Tenderness and a visible puffiness in right iliac region. Headache remains precisely as before. Temp. 98·0 at 10-0; 98·5 at 12·0; 100·2 at 14-0; 99·4 at 16-0; 99·0 at 18-0; and 98·2 at 20·0. Bowels once loose at 15-30. Tr. lavandulæ ʒj, aqua ℥j *ter die*, during the day.

1*st April.*—Feculent motions at 2-0 and 5-0. Temp. 99·0 at 6-0 and 100·2 at 8-0. Tongue clean. Tympanitis in right iliac and to left and right of umbilicus. Hepatic tenderness very slight; effervescing draught and vegetable bitter medicine during the day. Tongue clean but red. Temp. 101·2 at 10-0; 100·0 at 12-0; 100·4 at 14-0; 103·2 at 16-0; 102·0 at 18-0; 101·4 at 8·0. Semi-solid motions at about 10-0 and 15-0. Headache continues.

2*nd April.*—At 6-0 a copious pultaceous motion mixed with a quantity of slime; no more motions all day. Temperature normal all day, rising to 99·5 at 14-0. No abdominal tenderness or tympanitis. Hepatic tenderness very slight. Still some headache. Same medicine.

3*rd April.*—A quite natural motion at 6-0. Temperature normal. Still very slight headache and hepatic tenderness. Feels well; tongue clean.

4*th.*—Slight return of tenderness round the umbilicus. A healthy motion in the morning. Temp. 99·8 at 8 A. M.; a laxative ordered. Temp. 99·0 at 10-0 and normal all day, though bowels not open till next morning.

5*th.*—No abdominal or hepatic tenderness with the exception of slight discomfort on pressure above umbilicus. Head feels slightly dull. Is urgently required for duty. Discharged.

In this case the laxation of the bowels had been pushed too far on the 29th and 30th, causing a second rise of temperature on 1st April, due to passage of food residues over the inflamed tracts. This was terminated by a critical discharge of mucous mixed with fæces next morning, after which the abdominal symptoms and fever diminished. The case might easily have been mistaken for a malarial remittent fever, and is interesting for showing how an entero-septic febricula may be prolonged. For there is no reason whatsoever for supposing that the abdominal symptoms were here secondary to the fever, any more than there is for attributing to a septic fever the local inflammation or sepsis which is in reality its cause.

The above remarks refer only to natives. We must of course be careful not to mistake muscular for intestinal tenderness.

NOTES ON ANCHYLOSTOMIASIS, BEING, FOR THE MOST PART, A RESUMÉ OF A REPORT ON THE DISEASES KNOWN IN ASSAM AS KALA-AZAR AND BERIBERI.

BY SURGEON-CAPTAIN G. M. GILES, M.B., F.R.C.S.

WHAT is usually spoken of in Assam as beriberi has, for some years past, been known to be nothing more or less than anchylostomiasis.

This important fact was first made out by Dr. Rudduch, of Meesa, and, so far as I know, the profession in Assam are perfectly unanimous that he has established his point. The question of the appropriateness of the term will be considered below, but appropriate or inappropriate, the use of the word in connection with cooly anæmia is now so firmly established that I fear we shall have to put up with it.

"Kala-azar" of course means black fever. The diagnostic powers of the native are very limited, and he will usually tell you he has fever, whatever be the matter with him, so it is not surprising to find that in the cases that came under close observation in hospital, I found that a subnormal temperature was far more characteristic of the disease than pyrexia. That they may have had feverish attacks during the course of the disease is likely enough. In India malaria complicates, and not unfrequently masks every disease we have to do with, from stone in the bladder to typhoid fever. Now Assam is rather more malarious than most other parts of India, so that the inhabitants of that none too healthy province look upon an attack of fever as "quite in the day's work," and would probably be rather surprised than otherwise if they went many months without one. It is otherwise with Kala-azar, the appearance of which in a village causes a veritable and very well-justified panic.

The origin of the epithet black is not so easy to explain. The natives will tell you that subjects of the disease get darker skinned, but I believe I am in agreement with every medical officer who has studied the disease in saying that there is no real foundation for this notion.

Although, up to the time of my investigation, it had always been assumed that the disease was nothing more than aggravated malaria, it was early recognised that its characteristic symptom was not pyrexia but anæmia, and I think that the true explanation of the word kala is that which was suggested by Dr. J. Mullane, the then Civil Surgeon of Gauhati, as far back as 1887. This is that, though the complexion is not actually darkened, the extreme bloodlessness of the skin makes the normal pigment more apparent; the dull leaden hue of anæmia in fact intensifying the naturally dark complexion.

The local medical officers believed that the disease was also characterised by an enlarged spleen, but an examination of a very large number of cases in the districts showed me that even very advanced cases were quite commonly to be met with without any splenic enlargement; and, on the other hand, that enormous spleens were quite commonly to be met with in people who thought themselves quite healthy, and who for all practical purposes were so. In other words, when complicating kala-azar, enlarged spleen can only be considered a coincidence

In my report I have further demonstrated this point by a series of cases actually culled from the reports of those who entirely believed in *a causal* connection between the two pathological conditions. Microscopical examination of the fæces of a very large number of cases resulted in the discovery that the increased mortality of the affected districts was due to anchylostomiasis, and that it, and the so-called beri-beri, were practically the same disease, an opinion which my subsequent visits to the parts of the province where it is usually spoken of as beri-beri thoroughly confirmed. Of course every case that will be brought to one as kala-azar is not of this nature; for so great is the fear inspired by the disease, that all who are in any way sick are suspected of being victims to it, and if one only collected sufficient cases, one would find one had run through the entire nomenclature of disease—but that the real source of justification of this panic is anchylostomiasis, I do not entertain the slightest doubt.

As an example I may instance a case which was shewn me by a Hospital Assistant in Kamrup. The patient was a child whose very attitude rendered close examination almost unnecessary to diagnosis. "You look upon this as a 'pucka' case of kala-azar?" said I. "Yes, sir! he has an enlarged spleen," said the man, feeling the patient's abdomen through his clothes. I turned up the child's shirt and showed him a large lumbar abscess. The case was one of caries of the spine, and the supposed spleen happened to be the thickening round a psoas abscess.

In this case the patient's friends did not produce the case as one of kala-azar, but I have had brought me by them as such quite as obviously casual cases. The method, however, in which the medical subordinate arrived at his diagnosis illustrates very well how the notion of the connection between enlarged spleen and kala-azar has arisen,—and it is obvious that if valid, kala-azar would be found to be universally present throughout the length and breadth of the tropics.

Practically then my task resolved itself into clearing up such points in the etiology of anchylostomiasis as had been left uncertain by previous investigators. But before proceeding to describe the results arrived at, it may be well to devote some space to the consideration of what is meant by beri-beri here and in other parts of the world; as much confusion has arisen from the unfortunate application of the term to a variety of utterly different diseases.

To begin with, the word labours under the disadvantage of having no vernacular meaning in any language, and how it came into use at all is a mystery; for, as was long ago pointed out by Malcolmson, the word is absolutely without meaning for any native.

Unfortunately, this word beri-beri originally, as we have seen, meaningless, has come to be applied to several distinct diseases in different parts of the world.

Fifty years ago Malcolmson in Madras (*loc. cit.*) described a disease under this name in which the leading symptoms were paralytic.

I do not think there can be any doubt that the disease he describes was identical with that described by Pekelharing and Winkler in their report to the Dutch Government on an epidemic among their troops in Achin.

For cases illustrating the identity of the clinical pictures drawn by the two authors, I must refer the reader to pages 3—6 of my report, as their reproduction here would occupy too much space. These Dutch observers establish clearly that this disease they had to deal with is essentially a peripheral neuritis, and describe a micro-organism, which they succeeded in cultivating, and which, they advance good reasons for believing to be the actual cause of the disease.

It is noteworthy that the cases described by both Malcolmson and the Dutch investigators are mostly soldiers, Native and European. We know that, 50 years ago, Indian barracks were terribly over-crowded. There is internal evidence in the Dutch report that the same was the case in Achin.* Indeed what is recommended, is change of air and plenty of it, and I believe that an English combatant officer, without any scientific assistance whatever, might have arrived at the same conclusion by a simple reference to our barrack regulations as to the cubic space that should be allowed per man. Hence I am inclined to believe that the disease is essentially one arising from the over-crowding of large bodies of men in single rooms in tropical climates; and that is probable the reason why, at the present day, we see no such cases in India.

To the medical observer, no two diseases could well be more distinct than this peripheral neuritis is from anchylostomiasis, but to the lay mind the points of resemblance are sufficient to cause confusion, and a disease endemic in Ceylon, which has recently been proved without doubt to be anchylostomiasis pure and simple, in some way got to be also called beri-beri.

The "kakke" of Japan again is often so spoken of, but appears to be certainly not always anchylostomiasis. Its etiology was most carefully investigated by Dr. Wallace Taylor of Osaka (*vide* abstract of his Researches, *London Medical Record*, 15th March, 1886), and, if bacteriological evidence is to be trusted, the disease must be considered entirely distinct from the beri-beri of Achin, as no two micro-organisms could well differ more entirely than Taylor's does from that of Pekelharing.

* *Vide* also *Indian Medical Gazette*, 1887, p. 384.

Again Dr. Lacerda (in A Unāio Medico, January 6th 1884, of Rio de Janeiro) describes a microorganism which he cultivated from cases of "beri-beri" that occurred on board the corvette *Nictheroy*, and which may possibly be identical with that described by Taylor. We are not told where the corvette had been cruising. It is of course possible that she may have been to Japan.

On the other hand, cases occurring in Brazil in some reports spoken of indifferently, as hypoæmia and beri-beri are often instances of pure anchylostomiasis; for undoubtedly uncomplicated cases of that disease originating in Brazil, *vide* Cobbold's "Parasites," p. 213, and P. Polatti, abstracted in *London Medical Record*, August 15, 1884, p. 355.

No such disease as the epidemic peripheral neuritis of Achin is, however, to be met with in Assam, and though I have seen a good deal of India from the Oxus Valley to Cape Commorin and from Suddia to Bombay, I can nowhere remember meeting with any case at all resembling it, nor have I met with any one who has, and so very much doubt if it is still to be met with in India.

Hence it is rather amusing to find the *British Medical Journal* gravely taking the profession in India to task for leaving to foreigners the investigation of a disease which we have no opportunity of studying, while it falls into the error of imagining that the cases of anchylostomiasis which we speak of as beri-beri must necessarily be the Achinese disease with a quite unimportant parasitic complication.

A very short experience of a "Black listed" Assam tea garden would, I am sure, convince the writer of the journal's leaderette that anchylostomiasis, without the aid of any other pathological condition, is quite capable of acting as a plague to which Achin experiences of epidemic neuritis are mere child's play.

The only modern reference that I can discover to Indian cases at all resembling the epidemic palsy of Achin are two cases, quoted by Dr. Norman Chevers in a paper on "Beri-beri Fever" read before the Medical Society, London, March 31st, 1884, in which "Dr. Harvey noticed that in two cases there was an appearance of paralysis in the lower limbs," but these cases occurred in the course of an epidemic in Calcutta of an "exanthematous fever, characterised by general dropsy, effusions into serous sacs and other grave sequelæ." Anything more different than is this description from the apyexic-non-exanthematous peripheral neuritis of Achin cannot well be imagined, and moreover Dr. Harvey, even in these two quite exceptional cases, expresses a distinct opinion that the paresis was of central origin, being due to an extension of the general dropsy to the spinal canal causing pressure on the spinal cord.

The whole indeed of the paper in question is a very good example of the confusion that must necessarily arise from treating as necessarily one and the same disease anything that anyone may have chanced to describe under this pernicious heading of Beri-beri, an obvious outbreak of naval scurvy being included in the collection.

The sooner medical writers drop the use of the word the better, as the only way out of the present confusion is to use only intelligible medical terms for the future. If a case be epidemic peripheral neuritis let us call it so; if it be one of anchylostomiasis, let us speak of it as such.

Whatever may be the case elsewhere, the disease which is devastating Assam is anchylostomiasis, and nothing at all resembling the various other diseases that share the name of beri-beri, is to be met with there. Let us then return to the consideration of anchylostomiasis.

The Life History of the Parasite.

THE existence of a free stage, or rather a knowledge of the fact that the parasitic ova were capable of hatching out in the fæcal deposits of patient, has long been known; but, owing, as I believe, to too great dependence on observations conducted in small microscopic cells and other laboratory expedients, entirely erroneous ideas have prevailed as to the subsequent history of the hatched out embryos; and the cardinal new fact that I have been able to establish is that these embryos are capable of becoming sexually mature, and of giving origin to an indefinite series of generations of free living animals, utterly different in nearly every point of their anatomy from such of their brethren as succeed in effecting a lodgment in a suitable host.

For the details of the growth and development of these free stage rhabdites I must refer the reader to the pages of the report in question; but broadly speaking the process is as follows:—

Let it first be clearly understood that the parasitic *dochmius* is never viviparous. Within the intestine of the host, the segmentatives of the ova can only proceed to a certain extent, beyond which further development is impossible except in the presence of a free supply of oxygen, a condition which is wanting within the intestinal canal of the host. Hence the ova can never hatch out until, with the deposited fæces, they gain access to the outer air. For the same reason, infection by the ingestion of ova is clearly impossible, and can only take place by the swallowing of already hatched out embryos.

As found in the excrement of patients, the ova are generally 2-8 segmented, and measure $\frac{1}{120}$ inch in length by $\frac{1}{330}$ inch in breadth. They have an elliptical outline with the long sides somewhat flattened, and consist of a yelk surrounded by a broad zone of perfectly clear fluid

and enclosed in a very delicate shell. The only constituents of human fæces with which they can possibly be confused are the ova of oxyurides and certain psorosperms, which are very commonly to be found in anæmic native patients. From the former they may be distinguished by their larger size, oxyuris ova measuring only $\frac{1}{490}$ inch in length; by the ova of this parasite being flattened on one side only, and by their generally containing a well-advanced embryo: while psorosperms may be distinguished by their irregular size and shape, by far the larger proportion being very much larger than the ova of any parasite for which they could possibly be mistaken.

The time which elapses between the deposition of the fæces and the hatching out of the embryos varies a good deal with the temperature of the air and the supply of oxygen. At average temperatures they hatch out the second day: as minute worms 0·085″ in length by 0·005″ at their thickest part. The intestinal canal is well developed, the pharynx exhibiting two dilatations or bulbs, the hinder of which is in a constant state of pulsation. There is, however, no indication whatever of the generative organs.

Under normal conditions, it is obvious that the little nematodes will find themselves domiciled in the fæcal mass of their birthplace; and the second great point to understand is that this and fouled earth are their only natural habitats, removal from which will at once stop their further development, and ultimately result in their death.

Owing to this, the continuous observation of any individual specimen from birth to maturity is a matter of impossibility, and a correct idea of the process can only be gained by the frequent examination of a number of individuals taken at various stages from a large cultivation.

Microscopical examinations of these organisms can only be conducted when they are immersed in water, and, although they are wonderfully tolerant to all sorts of adverse influences, there is, so far as I can see, no condition more hostile to them than this. In water they become sluggish, get coated with a deposit of granular matter, and slowly die. And this process has been made the subject of elaborate papers, describing the "encapsuling and calcification" of the embryos. It has, however, no more to do with the normal life history of the worms, than the change, of a corpse into adipocere is in that of a man—for it is not even a part of normal *post-mortem* changes.

It is owing to the overlooking of this second point that previous investigators have entirely failed to follow out the natural history of the free stage worms.

(To be continued.)

A Mirror of Hospital Practice.

A CASE OF MINERAL OR CALCAREOUS DEGENERATION OF THE CRYSTALLINE LENS.

BY ASSISTANT-SURGEON H. CHATTERJEE,
Barabanki.

MUSSTT. ZAHURAN, an elderly Mahomedan female, aged about 50, came to this hospital with a view to seek advice for total failure of sight in both her eyes. The patient appeared otherwise to be in a fair state of health.

Previous history. — About 20 years ago the patient went on a pilgrimage to Mecca. Six years after her settling there, she got an attack of some nervous disease (probably facial palsy). Previous to this she had led a fairly healthy life. She consulted with some Hakims of Arabia about her malady, and was subjected to a prolonged and repeated course of antiphlogistic and depressing treatment. Bleeding was several times had recourse to. This course of treatment, though resulting in cure, proved worse than the disease itself. It seriously undermined her health and brought on an impoverished state of blood and increasing debility and interfered with the proper nutrition of the body. This weakened state of her health proved sufficient to induce failure of her sight first in one and then in the other eye. The process had been very slow and occupied a period of four years to reach to this extreme. Pain about the eye and any other sign of deep inflammation had never been noticed by the patient.

Present state. — On looking at her eyes here and there red streaks of conjunctival vessels were seen on the sclerotic, the eyes were a little watery and a slight mucous discharge was seen at both the canthi; tension in the right eye was = T1 and in the left normal; the anterior chambers were not shallow, but posterior synechia was discovered in both the eyes. The lens in the right eye presented a somewhat peculiar appearance, the inner half of it was of a dirty grey colour, and the outer half seemed as if it was covered with a blood clot advanced in the process of absorption. The left cataract presented a whitish yellow appearance. The pupil in the right eye was quite irregular; with the aid of a few drops of atropine, part of the lower and inner segments was dilated. The left pupil was transversely oval with constriction in the middle.

From a consideration of the above facts, the case did not seem to be a favourable one, and the relatives of the patient were informed accordingly. This discouraging statement made her leave the hospital. After a week or so the patient