pour out from the rectum as if from a tap. The patient becomes collapsed with a shrunken cyanotic appearance, resembling, but in a somewhat less degree, that of cholera. The urine may be suppressed for several hours, the stools may number ten, twenty, or even fifty in the first half-dozen hours. In old or feeble persons the result may even be rapidly fatal. More usually the violence of the attack grows less in the course of the day; a reaction sets in, which, however, is not so marked as is the reaction stage of cholera, the temperature may rise from 101° or 102°F., the stools become fæculent and only loose or semi-solid, and in uncomplicated cases the recovery is usually rapid. In a few instances the inflammation of the small intestine goes on to an ileo-colitis and stools resembling dysentery persist for a few days. This is especially liable to occur if the patient has formerly suffered from dysentery. One might quote dozens of cases, but the following three will illustrate the different degrees of severity:

1. *Mild.*—J. S., aged 24, weight 107 lbs. On April 6th, 1899, admitted to hospital at 5 A.M., with violent loose watery diarrhœa—10 to 15 stools in the hour. Urine passed, no cramps, no collapse; voice good, recovery rapid in the course of about six hours.

2. *Severe.*—B. D., aged 38, came to hospital on March 21rd, at 5 A.M., passing thin, very watery stools with urine, vomiting frequent, partially collapsed and cold. When seen by me at 6 A.M. he was in this state; no urine for the past hour, but frequent scanty stools; very restless, face shrunken, voice clear, pulse rapid but perceptible; no cramps, tongue clean, urine remained suppressed for 21 hours. On second day the stools were semi-solid, and next day he was practically convalescent.

3. *Fatal.*—N. K., an old man, came to hospital at the same time as No. 2 above. Stools very watery, vomiting and nausea frequent; no urine passed, body cold, but pulse was just perceptible. In a few hours he was quite collapsed passing frequent watery brown-coloured stools. Tongue soon became dry, the thirst was great. The stools never resembled "rice water." The diarrhœa continued, and he died at 4 P.M., after about 12 hours' illness. At the necropsy old pleural adhesions were found. The spleen was enlarged (8 by 5 by 2 in.), no rice water stools in the intestine, but drab-coloured fæcal matter; one old patch of ulceration at the sigmoid flexure.

Albuminuria is often found in these cases, but does not last usually longer than a few days, where the kidneys have been previously sound. Constipation for a few days after recovery is common. Microscopic examination of the stools shows abundance of epithelium, starch grains, particles of grain and husk and numerous bacteria.

Treatment.—The treatment of these cases is not difficult, for attacks lasting only for a day food is not urgent, but boiled milk or milk and Mellin's food may be given. Brandy or rum will often be necessary. As regards drugs, it is unwise to interfere with Nature during the early stage of the diarrhœa, but by the time a patient is usually seen by a medical man many watery stools have been passed, so I usually at once give chlorodyne or camphoridyne (the tinct. chloroformi et morphinæ co. of the new *British Pharmacopœia*), sometimes with small doses of brandy, enough to control the diarrhœa. Then when the diarrhœa has become much less a dose of castor oil will set the patient aright. Care should to be taken with the diet till the stools have become solid.

Etiology.—As regards the etiology of the disease this resolves itself into (1) bacterial and (2) predisposing causes. I have nothing certain to say about the bacterial cause. It is believed by many that the specific micro-organism of what Dr. Newsholme calls "epidemic diarrhœa" in England is the bacillus enteritidis, but as this micro-organism has already had attributed to it two diseases so entirely different as the well-known "rice pudding" epidemic of St. Bartholomew's (a sudden widespread and non-fatal diarrhœa), and the so-called colitis of English asylums (a fatal disease which anyone with tropical experience would not hesitate to call dysentery), it is clear that one should hesitate to attribute such a widespread disease as summer diarrhœa to it also. We are on safer ground in discussing the predisposing, or rather the "exciting," causes. Granted the presence of the organisms of decomposition and of fermentation in the intestinal tract the actual exciting causes are as follows: (1) Badly cooked food; (2) hastily eaten food, or over-eating; (3) unripe or over-ripe food; (4) raw grain, eaten either carelessly or on purpose; (5) drinking too much water after or before food; (6) too rich food, curried and spiced; (7) badly prepared grain, especially maize when weevil-eaten, as it usually becomes by the end of March in India; (8) over indulgence in alcohol, especially drinking freely at a big dinner by one usually abstemious, or a mixture of liquors—what is often called "upsetting the liver"; (9) excessive use of inferior fibrous vegetables, and many other similar causes. I have actually seen cases which were attributed to one or other of all the above causes. Some of them apply only to European habits and others to the habits of natives of India. In gaols I am always on the look-out for bad cooking when such cases occur, for example, on one occasion in Midnapur Central Gaol I remember finding, one morning in May, no fewer than 125 such cases of violent diarrhœa admitted in one night; the cause was not far to seek. On the afternoon before a very violent cyclone had raged, with the result that the cooks were driven out of the cookhouse and the fires had to be put out, so that the evening meal of about 1,000 prisoners was issued in a half cooked or rather half raw condition. On another occasion I had a series of 25 such cases admitted to hospital, owing, as I believed, to the temporary use of some bags of weevil-eaten maize, which were far worse than was expected. On this occasion it was remarkable that the cases only occurred from those on maize-flour diet, and not a single case among a large number of Bengali prisoners who got rice at all meals. Cases due to the use of over-ripe or half ripe fruit must have occurred in the experience of all who have lived in the tropics. One case I well remember. The patient confessed to having eaten the raw seeds of the jack-fruit tree, he suffered severely, having had suppression of urine for 36 hours and after the bowel complaint was over he suffered from successive crops of boils and his whole outer skin peeled off in handfuls, which symptoms I attributed to the endeavour made by the irritant poison to escape by the skin.

But while such factors as unripe fruit, raw grain, etc., are certainly important, I do not think that they are the whole cause, something more is necessary, and this something is the influence which is exerted by the hot weather on putrefaction or fermentation in the intestines. My reason for so concluding is as follows: I have seen all the above exciting causes in operation in cold season, but it is seldom or never that this violent form of diarrhœa is then produced, food may be badly cooked in the cold weather as in the hot, grain may be as badly husked, unripe fruit may be eaten, idle prisoners will eat raw grain in the cold weather as well as in the hot, but it is very rare for us to meet with these cases of diarrhœa in the cold weather. The same remarks apply to summer diarrhœa in England.

Diagnosis.—It is obvious that these cases in India must often need to be diagnosed from cholera. In cases where a bacteriological examination is not possible one must rely on the following considerations: (1) The presence or absence of cholera in the neighbourhood; (2) the low death-rate for the diarrhœa, and the usually high initial death-rate for cholera;

(3) clinically by the fact that cramps are seldom present in diarrhœa, or if present, rarely severe, the collapse is not so complete as in cholera, the voice is never so low and weak as in cholera, the pulse though weak and small is never so low as in cholera, the stools are never the pure "rice water" of the graver malady, but always somewhat stained with fæcal matter. The reaction stage is less marked than in cholera cases, and some irritant cause is also usually to be found. I am strongly inclined to believe that in this form of "hot weather diarrhœa" the clue will be found to many of those mysterious cases of isolated sporadic attacks of "cholera." If "summer diarrhœa" can occasionally kill adults in Europe, I am certain that "hot weather diarrhœa" in India can do so.

Colonel KENNETH MACLEOD said: This paper on hot weather diarrhœa raised two important questions; first as regards the distinction between cholera nostras and cholera Asiatica, and secondly, assuming that these are essentially different, or at any rate differently caused diseases, what the organism or toxin is which gives rise to the former. In the absence of bacteriological investigation the question of diagnosis is often very doubtful in India; but experience confirms the view taken by Major Buchanan in this paper, that these cases which are very common both in Europeans and natives, adults and infants, in the hot weather are not cases of cholera Asiatica. We are ignorant of what the precise causation is, and it is best that we should acknowledge this, and content ourselves to await further research, which is urgently desirable.

THE DISEASES OF GOORKHAS.

By ANDREW DUNCAN, M.D., M.R.C.P., F.R.C.S.,
Physician to the Seamen's Hospital, Royal Albert Docks, London.

DURING the last year of my service in India, I had the good fortune to be associated with many Goorkha regiments, and as the result of this experience I found certain facts in their medical history to exist which I had not previously noticed in connection with the men of other native regiments. These facts I propose to briefly put before you.

Malarial Fevers.—Shortly after taking over medical charge of the 2nd (P.W.O.) Goorkha Rifles I was struck with the intractable character of the malarial fever in many cases amongst the men. The fever proved to be peculiarly resistant to the usual anti-malarial remedies, so much so that in no other race in India have I experienced such difficulty in combatting the disease. A number of drugs were tried, and I came to the conclusion that two methods of treatment only were of avail in these cases. First, the treatment by the rectal injection of 15 to 20 grs. of quinine. Should this not avail, then the only measure left was sick-leave to another district. Cases in which for three or four weeks 30 grs. of quinine had been taken by the mouth without avail would often yield at once to 20 grs. by the rectum. Next to quinine, kreat gave the best success in these severe cases.

Phthisis.—The manner in which phthisis occurs and its course, is also somewhat peculiar. Cases of an irregular fever used to come into hospital. Repeated examination of the lungs, which I was taught by experience never to omit, for many weeks would show no physical sign. Suddenly, one morning I would find sub-crepitant *râles* commencing at the apex. The early symptoms then almost invariably advanced with great rapidity. In fact, I have never seen such rapid cases of tuberculous disease of the lung as I have seen in Goorkhas. And in reference to this I may perhaps mention an extraordinary case I once had under my care. A Goorkha had been for three or four weeks in hospital when the signs of rapid disintegration of the lung appeared. Subcrepitant *râles* were succeeded by humid crackling *râles*, and a large mass of tuberculous glands appeared above the clavicle. So ill was he that for two or three days I went to the hospital fully expecting he had died in the night. But one morning, to my astonishment, he appeared much better, and, to cut a long story short, the signs abated, the gland enlargement disappeared, and eventually the man went out perfectly well. The treatment was by inhalation of creosote. I do not think there could be any doubt as to the accuracy of the diagnosis.

Mumps.—Goorkhas are very prone to mumps in their native country. Nearly always in the few weeks immediately subsequent to the joining of the new recruits, mumps would occur in the regiment. And the same remark must be made with regard to measles.

Ophthalmia.—Shortly after joining my regiment I was struck with the comparatively large number of cases of ophthalmia met with. I was at first inclined to suppose that the Goorkhas were in some peculiar way predisposed to this disease. However, I was called in one day to attend the wife of a Goorkha Rifle officer for phthisis, and was at once struck with the bad ventilation of her hut. Examination of the huts of the married and bachelor lines now showed the following important differences: Bachelor huts had no doors, had large airy windows opposite one another, fully 2 feet square and kept open; the food was cooked in separate buildings. The married huts were irregular in shape, windows smaller and not opposite one another, and generally closed with articles of dress, and the food was cooked in the huts. The inner room was dark and the atmosphere not fresh.

Next the incidence of ophthalmia was as follows:

1895	Married, 30	...	Single, 11 cases.
1896	,, 68	...	,, 7 ,,
1897 up to Aug. 28th.	,, 50	...	,, 12 ,,

On August 28th the regiment went on service and ophthalmia at once disappeared. I inquired of the medical officers of the other Goorkha regiments and found no such incidence of ophthalmia, with the exception of the 3rd Goorkha Rifles. Here the lines consisted of four airy double-storeyed barracks for a certain number of bachelors; the remainder of the latter and all the married men living in a series of tents closely aggregated under the brow of a hill. Twenty-one cases of ophthalmia occurred in 1896, of these 20 came from the latter barracks.

Enteric.—It has been often stated that the Goorkha soldier is prone to enteric. This has not been my experience. I have only seen one case of enteric (and this was judged to be enteric from the typical nature of the temperature chart chiefly) during my service in India amongst native troops. The case was however in a Goorkha. Surgeon-Major Armstrong, the Residency Surgeon in Nepaul, informed me that in his experience the Goorkha race is by no means prone to the disease. He has never had a case in hospital, civil or military in Nepaul, and has only seen 2 cases in females.

A DISCUSSION ON ANKYLOSTOMIASIS.

I.—Major G. M. GILES, M.B., F.R.C.S., I.M.S.,

Sanitary Commissioner N.W. Provinces and Oudh, India.

THE phenomenon of parasitism is so widely diffused throughout the animal kingdom that it is more than probable that the evolution of the parasite has commonly coincided with that of the host, so that it is by no means surprising to find Dr. Sandwith, of Cairo, quoting a translation of a Hieratic papyrus 3,450 years old, which shows that even then ankylostomiasis was known as the A A A disease, though the writers assumed that the worms were the effect instead of the cause of the disease.

Quoting still from Dr. Sandwith's paper (written for the eleventh International Medical Congress held at Rome in 1894) we find that the earliest mention of this special anæmia in modern times was from Brazil in 1648, and in Guiana in the beginning of the eighteenth century, but it was not till 1843 that Dubini, of Milan, first discovered the parasitic cause of the disease. After this Pruner (1847), Grasinger (1851), and Bilharz found it to be very common in Egypt, but these discoveries attracted no particular attention until the memorable outbreak of this disease amongst the workmen engaged on the construction of the St. Gothard tunnel brought its importance prominently to the notice of the medical world.

A few years later Leichtenstern and Lutz demonstrated the existence of a free stage, but owing possibly to climatic difficulties did not succeed in rearing the so-called rhabdites to sexual maturity; Lutz describing the still immature worms as becoming "encapsuled" and subsequently calcified. I believe myself that this is really a death and not a life history, and that the appearances described relate merely to the slow destruction of the embryos placed under conditions unsuitable to their well being.

Since the St. Gothard outbreak the disease has been recog-

nised in all the warmer parts of the old and new world wherever civilisation and sanitation are so little developed as to admit of its progress. It has however not yet been certainly found above latitude 52° N., and above 47° N. the parasite apparently can only flourish in the sheltering warmth of mines.

This was then the state of our knowledge in 1889. As to the serious effects of the parasite on its human host all were fully agreed, for no one had then advanced the fantastic notion that it was a mere harmless tenant of the human bowel.

It was at this date the Indian Government became alarmed at the very high mortality prevailing in Assam, alike among the indigenous population and among labourers brought from other parts of India to work in the extensive tea-gardens of that province. In the case of the latter, the causation of the mortality had already been made out to be ankylostomiasis by Dr. Ruddock, though it had unfortunately come to be known by the erroneous denomination of beri-beri, while among the indigenous population the mortality was called kala-azar. I will not here enter into the old controversy as to whether this increased mortality known by that name is due, as I maintain, to ankylostomiasis *plus* malaria, or to malaria *plus* ankylostomiasis, as advanced by Captain Rogers and Major Ross. We are all agreed that both these causes of cachexia are present in Assam, and it is probable that our real differences are of less dimensions than the volume of the controversy would suggest. It is, however, obvious that, from my particular point of view, the exact ascertainment of the life-history of the worm was the first importance. In the course of the investigation I was enabled to bring to light many entirely new points. Of these by far the most important was the demonstration of the fact that the full life history of ankylostoma doudenale is an example of dimorphism or heterogenesis.

My attention was first drawn to this by my finding sexually mature nematodes, the females heavy with eggs, which were absolutely identical in form and dimensions with those of the parasitic form, in ordure contained in a small pit such as is common in Assamese villages. I then set myself to see if I could breed a similar sexually mature free form in the laboratory, and after a few experiments hit upon the following method: A layer about an inch in depth of clean white river sand is spread over the bottom of a crystallising dish or other suitable vessel. Nematode worms are extremely rare in river sand, but it is well, before employing an apparatus so prepared, to moisten the sand and examine it for several successive days to ensure that no such organisms are present.

Having selected a suitable case of ankylostomiasis—that is, a case in which repeated examination of the dejecta has demonstrated the absence of other parasites or their ova, such as the embryo of anguillula stercoralis or the ova of oxyuris, etc., a small quantity of the dejecta is mixed with about three or four times its bulk of water, and poured on to the surface of the sand. The dish is then covered with a sheet of window glass, and placed on a shelf in the laboratory, in ordinary diffused light. Every day a small portion of the thin upper fæcal layer is removed on a spatula, and mixed with water in a shallow glass tray and examined, either under the simple microscope or under a low power of the compound; but the former is preferable, and suffices for all but the study of the minute anatomy of the worms. Working in this way it will be found that the embryos hatch out in the course of a few days, the time varying with the air temperature from five days at a temperature of 60° F. to the next day at 84° F.; at 70° usually on third or fourth day.

As found in the fresh dejecta the ova are usually 2 4 segmented, but unsegmented ova may often be found. Day by day it will be found that the specimens are more advanced, the segmentation gets finer, till a morula and finally a fully-formed embryo is developed. It is useless to attempt to observe the further development of these embryos in clear water, for in this they cannot thrive, and slowly die for want of nourishment; but by taking samples from the cultivation at intervals and placing in water, the process can be can be followed with ease. On emerging from the egg the embryo measures 0.085 inch in length by 0.005 inch in diameter. At this stage they show little else than an intestine and œsophageal bulb. Within the next forty-eight hours (at 70° F.) they have increased to three times their original length and have undergone several ecdyses, the exact number of which I am unable to state, owing to the difficulty of keeping an individual properly nourished while under continuous observation, but roughly about twice in the twenty-four hours. In large fæcal masses under natural conditions, in the plains (temperature 80° to 90° F.), the embryos may develop into free sexually-mature worms in as little as four days, but under the somewhat less favourable conditions of my laboratory they occupied from six to ten days. As far as I could make out, the sexual organs were first clearly distinguishable in the last ecdysis but one, at which period the male measures about $\frac{1}{3}$ inch in length by $\frac{1}{100}$ inch. When completely grown up, he may reach $\frac{1}{2}$ inch in length by $\frac{1}{50}$ in diameter, while the females may attain $\frac{1}{1}$ inch in length by $\frac{1}{25}$ in thickness. It would occupy too much of your time to enter into the details of the anatomy and development of these free forms, for which I must refer you to my report on the subject, published by the Assam Secretariat Press, Shillong, 1890—but it may be well to meet here objections that have been advanced as to the validity of the above mentioned observations, and as far as I have been able to follow the literature of these subject, these objections are all based on a short paper by Dr. Sonsino [L. Anchylostoma E. La Sua Profilassi, 1889], the other criticisms I have met with being apparently mere mechanical transcripts of this paper, by commentators who obviously have not been at the pains to consult my report of the original. Dr. Sonsino's criticism throws doubt on the accuracy of my observations on the two following grounds:—

(*a*) That the organisms described by me are rhabdites terricola or other free nematodes; (*b*) that the appearances were really the free form of rhabdomena intestinale.

The first objection is obviously quite untenable in view of the method of examination adopted. Owing to the absence of suitable nourishment, clean river sand is, at best, an unlikely habitat for rhabdites terricola, but, as already noted, the sand used was always carefully examined, in a manner that entirely excluded the presence of such intruders, before being used for the cultivations. Moreover I have, on more than one occasion, made successful cultivations in which the sand as well as the water used for dilution of the infected fæcal matter was sterilised by boiling.

That experiments conducted in this way do not succeed so uniformly as those in which the sand has merely been previously examined by the biological test of moistening and examining on several successive days, and the water simply filtered. is probably owing to the destruction of bacteria suitable to bring about the decomposition of the fæcal matter surrounding the eggs, as the rhabdites of the ankylostoma can thrive only in decomposed fœces. As to the second objection, I can only say that the absence of anguillula stercoralis, that is, of the embryos of rhabdomena intestinale was carefully noted in the case of each specimen of dejecta used for experiment: Had the so-called anguillula stercoralis been present it is inconceivable that it should have been overlooked in any examination that detected the presence of the ova of ankylostoma.

Assam is a paradise for the helminthologist, and I was therefore greatly surprised at never once meeting with this particular human parasite in any of the many hundreds of examinations that I made of human dejecta. Further, at the same time as I was conducting these observations of ankylostoma rhabdites I did a good deal of general helminthological work on animals, detecting many parasites at least as insignificant as the rhabdomena intestinale, and had that parasite been present in the duodenum it could not have been overlooked in the specially minute examinations I made of the human intestine in all cases in which a necropsy was obtainable.

It is true that the adult parasite (rhabdomena intestinale) was not known at the time of my investigation, that the embryos (anguillula) had been met with in Africa and in China, and being anxious to compare them with my ankylostoma rhabdites, I was keenly on the look-out for them; but they did not exist in the parts of Assam I visited, nor have I heard that they have been met with by any other observer in any part of Continental India, though I gather that Macdonald has met with them in Ceylon.

In addition to this it is to be noted that it is stated that

anguillula stercoralis can only mature in foul water, and rapidly dies in decomposing fæces. The rhabdites of ankylostoma, on the other hand, mature rapidly and best in the decomposing fæcal matter which is their natural habitat. Up to the time of my leaving Assam I had never once succeeded in bringing them to maturity in water; but since then when repeating my experiments at Saharanpore in the N.W.P. I succeeded on one occasion in rearing them in water containing a considerable amount of fæcal matter. From these considerations it is evident that these suggested sources of fallacy are entirely excluded by the method of observation adopted, and I may add that the mere fact of their being suggested shows that my critics had not been at the pains to read my report with any approach to attention. As a matter of fact, all these three nematode forms under discussion do very closely resemble each other, though doubtless if one could place them side by side it would be easy to find specific differences, for example, the males of the ankylostomua rhabdites and of rhabdites terricola have a caudal membrane supported by seven or eight ribs, but Sonsino's figures of mature anguillula do not show any such structure. This, however, may be merely a matter of careless drawing, or, as I should judge from the figure, the selection of a not quite mature specimen, as this structure only appears in the last ecdyses. As a matter of fact, however, any other life-history for ankylostoma is becoming increasingly improbable.

Ralliet has demonstrated a similar dimorphism in strongylus strigosus and in s. retortiæformis, while I myself have done so in two other sclerostomes, and am strongly inclined to suspect that a large proportion of the so-called free nematodes will ultimately be demonstrated to be nothing more than dimorphic representatives of known parasites.

On no occasion have I found these rhabdites in drinking, or, indeed, in any natural collection of, water. On the other hand, the fouled soil round infected villages and among the crops, where the natives resort for purposes of Nature, teems with them, and hence I am inclined to believe that soil carried to food by unwashed hands is the commonest vehicle of infection. In this connection I am inclined to think that it matters not whether the embryo swallowed is the progeny of a parasite or free form, provided only it be swallowed when young. The progeny of both, in other words, develop into parasites or rhabdites, according to the environment in which they find themselves.

Prolonged exposure to the direct rays of the sun kills the free stage worms, probably because they are thus often brought to a temperature exceeding 140°. The prolonged desiccation of even a Punjab summer will not do so, as after a lapse of over twelve months some of the cultivations I had brought from Assam with me to the Punjab were easily revived by moistening; and on being fed with normal fæcal matter were through several generations easily brought to maturity. Burying the embryos also destroys them, and they are easily killed by most of the ordinary disinfectants, such as perchloride of mercury or carbolic acid in moderately strong solution.

I will not take up your time by discussing the clinical side of the question, but will merely express a hope that some of those present at this meeting from various parts of the world, will give us the benefit of their experience as to the harmfulness of the parasite. My own impression is that it is responsible for a formidable mortality and even greater amount of chronic sickness wherever it exists, but a small group of medical men in Assam are inclined to regard this view as, to say the least of it, exaggerated, while their leader, Major Dobson, of Dhulia, regards the worm as rather beneficial than otherwise, in fact a sign of robust health.

Major Dobson's duties for many years included the examination of large numbers of coolies coming from various parts of India. I had myself demonstrated to him the then new fact that a very considerable proportion harboured the ankylostoma. I also did my best to show him how to diagnose such cases by means of the microscope, but mistrusting an instrument with which he was unfamiliar, he adopted the somewhat heroic course of dosing all comers, healthy and sickly, with thymol, and then examining their dejecta for worms, and subsequently to my visit demonstrated what might have been easily predicted by anyone familiar with the general phenomena of helminthology, namely, that the worm was present not only in those who exhibited perceptible symptoms, but also in a large proportion of the really or apparently healthy.

My object in instituting these examinations of immigrants at Dhulia was to ascertain if the disease existed in the coolies brought from other parts of India, because if this were the case it would explain the fact that the appearance of kala-azar and beri-beri in Assam is coincident with introduction on large scale of the system of imported labour. The fact that the disease existed amongst these immigrants sufficed for my purposes. I therefore examined only such cases as presented some symptoms of ankylostomiasis, though necessarily none were at all advanced cases, as the labour recruiters knew too well that no obviously sickly coolie would pass Major Dobson's critical eye.

Wherever this disease is common, it necessarily follows that a large proportion of the healthy population will harbour the worm also. The presently healthy people may be either severely-infected early cases or slightly-infected cases of any duration; but overlooking the fact that the men he saw had already been selected because they were healthy, and that on this account no really advanced cases could possibly come before him, Major Dobson fell into the fallacy of proclaiming to the world that the presence of ankylostoma was a sign of health rather than disease.

Added to this, I am convinced that the wish to escape the disagreeable and almost insoluble problem of the introduction into Assam of measures of "conservancy" which obviously alone can be of any avail in preventing the disease has much to say in the genesis of the thought that these worms are after all harmless.

A second point on which our knowledge of the subject is incomplete is the way in which the parasite brings about the characteristic anæmia. It may, I think, be conceded that the mere loss of blood is insufficient in the majority of cases, only when enormous numbers are present can the drain be adequate.

Personally I am convinced that dyspepsia due to the damage brought to the mucous membrane (a point which I verified *post mortem*) has much to say in the matter, but a further hypothesis is that the parasite produces a poisonous excretion. So far as I am aware, no one has as yet advanced proof of this; but it has been established for certain other entozoa. Lastly, the experience of any gentlemen who may have met with the disease in non-malarious localities would be specially valuable and interesting.

II.—Captain C. F. FEARNSIDE, I.M.S.,

Superintendent of the Central Prison, Rajahmundri.

A SYSTEMATIC search for the ova of this parasite amongst the convicts and new arrivals in the Central Prison, Rajahmundri, was commenced in February last. Those examined came from the Northern Circars (Ganjam, Godaveri, Vizagapatam, Krishna), also Kurnool and the hilly tracts of the East Coast. The microscopic examination of the stools of new admissions was carried out within a few days after their arrival, and on an average three slides were carefully gone over.

At the end of a year it is intended to publish the full results, and the information furnished is still incomplete. From February 22nd to June 13th, 1900, 678 new arrivals were examined. In addition to these, more than 300 convicts, who have been confined over six months in the prison, have also been scrutinised. Having a limited time at my disposal, it was only possible, as I mentioned before, to examine three slides. So I feel certain that had more time been devoted to the work the percentages would be higher.

Of the 678 new arrivals, 462, or 68.1 per cent., harboured this parasite. Of 100 of these, taken haphazard between April 20th and May 3rd, the percentage affected is 72, a figure somewhat higher than that of the total 678 persons. The percentage of round worms is the same—36—and thus, for the purpose of comparison, the 100 cases may be taken as a standard. Of the 72 persons affected with ankylostoma, 50 (or nearly 70 per cent.) were in good health, 12 (or 16.6 per cent.) were in indifferent health, and 10 (or 13.9 per cent.) were in bad health. Thus 72 per cent. of persons in the Northern Circars harbour this parasite and remain in good health. Nearly 35 per cent. harbour in the bowel simultaneously both ankylostoma duodenale and ascaris lumbricoides.

It is interesting now to compare what are the effects on these entozoa of better hygiene and cleaner food in our prisons. I have attached the analysis of 200 convicts who have served over six months in prison. The percentage of ankylostoma has dropped from 72 per cent. to 58 per cent., and that of ascaris lumbricoides from 36 per cent to 18.5 per cent. When the inquiry is finished at the end of a year, I am of opinion that the results will be much the same as those just mentioned.

Of the 105 *post-mortem* examinations made by me in the prison, 74.3 per cent. revealed the presence of this worm; 57.9 per cent. showed congested areas, from one to several centimetres in diameter, in the bowel; and 11.4 per cent. disclosed small erosions and ulcers about 1 mm. to 2 mm. in diameter.

These figures, as well as the experience gained in the gaol after 3½ years, go to show that the effects of the ankylostoma are for the most part secondary and not primary. They seldom occur in such numbers as to cause true ankylostomiasis, are not the result of the ankylostoma primarily, but of such disorders as malaria, dysentery, etc. The presence of ankylostoma in malarial or other cachectic states is of great importance. It bleeds the patient who can ill afford to lose more blood; it sets up local congestions and erosions of the bowel which cause a catarrh and thus retard the proper assimilation of the food and recovery of the patient. It is this secondary effect of ankylostoma that I look upon as most injurious.

A word now about thymol. Many are enthusiastic about this drug, and think that it acts like a charm. Observers speak of washing the stools and finding hundreds of dead ankylostomata after its administration. I have seen dead ankylostoma in the bowel, and it is by no means easy to say that they are dead ankylostoma, so like are they to decomposed tissues of meat, vegetables or husk of grain. I do not trust this form of investigation of stools by washing them, etc. I prefer the simple plan of microscopic examination of the motions a few days after the administration of the drug. I attach notes of 13 cases who have been consuming large doses of thymol for varying periods from 10 to 60 days, who were frequently purged and who at the end had as many ova of ankylostoma in their stools as at the beginning.

For the diagnosis of ankylostomiasis, therefore, it is necessary to exclude all other blood-destroying diseases rather than depend on the mere presence of the parasite in the bowel.

TABLE I.—*Analysis of 678 Convicts whose Motions were Examined on Arrival from February 22nd to June 13th, 1900.*

—	Ankylostoma Duodenale.	Ascaris Lumbricoides.	Trichocephalus Dispar.
Number of cases in which ova was present	462	245	51
Percentage... ...	68.1	36.1	7.3

TABLE II.—*Analysis of 100 Convicts whose Motions were Examined on Arrival from April 20th to May 3rd, 1900.*

—	Ankylostoma Duodenale.	Ascaris Lumbricoides.	Trichocephalus Dispar.	Ankylostoma Duodenale and Ascaris Lumbricoides associated in the same Individual.
Number of cases in which ova was present	72	36	12	25

TABLE III.—*State of Health of 100 Convicts in whose Motions Ova of Ankylostoma were Found.*

—	Good Health.	Indifferent Health.	Bad Health.
No. of cases in which ova was present	50	12	10

TABLE IV.—*State of Health of Convicts Unaffected by Ankylostoma Duodenale.*

—	Good Health.	Indifferent Health.	Bad Health.	Number Unaffected by any Parasite.
Number	14	10	4	17

TABLE V.—*Analysis of 200 Convicts who have Served 6 Months and upwards.*

—	Ankylostoma Duodenale.	Ascaris Lumbricoides.	Trichocephalus Dispar.
No. of cases in which ova was present	116	37	10
Percentage	58.0	18.5	5

TABLE VI.—*Necropsies carried out in Rajahmundri Central Gaol.*

Cause of death.	Number of Deaths.	Ankylostoma Duodenale	Hæmorrhagic spots.	Erosions
Dysentery	29	25	18	8
Diarrhœa	12	11	7	8
Ague	14	2	9	2
Pneumonia	17	14	10	2
Tubercle of lungs	8	7	5	2
Valvular disease of heart...	5	4	3	1
Disease of liver	3	2	2	1
Disease of kidney	13	11	9	1
General diseases	4	2	2	1
Total	105	78	65	12
Percentage	105	74.3	51.9	11.4

TABLE VII.—*List of Convicts Affected with Ankylostoma, Treated by Thymol and Occasional Purgatives.*

Number of Convict.	Period Treated by Thymol.	Dose of Thymol Daily.	Remarks.
994	10 days	20 grains	Ova as numerous at the end of treatment as at the beginning.
970	17 ,,	20 ,,	
9,635	27 ,,	20 ,,	
767	24 ,,	20 ,,	
1,305	15 ,,	30 ,,	
9,708	30 ,,	20 ,,	Ova still numerous.
629	30 ,,	20 ,,	
774	30 ,,	20 ,,	
743	30 ,,	20 ,,	
9,708	30 ,,	30 ,,	Second period of treatment } Ova still numerous.
9,711	30 ,,	30 ,,	
774	30 ,,	30 ,,	Second period of treatment } Ova still numerous.
743	30 ,,	30 ,,	,, ,, ,,

III.—LEONARD ROGERS, M.D., M.R.C.P., I.M.S.,
Officiating Professor of Pathology, Medical College, Calcutta.

THE very great differences of opinion on the subject of ankylostomiasis which the recent literature of the subject reveals affords ample reason for this debate. For example, while Mr. Thornhill[1] has frequently written most eloquently on the terrible ravages caused by this parasite in Ceylon, that careful observer, the late Dr. Macdonald,[1] wrote of the same colony that in a great number of cases the worm does very little harm, although capable under certain conditions of destroying life. Unfortunately Dr. Thornhill gives very few figures in his extensive writings, and on turning to the Ceylon medical reports for from 1886 to 1893 I was astonished to find very little reference to the disease in them, while, as I have elsewhere shown,[2] the figures given indicate a great decrease of the disease during recent years in that colony, while in Thornhill's own district, among a population of 160,000 persons, the recorded

death-rate from this disease in the hospitals was but 32 in 1894, so that the evidence of these reports strongly supports the moderate views of Macdonald rather than the extreme ones of Thornhill. The same thing is seen in Assam, where ankylostomiasis was first discovered by Dr. Ruddock, who is of the opinion that a healthy person can support a fairly numerous colony of them (ankylostoma) without damage, and the parasite only assumes importance when the patient is lowered by some other disease,[3] while Dobson has recorded instances of perfectly healthy men harbouring from one to two hundred of the worms, and finding small numbers in over 60 per cent. of healthy Assamese, and in upwards of 80 per cent. of healthy natives from Bengal and other parts of India, asks, "At what stage does the parasite become injurious?" On the other hand, Major Giles wrote with regard to the terrible disease kala-azar, which has certainly carried off at least one-fifth of the population of a tract of country 200 miles in length, "All I wish to convey is that the increased mortality is due to ankylostomiasis and to no other cause."[7] And again, "What, then, is kala-azar? Kala-azar is ankylostomiasis," with no qualification whatever. In another passage he wrote that the disease was ankylostomiasis, acting in a population which "has for generations been continuously poisoned by malaria," but fortunately it is unnecessary to discuss this question now that so high an authority on malaria as Major Ross[4], after a short personal investigation of kala-azar in Assam, has recorded his opinion that "I think, then, with Rogers, that kala-azar is malarial fever," and further "that in the latter epidemics studied by Rogers this fever is also communicable from the sick to the healthy," and again, "I agree with him (Rogers) that kala-azar is not ankylostomiasis," although he adds that in several instances these words amounted to a serious complication, as I had previously stated was sometimes the case. Moreover, since Ross's confirmation of my conclusions on the subject, Major Giles has admitted[8] "as I am thus reduced to the conclusion that the disease now called kala-azar is something quite different from what was shown me," which should be read in connection with the following statement in my original report: "Dr. Giles's opinion that fever was not a common or a marked feature of the disease, is accounted for by the fact that he was unfortunate enough to happen to commence his clinical work in Gauhati late in November, which is just the very time when the fever is at a minimum, and remains so until the commencement of the next rains, by which time Dr. Giles was engaged on microscopical work in Shillong" (a hill station which kala azar never reached) "so that he only studied the disease at the season when most of the cases which have survived the previous rains, have lost their fever, and fresh infections are at a minimum." As far as I know, Mr. Thornhill remains the only advocate of the view that ankylostomiasis is the essential factor in the cause of kala-azar; and as he admits he has never seen a case of the disease, and his extreme views on the ravages of the disease in Ceylon are not accepted by his colleagues there, his opinion cannot be allowed any weight against the accumulated positive evidence, and now practically unanimous opinion, of all who have seen the disease in Assam, that it is purely malarial, while the successful eradication of the disease from tea gardens by the methods advocated in my report, which have been recorded elsewhere, may also be fairly claimed as evidence as to the soundness of my conclusions.[11 12]

The Sources of Fallacy.

How can the above differences of opinion be explained? This question has been answered by the researches of Dobson,[1] for Giles furnished no evidence in his report that he ever controlled his observations by examining healthy persons for the ankylostoma, and appears to have been quite ignorant of its nearly universal prevalence of the worm in them (although previous to his investigation Dr. McConnel of Calcutta had found the worms in the great majority of necropsies in Calcutta as far back as 1882), and he gives no evidence to show that these parasites were met with in larger numbers in kala-azar cases than in healthy persons in Assam (which I subsequently showed they were not); yet soon after Giles's report was published, Major Dobson, as a result of examining over 1,000 persons for the worms by the thymol method, showed that they were present in some 67 per cent. of healthy Assamese, and 84 per cent. of several hundred healthy coolies who had been passed by medical officers as fit for emigration from Bengal, Madras, Chotta Nagpur, the Central Provinces, and the North-West Provinces; in short, a large part of India. I also found them in 66 per cent. of healthy Assamese, in slightly larger numbers than in kala-azar cases, and showed that in the latter disease less than 20 of the worms were present in upwards of 80 per cent. of the patients, while Thornhill[1] admits that "50 of these worms is a number altogether too small to have any deleterious effect," and the same writer agrees with others in considering that in order to produce anæmia at least 500 of the worms must be present for from six months to a year; with which I also agree. Another writer[5] has recorded "Short notes of 130 cases of ankylostomiasis," in which it appears probable that only 8 cases passed more than 50 worms after thymol, while certainly three-quarters of them had less than that number present. The cases were admitted to hospital for all causes, from measles to locomotor ataxy, while 31 of them were admitted for malaria, and 19 more had malarial symptoms. Yet because they passed a few worms after thymol they are called cases of ankylostomiasis! At this rate upwards of 80 per cent. of the healthy inhabitants of large provinces of India suffer from ankylostomiasis! I have no hesitation in saying that nearly all the confusion with regard to this disease has arisen owing to the loose way in which the term ankylostomiasis has been frequently used.

Definition and Classification of Ankylostomiasis.

In order to avoid this source of confusion, I shall in this paper use the term "ankylostomiasis" to indicate a disease characterised by anæmia produced by long-continued small losses of blood through the gastro-intestinal mucous membrane caused by the presence of several hundreds of ankylostoma acting for many months; or by a still larger number acting for a shorter time. The very fact that in the St. Gothard tunnel outbreak as many as 2,000 to 3,000 of these worms were found in some cases which had suffered from anæmia for several months is alone enough to show that very small numbers of the worms cannot produce any appreciable effect on the system. That the abuse of the term ankylostomiasis is a matter of practical importance is shown by an instance, which I have elsewhere recorded,[6] in which out of 72 consecutive cases which were treated by thymol for anæmia in an Assam dispensary, 17 died, no fewer than 8 of which succumbed within six days of the last dose of thymol, although the average number of worms passed by them was but 5 and the maximum 10 in these 8 cases, while not one out of the 72 cases passed over 50. The fatal cases, as well as the great majority of the others, were suffering from malarial cachexia, while many had diarrhœa or dysentery. Yet because they were anæmic, all were given drastic doses of thymol, and the end of some at least who might otherwise have recovered was certainly hastened by the treatment.

Most writers on this subject admit that in order to produce anæmia, at least 500 ankylostomata must be present for from six months to a year, although several thousands would doubtless produce symptoms in a shorter time, but the latter are rare. It is to these classes of cases that I would limit the term ankylostomiasis, and using it as defined above I may say that the disease is rare in Assam villages apart from tea gardens or railway coolies, in the Nowgong district at any rate. I have met with several cases in which I at first sight took the anæmia to be of this class, but which close investigation showed other causes, such as Bright's disease, bad feeding, malarial fever, syphilis, etc., to have been responsible for the condition.

On the other hand, there is another class of cases in which anæmia due to some other cause is complicated by the presence of from one to three hundred ankylostomata, the drain caused by which alone might be withstood by a healthy person under favourable circumstances for a long time, (instances of these I have recorded in a previous paper),[6] but which when added to other debilitating and anæmia-producing causes becomes an important pathological factor. These constitute the greater number of cases in which the parasite is seriously injurious in my experience, but they should be classed under the heading of the primary disease, malaria, dysentery, syphilis, etc., with the qualification that they are

complicated by a harmful number of ankylostomata. Lastly, healthy men or those suffering from common diseases should not be classed as ankylostomiasis just because they happen to harbour a few of these parasites, the very small loss of blood caused by them, amounting to but a few drops daily, is repaired by the system without any ill-effect whatever, for I have shown elsewhere[6] that there is no reduction in the hæmoglobin or red corpuscles in such cases.

The Differential Diagnosis of Ankylostomiasis.

In advocating above the careful restriction of the term ankylostomiasis to its proper meaning of disease produced by this parasite, it is very far from my wish to minimise the importance of the *rôle* played by the worm in various parts of the world, of which I am fully convinced, but rather desire to lessen the confusion which hampers efficient action to lessen the scourge, for such a disastrous result as that referred to above can only bring discredit on the thymol treatment, and so lead to its being omitted in suitable cases. What is wanted is more careful diagnosis, a matter of no small difficulty as experience has taught me for although the number of ova in the fæces is a sure guide to the presence of large numbers of these parasites, the absence of them does not prove that the anæmia has not been caused by previously present worms, for they may have dropped off, but in that case thymol will be unnecessary. Moreover, I have found that ova can be easily found in a single cover-glass preparation of the fæces when but five to fifteen worms are present; but, on the other hand, if they are met with in nearly every field of the microscope, then it is certain that the parasites are present in large numbers, and thymol is indicated if the patient is in a condition to stand it. But the mere discovery of one or two ova is not an indication for thymol unless it is quite certain that the drug will do no harm, or much greater injury may be inflicted than will be compensated for by the removal of the very few worms which may be present. This remark applies especially to cases of chronic malaria, in which I have recently found a condition of pigmented atrophy of the mucous membrane of the small intestine, which I am convinced is a very important factor in fatal cases with intractable diarrhœa, and which very probably accounts for the low fever seen in these cases, which my be caused by the absorption through the thinned intestinal mucous membrane of more poisonous material than the liver can adequately deal with. It is in these cases that thymol is so dangerous.

The clinical features of ankylostomiasis are too well known by workers in tropical diseases to need description, but they may be very closely simulated by Bright's disease, the œdema in which is so similar, while albuminuaria not infrequently appears in the most advanced stages of ankylostomiasis. Very similar, too, is the anæmia produced by bad feeding, more especially a deficiency of nitrogenous foods, an example of which I have elsewhere recorded[3]. Cases of both these diseases I have myself mistaken for ankylostomiasis until I made the blood examination to be mentioned immediately. In the anæmia of dysentery the patient is usually very wasted as opposed to the fat appearance in the worm disease. Most important of all is the differentiation of malarial anæmia from that due to ankylostomiasis, and although in typical cases the clinical pictures are in marked contrast (a table of the differences between which I have published in a previous paper[3]), yet in certain cases, more especially when the two diseases complicate each other in various degrees, it is well nigh impossible to solve the problem of their relative importance by clinical means alone. Yet it is just in these very cases that it is most important to do so, for not only is thymol particularly dangerous in advanced malarial cachexia but the treatment of the anæmia of the two conditions is quite distinct. Fortunately a way of differentiating them has been discovered in the essentially different type of anæmia in the two conditions, which I found to be of the greatest value in the solving of the difficult problems surrounding the mysterious kala-azar, which largely resolved itself into deciding whether malaria, or ankylostomiasis, or both together were the cause of the anæmia, which all workers acknowledged to be present in the disease.

The Types of Anæmia in Ankylostomiasis and Malaria.

In such a country as Assam, in which the majority of healthy persons harbour a certain number of ankylostomata, while in the latter stages of ankylostomiasis few or none of these parasites may be present, it is exceedingly difficult, if not altogether impossible, to decide from the number of worms found in a given case what part they have played in the production of the anæmia, while I have just shown that clinical evidence may also be at fault in these cases. The problem, then, was a very difficult one, but it appeared to me that the type of the anæmia produced by chronic malaria, in which the red and the white corpuscles of the blood are destroyed, but the hæmoglobin is largely retained in the body in the form of pigment in the liver, spleen, etc., might be expected to differ from that produced by the slow hæmorrhage, which is the essential feature of ankylostomiasis. The examination of such literature as I could obtain in the Calcutta Medical College Library, however, revealed such a hopeless difference of opinion as to the type of the anæmia of more especially ankylostomiasis, that I determined to make as full an examination of the blood as possible in both diseases. The results exceeded my most sanguine expectations, for not only did the type of the anæmia in the two diseases prove to be essentially different but the differences were found to be so constant as to furnish a certain means of diagnosis between the two diseases, and even served to indicate the presence of an injurious number of ankylostomata in a case of typical malarial fever when the anæmia was so slight as to be only detected by a quantitative examination of the blood.

The details of this research have already been published,[14], but their practical importance is so great that I gladly avail myself of this opportunity of bringing them before a larger audience, so will briefly describe them. In the first place it must be mentioned that I found the blood of healthy inhabitants of Assam to be very different from the European standard. Thus, although the numbers of the red and white corpuscles were about the normal, the percentage of hæmoglobin averaged only 62 per cent. of Gowers's standard, in spite of all cases in which ankylostoma might have been a disturbing factor having been carefully excluded. The colour index in these healthy cases (men) worked out at 0.65, which must be borne in mind in studying the figures in the different forms of anæmia, which are given in the accompanying table:

—	Percentage of Hæmoglobin.	Red Corpuscles per Cubic Millimetre.	White Corpuscles per Cubic Millimetre.	Ratio of White to Red.	Specific Gravity.	Hæmoglobin Value or Colour Index.
Healthy Assamese	62.00	4,734,000	7,325	1 : 684	1054	0.65
Epidemic malaria of Assam (Kala azar)	33.45	2,462,000	2,600	1 : 1170	1048	0.65
Ordinary chronic malaria ...	31.60	2,000,000	1,600	1 : 1400	1042	0.73
Ankylostomiasis	15.20	1,145,000	5,338	1 : 524	1034	0.31
Malaria + ankylostomiasis ...	27.40	3,120,000	3,200	1 : 975	1039	0.43

Secondly, the average blood estimation in ordinary cases of malarial cachexia in a part of Assam which was unaffected by the epidemic, malarial fever, or kala-azar, which are given in the third line of the table, show a nearly equal reduction of the red corpuscles and the hæmoglobin, so that the colour index is 0 73, or slightly higher than in healthy Assamese. Further, the white corpuscles are greatly reduced, both absolutely and relatively, to the red, so that there is only 1 white to 1,400 red, or about half the number in healthy people. The specific gravity is also reduced but only to a slight degree in proportion to the anæmia. Precisely similar conditions were met with in a large number of cases of the epidemic malarial fever of Assam, as is shown in the second line of the table, which fact alone, contrasting as it does with the condition met with in ankylostomiasis, is sufficient to indicate that kala-azar is essentially malarial. In these cases the colour index is precisely the same as in the healthy people of the province, showing that the hæmoglobin and the red corpuscles are equally reduced, and the colour index is 1, allowing for the low amount of hæmoglobin met with in these people. Again,

the white corpuscles are reduced much more than the red, so that there the proportion is 1 to 1,170. The specific gravity is but slightly reduced in proportion to the anæmia. The figures given by Cabot and by Waddell[15] in India in cases of chronic malarial anæmia agree very closely with those recorded above, so that it is evident that the anæmia of the Assam epidemic is identical with that of malaria in other places.

We may now turn to the conditions met with in ankylostomiasis, with regard to which there is so much difference of opinion and positive error in even the most recent works on tropical diseases. A glance at the fourth line of the table shows at once the contrast between the type of the anæmia in the worm disease and that of malarial cachexia. The most important distinction is that in ankylostomiasis the hæmoglobin is reduced twice as much as the number of the red corpuscles, so that the colour index sinks to 0.31, or about one-half of the normal in healthy people of Assam. Again, the white corpuscles, although absolutely reduced in numbers, are relatively less reduced than are the red, so that the proportion rises to 1 to 524 against 1 to 684 in the healthy controls; a change which I subsequently found had also been noted by Lutz. Once more the specific gravity of the blood is greatly reduced having averaged only 1034, the reduction being greater in proportion to the degree of the anæmia, as estimated by the percentage of hæmoglobin, than it was in malarial cases of an equal degree. Thus in every point the type of the anæmia of the two diseases is radically different. But this is not all, for it is not sufficient from the diagnostic point of view to show that the average changes differ unless it can also be proved that the extremes met with in either condition do not overlap to any extent those met with in the other. As a matter of fact I have found that they do not overlap at all in the case of the essential point, namely, the colour index, for the lowest point met with in uncomplicated cases of malarial anæmia was always over 0.5, while the highest encountered in ankylostomiasis was always under 0.4 in Assam, so that this point serves as a certain means of differential diagnosis between the two forms of anæmia. In view of the statement in recent textbooks on tropical diseases that the hæmoglobin value of the corpuscles is not depressed correspondingly to the fall in numbers—a statement which appears to have been copied from one book to another—I may be asked if there is any confirmation to form the results recorded above. There is, for Dr. Sandwith of Cairo, in a valuable paper on ankylostomiasis in Egypt, gives estimations of the hæmoglobin and the number of the red corpuscles in 173 cases of the disease, which show the percentage of hæmoglobin to be reduced nearly twice as much as that of the red corpuscles, so that the colour index was reduced to about one-half, just as it was in my own cases, allowing for the abnormally low hæmoglobin value of healthy Assamese. Further, I have received communications from several workers in India to the effect that they had been able to confirm my observations on these points.

Lastly, in a few instances of malarial cachexia (under 7 per cent. of kala-azar cases) there were sufficient of the worms present to constitute a definite factor in the production of the anæmia, and in all of them the blood changes were found to be intermediate between those of the two primary conditions, as is shown in the fifth line of the table. One of these cases is of particular interest. It was an early case of the epidemic malarial fever in which no clinically evident anæmia had yet appeared, but in which the spleen and liver were enlarged. On examining the blood the hæmoglobin value or colour index was lower than usual in pure malarial anæmia, so that I at once told the medical man in charge of the case that I suspected the patient to harbour an active number of ankylostomata, and proposed to give him thymol. This was done and 159 of the worms were passed. In this case, then, the blood test was found to be delicate enough to detect the presence of a not very large number of the parasites before even clinically evident anæmia was present. Further, I have found the characteristic alterations to persist for many months after the removal of the worms, especially in the absence of proper treatment by complete rest and iron, and in one case in which a patient had been successfully treated for ankylostomiasis, but was subsequently attacked by kala-azar, a colour index intermediate between those typical of either disease was found although no worms were present, showing that the effect of the previous attack of ankylostomiasis on the blood had not completely passed away. Now the duration average of kala-azar cases is but seven months (against several years in ankylostomiasis as a rule), so that it is evident that in any case of the epidemic fever in which ankylostomata have played a definite part in the production of the anæmia, their action will be at once revealed by the approximation of the type of the anæmia to that of ankylostomiasis, as evidenced by a low colour index, even if all the worms have already dropped off. Yet in less than 7 per cent. of the cases was this found to be the case, which alone is complete proof that ankylostomiasis is not an essential or even a common factor in the production of the anæmia, which is found by all observers to be a constant feature of kala-azar, and hence the worms cannot be a primary cause of the disease itself. Other examples of the value of the blood examination in differentiating forms of anæmia are given in my report.[3] One was a man who had a large liver and spleen, but who said he had not suffered from fever recently, while in some respects he resembled a case of ankylostomiasis, so that it was impossible by clinical means to decide by which disease his anæmia had been caused, the blood estimations showed the ankylostomiasis type, which was therefore diagnosed and subsequently was found to be correct and successfully treated.

It only remains to briefly explain the differences of type in the anæmia of the two diseases, and to point out the therapeutical indications to be derived from them. First, with regard to the colour index, it will be at once evident that the differences noted are just such as might be expected, for in the case of ankylostomiasis the hæmoglobin is being continually slowly drained out of the system, and while it can only be replaced with great difficulty, especially in the case of people who are living on purely vegetable diet, on the other hand the red corpuscles are being rapidly formed in the bone marrow, for I have found the yellow marrow of the shafts of the long bones to be converted into red marrow both in ankylostomiasis and in malarial anæmia, just as it is in pernicious anæmia. The red corpuscles being more easily and rapidly replaced than the hæmoglobin, the colour index or amount of this substance in each corpuscle must necessarily fall, just as it does in all forms of slow hæmorrhage. In malaria, on the other hand, the hæmoglobin is not lost to the system, but is stored up in an organic combination in the liver and spleen, etc., from whence it can be absorbed to restock the red corpuscles as they are turned out. This process probably accounts for the occasional disappearance of the black pigment from these organs in the later stages of the more chronic cases of kala-azar, which has been pointed out by Ross,[4] especially those who are tending toward recovery. The decrease of the white corpuscles in the malarial anæmia is doubtless due to to the constant fight that they are carrying on with the parasites in the blood, as indicated by the slight leucocytosis which may occur, while in ankylostomiasis, on the other hand, they are not especially destroyed, while they are more easily replaced than are the red corpuscles, and hence a relative increase of the white cells occurs. It should be mentioned that in malaria the white corpuscles should be counted during the absence of fever, or else they may appear to be normal when greatly deficient, owing to the occurrence of a relative temporary leucocytosis during fever. The lower specific gravity of the blood, relatively to the degree of anæmia in ankylostomiasis, than in malarial anæmia is explicable on the ground that in the former disease there is a steady loss of albuminous constituents of the blood, so that the blood becomes more watery. In this connection it is worthy of notice that I found hæmic murmurs to be much more common in the anæmia of ankylostomiasis than in that due to malaria of similar degrees—a fact which supports the theory which attributes these murmurs to alterations in the specific gravity of the blood. All the changes met with, then, are just those which might have been expected to result in either case.

Finally, the correct treatment of these two diametrically opposite forms of anæmia should obviously be different, for in that due to ankylostomiasis iron is urgently indicated in order to assist in the production of hæmoglobin, while arsenic will be of less importance, as it is not much use stimulating the output of red corpuscles when there is no hæmoglobin with which to stock them. In the anæmia of malaria pre-

cisely the opposite holds, for there is no urgent necessity to pour iron into the stomach, very little of which will be absorbed (Burge), when there is already a large stock in an organic combination ready to hand in the liver and spleen, while on the other hand arsenic and bone-marrow tabloids (the latter of which more especially I have found to be of extreme value in malarial cachexia) are strongly indicated in order to increase the output of the red corpuscles which are being or have been destroyed by the fever. These indications I have followed with great success in many cases, and although they are often carried out as a result of empirical knowledge, a rational basis is afforded them by the research which I have described.

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IV.—OSWALD BAKER, M.D., Surgeon-Lieutenant-Colonel I.M.S. (Ret.).

Physician to Out-patients, Seamen's Hospital, Royal Albert Docks, London.

THE contribution to the discussion of this subject which I wish to make relates entirely to a consideration of the prevalence of ankylostomiasis in Burmah. My attention was first drawn to this question by the perusal of Dr., now Sir William, Kynsey's report on anæmia in Ceylon, issued by the Ceylon Government in 1887, of which I was fortunate enough to obtain a copy shortly after its publication. It appeared to me on reading this report that the anæmia so common in Burmah might also, as in Ceylon, be the result of ankylostomiasis. Although the ankylostoma duodenale was not at that time known to exist in Burmah, it seemed highly probable that on investigation this parasite would be found to be very prevalent throughout the province, for both the habits of the people and the climatic features of the country were in the highest degree favourable to its existence and dissemination.

In returning to Burmah, therefore, I lost no time in making an inquiry into the matter; nor had I long to wait, for within a few days of resuming charge of my duties a Hindu lad was admitted into the civil hospital suffering from anæmia in a profound degree, for the production of which there was no obvious explanation. The condition of this patient was practically identical with that of a person who had bled to the verge of death, and the symptoms he presented were precisely those and those only which result from great loss of blood from whatever cause arising. Ankylostoma ova in abundance were at once found in his stools, and on the day following this discovery two of the worms, both dead, were seen in his evacuations.

While the existence of the ankylostoma in Burmah was thus proved, the fact that this blood-sucking parasite was extremely prevalent throughout the provinces was soon afterwards established beyond doubt. In the Moulmein Gaol, of which I held medical charge, so prevalent was the ankylostoma among the prisoners that I seldom failed to detect its ova in cases not only of simple anæmia, but also of anæmia associated with specific diseases. As, however, an investigation limited to convicts already inhabiting the gaol, was open to the objection that the prisoners in question might have acquired the parasite subsequent to their admission, it was resolved to examine the stools of a large number of prisoners at the time of admission. Now, the gaol at Moulmein is what is known as a central gaol, and as such receives within its walls, criminals sent not only from the town but also many who are brought in from different parts of the province. Obviously, if large numbers of newly admitted prisoners were found infected with ankylostomata, my belief that the parasite was extensively disseminated would receive substantial confirmation. I determined, therefore, with the aid of a trained assistant, to examine microscopically the stools of 100 prisoners admitted consecutively into the gaol, as soon as possible after their reception.

Before the observations were fully completed, however, I was transferred from Moulmein to an appointment elsewhere, but the transfer did not take effect until 89 of the proposed examinations had been made. The results obtained may I think be accepted as representing the degree of prevalence of helminthiasis among the lower classes of the adult population of British Burmah.

Of the 89 prisoners, 17 were natives of India, and 3 were Chinamen. Taking the natives of Burmah alone, of whom there were 69, it was found that no fewer than 38, or 55 per cent., were infected with ankylostomata; that 33, or 49 per cent., had whip worms; and 30, or 43 per cent, had round worms; while the stools of 2 contained the ova of the tapeworm. Only 10 out of the 69 were without some species of entozoon. So that whilst 55 per cent. harboured ankylostomata, as many as 86 per cent. were the subjects of helminthiasis of some form or another. It may, I think, therefore, from these observations be safely concluded that the ankylostoma duodenale is present in the intestines of at least half of the poorer classes of the inhabitants of Burmah.

That a high prevalence of ankylostomiasis is both directly and indirectly responsible for much mortality there can be no question, but the extent of this mortality unfortunately cannot be ascertained. Numerous deaths take place annually, especially among the lower orders, from "unknown causes;" and in hospitals and gaols the mortality from anæmia and general debility of uncertain origin is often high. Much of this loss of life, set down to unknown causes, to anæmia and to general debility, is, I think, unquestionably due to ankylostomiasis.

I drew attention to this mortality in a short paper on Ankylostomiasis in Burmah, which was published in the *Indian Medical Gazette* some twelve years ago. I further pointed out that an infection with ankylostomata, unless adequate to the production of pronounced anæmia, would probably altogether escape recognition. I also explained that the reason ankylostomata were not always found *post mortem* in fatal cases of ankylostomiasis was due to the circumstance that these nematodes, subsisting on human blood, often abandoned their impoverished and anæmic victims before death, owing to want of sufficient or suitable nourishment. Subsequent observers, amongst whom I would mention Giles, have expressed very similar views.

That ankylostomiasis should obtain so high a degree of prevalence in Burmah can, when the habits and customs of its population are considered, hardly cause surprise. There is practically no night soil sanitation in the out-lying villages of Burmah, and most of the inhabitants defæcate on to the ground through holes in the floors of their dwellings. Left on the surface of the soil, exposed to wind and rain, a portion of this excrement becomes spread broadcast over the land, whilst some of it, in the form of mud, is carried into the houses of the people, and is deposited on their floors.

Now the majority of the Burmese do not use chairs and tables at their meals, but squat on the floors and eat their food with their fingers out of plates and bowls set down on the floor in front of them. When travelling they often dispense altogether with feeding utensils, and sometimes substitute the leaves of certain trees.

Under such circumstances, the general prevalence of intestinal entozoa is, I would affirm, inevitable.

Kynsey has maintained that water is undoubtedly the chief source of infection in ankylostomiasis, and has stated that the prevention of this disease consists essentially in the use of pure water. I do not think that such is the case. On the contrary, I believe the infection is conveyed through the medium of contaminated food, and that water has but little to do with the matter. In support of this opinion I would point out that among the 89 prisoners already referred to there were, as stated, 17 natives of India and 3 Chinese. Now these men, both Indians and Chinese, are far cleaner feeders than the Burmese, although the source of their water supply is the same.

Of these 20 aliens only two were found infected with ankylostomata being 10 per cent., as against 55 per cent. of the Burmese. Moreover, Giles has shown that water retards and finally arrests the development of ankylostoma embryos. There is, Giles states, "no condition more hostile to them than when immersed in water."

I would take this opportunity of pointing out that I believe the spread of many other entozoa is effected in precisely the same manner, that is through the medium of food and not of water. In the town of Moulmein, which in regard to sanitation is probably the most neglected town in the whole of Burmah, infection with ascarides is almost universal. About every third person applying for treatment in the outdoor department of the Civil Hospital is an applicant for worm medicine. The water supply is mainly derived from uncovered shallow wells, of which many are without parapets, and in seasons of drought often run dry. I have taken water from one of the dirtiest of these wells when nearly dry, have filtered it and subjected the residue to microscopical examination without finding therein a single ovum or embryo of any entozoon.

It would seem, therefore, from a consideration of these data, that no marked diminution in the prevalence of and mortality from ankylostomiasis in Burmah can be looked for until some system of sanitation has been generally adopted which will obviate the broadcast distribution of infective night soil. And to this question of night soil conservancy the attention of the authorities concerned should, I think, be given.

V.—Patrick Manson, C.M.G., F.R.S., LL.D., M.D.,
Physician to the Seamen's Hospital Society, Greenwich.

Dr. Patrick Manson said: It is somewhat singular that rhabdomena intestinale was so seldom found by Giles in Assam, considering that it is so frequently associated elsewhere with the ankylostoma, and that the climate and other physical conditions closely resemble those of Cochin China, where the parasite is extremely common, and where, indeed, it was first discovered by Normand. I have frequently met with it in the stools of Indians. I have twice encountered the ankylostoma in dangerous profusion in Englishmen from abroad. One case came from the West Indies. He had been in hospital for many months, and had been discharged as hopelessly all with pernicious anæmia. Fortunately after his discharge as an incurable he consulted a practitioner in the country, Uho found ova recognised to be those of the ankylostoma. sender treatment with thymol the patient got quite well. A dicond case came from Singapore. Ankylostomiasis had been diagnosed, but specific treatment was postponed until it could be undertaken under more favourable climatic conditions. When I saw the patient he was profoundly anæmic, as in advanced Bright's disease, with vertigo, tinnitus, palpitation, and all the symptoms of advanced anæmia. Every slide of his fæces showed numerous ankylostoma ova. Under thymol he rapidly recovered, and left for the East again quite well. I am astonished at Dr. Fearnside's statements as to the therapentic impotence of thymol. In my experience it rarely fails, provided it be given in adequate and rapidly repeated doses, say 30 grs. every hour, for four times. The diagnosis of malaria from ankylostomiasis by hæmocytometric methods, though interesting and valuable from a pathological point of view, is impracticable under ordinary circumstances. Dr. Rogers's statements as to the presence of a leucocytosis during the acute stage of malarial infection are not in accordance with recent observation. Perhaps I misunderstood his meaning; but the presence of a leucocytosis is generally held nowadays to exclude a diagnosis of malaria. I am disappointed that none of the papers have alluded to Powell's interesting observation that betel nut chewing is possibly a protective habit in the natives of Assam, Burmah, and the Eastern Peninsula, acquired instinctively in consequence of its prophylactic virtues against the ankylostoma. I agree with Colonel Baker's remark that the parasite is generally acquired in food and dirt rather than in water, and that improved methods of night soil conservancy are much to be desired on this account in the East. I believe the Chinese method of dealing with night soil to be a good one on sanitary grounds. The soil is stored in cemented tanks, where it rots and ferments for months before it is used as a fertiliser. During this period of storage it is probable that the ova and embryos of the intestinal entozoa it may contain are killed.

VI.—Major Ronald Ross, I.M.S.(Ret.).
Liverpool School of Tropical Medicine.

Major Ronald Ross remarked regarding the life-history of the parasite, that he had carefully studied Major Giles's experiments on a rhabditiform stage, had partly followed them and considered that they were sound. He thought that Sonsino's criticism of these experiments was unsound. Sonsino said that Giles had mistaken rhabdomena for ankylostoma; Major Ross supported Giles in stating that the former were uncommon in Assam—at least in Nowgong, and thought that such a mistake was too obvious a one to be easily made by a competent observer. He held that the well-known fact of the disease being so commonly an earthworker's disease supported Giles's discovery. He had long maintained the opinion regarding its mode of infection just given by Colonel Baker, and thought that there was little to be said in favour of infection by the route of drinking water. Regarding the clinical effects of the parasite, he scouted the idea that it was always harmless. It was impossible to fix the exact number of parasites required to produce pathological reaction. Rogers's estimate of 500 was probably much above the mark in many cases. Obviously, numbers required to produce reaction must vary inversely as the strength of the host. Fifty ankylostoma might destroy a patient already debilitated by other disease or starvation. The speaker considered that the parasites might also produce a toxic effect, and referred to Daniel's discovery of yellow pigment in the organs of cases of ankylostomiasis—examples of which he had seen. He concurred with the observers in thinking that the worms might leave the patient in the last stages of the disease. He entered a plea for the much more general use of the microscope for the detection of the ova in the localities where the worms were prevalent, and cited the instance of a hospital assistant in charge of a dispensary full of cases of ankylostomiasis who had not even heard of the disease, and who was treating the cases for malarial cachexia.

VII.—James Cantlie, M.B., F.R.C.S.,
Surgeon, Seamen's Hospital, Royal Albert Docks, London.

Mr. James Cantlie stated that, stimulated by the researches of Dr. Walker in Borneo in connection with the presence of the ankylostoma in beri-beri, he had carried out a careful investigation on the subject in Hong Kong in 1893, but failed to find either the ova or the fully-grown parasite amongst Chinese coolies suffering from beri-beri.

TROPICAL LIVER ABSCESS.

THE MANAGEMENT OF LUNG LESIONS CONSEQUENT ON LIVER ABSCESS.

By Colonel Kenneth Macleod, L.L.D., M.D.,
Professor of Clinical and Military Medicine, Army Medical School, Netley.

Of all the routes by which spontaneous evacuation of abscesses of the liver takes place the pulmonary route is the most common. Randus's statistics show that among 159 cases in which liver abscesses discharged spontaneously, 59, or 10 5 per cent., opened into and through the right lung, and 31, or 5.5 per cent. into the right pleural cavity; or, taken together, in 16 per cent. of these cases the pointing of the abscess took place upwards, through the diaphragm and into the thoracic cavity. The selection of this route in preference to others depends mainly upon the situation of the abscess, the principle of progress in the direction of least resistance coming into play. Whether the abscess bursts into the pleural cavity or irrupts into or erodes the lung, so as to establish a secondary abscess in that organ, must depend upon the extent and strength of protective pleuritis to which the progress of the pointing has given rise.

When the discharge into the pleural cavity is primary, the event is indicated by characteristic physical signs, and can be very readily and safely certified by exploration. In such cases there is but one indication and rule of treatment, which is to make a free opening into the pleural cavity, removing, if necessary, a bit of rib, and to establish efficient drainage. The opening must be made and the drainage conducted under strict antiseptic precautions. It happens occasionally that the discharge of a hepatic abscess into the right pleural cavity is a secondary event caused by the bursting into it of a lung abscess of hepatic origin, or by a wound of the pleural fold in exploring or cutting into a liver abscess. In these cases the cavity and its contained material are apt to be septic. The

Clinical lectures should still be delivered, and might with advantage, I think, be increased in number.—I am, etc.,

Cornwall Gardens, S.W., Oct. 5th. C. Frank Wightman.

Sir,—Your recent article on the above subject deals with a matter of great interest and practical importance. The respect paid to the lecture fetish seems visibly declining. Once thoroughly laid it is to be hoped that not even Plato's year will witness its resurrection.

It is of course evident that two factors must enter into the training of the medical, as indeed any other, student, practical observation resulting in first-hand knowledge, and some means by which he may be brought into contact with the observations of others, this latter having both a direct and an indirect bearing upon the former, serving at once to widen and direct it. These means resolve themselves into two—the lecture and the textbook. The lecture is a kind of middleman's product coming between observation and reading, and is unnecessary, as such products usually are. I dare affirm that very few students, even the most brilliant, ever come away from a lecture with the consciousness of a thorough mastery of the subject descanted upon. A good lecture delivered by an impressive speaker instinctively sends a man to his book "to read the subject up." His practical work will do this even more surely. No one, however, having mastered a division or subdivision of a subject in his textbook ever feels impelled to attend a lecture to cement his knowledge. He may go to the wards.

Then, again, it seems to me that the psychological processes involved in earnest book study are more conducive to comprehension and retention than those called into play in listening to a lecture. If invoked, the shade of Macaulay's schoolboy would doubtless tell us that visual perception, at least in adult life, is the most important of the psychological processes underlying cognition. The eye, moreover, is capable of a more prolonged stimulation that the ear without lapse of attention or sense of fatigue. In listening to an ordinary lecture the presentative aspect is purely auditory, the visual element being merely representative (as regards subject matter). In conning one's textbook the visual process is both presentative and representative, and we can get auditory presentation as well if we read aloud. We can also invoke the powerful aid of repetition. Who would not rather follow a long close uninterrupted stream of reasoning from his book than from a lecturer however eloquent?

Lastly, the matter gained from a chapter in a good textbook well-digested and self-arranged is far more the product of one's own mind, and the action and interaction between the knowledge thus gained and the first-hand knowledge born of observation far more valuable than can ever be the case with the predigested pap of the explanatory lecture, or the undigested encyclopædic lucubration which does duty for the ambitious one.—I am, etc.,

Broomhill, Oct. 9th. E. W. Adams.

INTRODUCTORY ADDRESSES FOR THE ROYAL COLLEGE OF PHYSICIANS AND SURGEONS OF EDINBURGH.

Sir,—After reading in the British Medical Journal the various introductory addresses given to the students on the occasion of the opening of the session of some of the universities and colleges of London and the provinces, it occurred to me what a pity it was that at the Royal College of Physicians and Surgeons of Edinburgh (one of the oldest, if not *the* oldest college), on the commencement of a new session, no such address of welcome and advice is given to the students.

There is no doubt that such an address, which need not necessarily be a lecture, would appeal to students at once, especially to those coming probably direct from school or home, loaded with good advice perhaps from relatives, but not such advice as would be of use in giving them an all-round view of their studies. What an amount of guidance they would obtain at the commencement of their medical career—advice and guidance of such a kind as they could only get from a lecturer or professor of their college.

There always appeared to me to be some want of co-operation on the part of the lecturers and a lack of interest in the students as a body, which cannot but have some effect on the students individually.

It must be evident to a good many that a good introductory address, such as is delivered at other colleges from time to time, given by one of the lecturers or by one of the members of the Governing Board, would be of infinite worth to a young student fresh to his work, and probably with not over-abundant knowledge as to the best steps he should take in his education for a medical career.

The Royal College of Physicians and Surgeons of Edinburgh has many things in its favour and it ought to be to the fore; but is that interest for the well-being of its students shown to them by the lecturers which should ever be evident in a well-organised college, and which would do much to improve the tone of a college, not altogether in the background?—I am, etc.,

Rusden, Northants, Oct. 9th. P. Ashworth Wedgwood.

THE PREVENTION OF PLAGUE.

Sir,—It may interest Dr. Haynes to know that an officer in India offered to stop the plague in Bombay by burning down all infected houses when the disease was somewhat limited, and thus (so he thought) effectually putting an end to plague in Bombay. His offer was declined by the municipal authorities. A large number of houses and outhouses were, however, levelled to the ground (without fire) with the hope of destroying every possible nidus for the plague germ, but without success, and many of the inhabitants were put to a great deal of inconvenience for want of houses. Other and less expensive methods have since been adopted, and appear to have been equally if not more effective, and certainly less inconvenient to householders. Would it not be as well, therefore, to study these methods before adopting the somewhat radical cure suggested by the officer referred to above and Dr. Haynes, which will of necessity increase the troubles of the already overburdened taxpayer, and may after all be of little use in staying the disease, or preventing a recrudescence of it, as it must be remembered that it is always liable to appear in one of our large ports.—I am, etc.,

Bedford, Oct. 10th. G. F. Poynder.

THE DEBATE ON ANKYLOSTOMIASIS: A CORRECTION.

Sir,—In the recent debate on this subject at the British Medical Association meeting, Major Giles is reported in the British Medical Journal of September 1st to have said:

> I will not here enter into the old controversy as to whether this increased mortality known by that name (kala-azar) is due, as I maintain, to ankylostomiasis *plus* malaria or to malaria *plus* ankylostomiasis, as advanced by Captain Rogers and Major Ross.

What an utterly incorrect statement of my views this is will be at once evident from the following quotation from my original report on the subject, in which I sum up the conclusion derived from a large amount of evidence on this point, thus:

> These facts prove incontestably that whatever kala-azar may be it is not ankylostomiasis, and, what is more, the latter disease is not even an essential factor in the production of the disease, and only complicates it in some 6 or 7 per cent. of the cases, just as it occasionally complicates every disease in Assam, and especially on tea gardens, and also I have no doubt in nearly every other part of India.

Many other passages to the same effect might be quoted, but that my meaning was quite clear is evident from the fact that Surgeon-Colonel Stephen in his review of 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 ankylostomiasis, and that ankylostomiasis is not an essential factor in its production." I have never, then, advanced the view that kala-azar is malaria *plus* ankylostomiasis, but have always maintained that it is purely malarial in its causation and pathology, so that I regret to say that the differences between Major Giles and myself are not so slight as he has deemed it convenient to assume, since Major Ross has so definitely confirmed my work upon the subject, as shown in the quotations of my contribution to the recent debate.

I may also take this opportunity of pointing out that when Major Ross says, in his paper in the same issue, that Giles's work on the life-history of the ankylostoma has not yet received either confirmation or rejection, he appears to have overlooked the paper of the late Dr. Macdonald in the report of the first Indian Medical Congress, in which he states that

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