amblyopias, a single track is attacked; sometimes, as in quinine amaurosis, the entire optic nerve is attacked. What particular active principle produced these changes is not known, but de Schweinitz has long contended, in the case of tobacco, that it is not the tobacco alone, or its active principle nicotine, which is the essential poisonous agent, but that one or more of the many principles freely present in tobacco smoke, or some toxic influence which they liberate in the system, is to blame. He mentions that experiments now being performed in Chicago by Dr. Casey Wood indicate that certain stomachic toxins are capable of causing in animals blindness, probably of the type under consideration.

Although excessive tobacco smoking and the taking of quinine in large doses are both very common in India, as pointed out in the case of quinine in the October number of the *Indian Medical Gazette*, cases of toxic amblyopia due to them are very rare, if one is to judge from the journals or from what one hears. In the *Transactions* of the Indian Medical Congress (p. 505) a case of quinine amaurosis is described by Surgn.-capt. A. E. Roberts, I. M. S., which followed the swallowing of five drachms of the sulphate. The auditory nerve recovered within a week, but the optic nerve took much longer, moving objects not being perceived until the third or fourth week. Colour vision returned about the sixth week.

In the *Journal of Eye, Ear and Throat Diseases* (*Baltimore Md.*) July 1897, there is a paper by de Mecker of Paris recommending the injection of serums for the cure of toxic amblyopias. The paper does not pretend to be more than a sketch, and nothing much in the way of evidence is adduced. De Mecker regards these cases as due to direct infection of the optic nerve trunk and injects large quantities of serum with the idea of washing out (*lavage*) the harmful infectious substances from the blood and lymph currents. He injects 50 to 60 grammes a day (even up to 150 or 200 grms.) of Cheron's 'serum' which consists in 100 parts of, 1 of white phenic acid, 2 of chloride of soda, 8 of sulphate of soda, and 4 of phosphate of soda. The results are described as good, and there is rapid improvement in vision, from $\frac{1}{20}$ up to $\frac{1}{4}$ or $\frac{1}{3}$ in a few days.

Medical and Surgical Reporter, Philadelphia, July 26th, 1897.—A. S. Thomson reports a case of toxic amblyopia due to acute poisoning from over-use of Jamaica ginger. It went on to atrophy. Several other cases are said to have occurred from drinking Jamaica ginger as a substitute for whisky.

Otitic Brain Disease (*Boston Medical and Surgical Journal*, August 12th, 1897). —J. Orne Green has a short paper on some of the general principles which should govern operations for otitic brain-disease. It is mainly statistical, and includes tables taken from Körner and Pitt. Körner's figures show that in Prussia 0·63% of all deaths are due to otitic brain-disease. These deaths occur mainly between 10 and 30 years of age; and 5·15% of all deaths between 10 and 20, and 3·85% of all between 20 and 30 are due to this disease. About 5% of meningitises Pitt found *post-mortem* to be otitic, and about one-third of all brain abscesses to be of otitic origin. Körner found 8·7% of all brain abscesses to be multiple. In 109 cases of otitic brain disease, Körner found that the bone was diseased directly opposite to the dura mater in 79%, which is an important fact as justifying early exploration of the bone. Dr. Green points out how positive localising symptoms are in a very large proportion of the cases wanting throughout the disease. He says: "Given a suppuration of the ear with symptoms however slight pointing to the brain, open the ear cavities and explore the bone;" and summarises as follows:—

"(1) In otitic brain-disease early operation is advisable, but an early exact diagnosis is often impossible. (2) The chances are 79 in 100 that a fistula through the bone will lead directly to the brain-disease. (3) The infected ear requires operation in any case, and this operation can be combined with an exploration for the bony fistula and the recognition and treatment of the brain disease."

In the same journal for August 19th, 1897, Drs. Richardson and Walton report a case of *temporo-sphenoidal tumor* presenting symptoms suggestive of abscess. It proved to be a small-celled glioma, but the chief interest in the case lay in the fact that the patient (aged 69) had suffered for several years from otitis media with pain and discharge, and that the glioma, whose origin was of course entirely independent of the ear (all may not agree with the reporters in this opinion), appeared in the exact location usually occupied by cerebral abscess arising from aural infection.

F. P. Maynard, M.B., D.P.H.

Reviews.

THE KALA-AZAR REPORT.

REPORT OF AN INVESTIGATION OF THE EPIDEMIC OF MALARIAL FEVER IN ASSAM OR KALA-AZAR.

By Leonard Rogers, M.B. (Lond.), F.R.C.S. (Eng.), Surgeon.-Capt., Indian Medical Service. Shillong Secretariat Press, and Thacker, Spink & Co., Calcutta, 1897.

We propose here to consider in detail this valuable report by Dr. Rogers on a disease which has been almost as fertile a source of confusion to medical men as it has been destructive to

life and labour in the districts affected. We welcome it all the more because it contains incidentally the fullest exposition of the sequelæ of malaria which we are acquainted with by any Anglo-Indian writer. We will take up the subjects in the order they are given in the report. It is not necessary to summarise it, as an excellent *resumé* of its contents has already been published in the form of the letter of the Sanitary Commissioner of Assam given in our last issue (November).

We need not trace the well-known history of *kala-azar* from its recognition in the Garo Hills to its present widespread distribution. The descriptions written by Dr. Clarke and by Mr. McNaught fifteen years ago are still excellent and true, nor is it necessary to more than briefly refer to the recent controversy about the disease which arose over the publication of Dr. Giles' extremely able report of 1889. It is well-known how Dr. Giles, having found the anchylostoma parasite in cases of *kala-azar*, concluded that the disease was an anchylostomiasis, complicated with malarial cachexia. Dr. Giles, however, began his study of the disease at a period of the year, the cold weather, when it is, as is now shown, at its minimum, and as *kala-azar* is essentially a chronic disease, it is clear that it is only by observing cases all through, especially during the rainy season, that it is possible to get a complete picture of the disease * We may state that after reading the clinical descriptions of cases of *kala-azar*, we are convinced of the identity of *kala-azar* cachexia with the ordinary severe malarial cachexias, only too familiar to us in our jails and civil dispensaries; but for the purpose of emphasizing these points we must briefly run through some of the symptoms. Just as in our jail returns so in *kala-azar* dispensaries we find the disease (malarial cachexia) often entered under various headings, *viz.*, anæmia, diarrhœa, dropsy, or that quaintest of diagnoses (!) "debility."

It is only when the sick person is continuously observed for months that a correct view can be taken of the disease, as Dr. Rogers has pointed out and acted upon, for it is essential to grasp the fact that the disease is a cachexia. Fever of the intermittent or remittent type may be present or absent at any particular date, but the progressive weakness and prostration remain.

Turning now to the detailed symptoms of the disease an enumeration of them will show how closely they are alike in both chronic and epidemic malaria. The pearly blue or dusky yellow conjunctivæ, the pigmented patches on the buccal mucous membrane (though here we agree with Dr. Rogers and Dr. Maynard [*Indian Medical Gazette*, October 1897], in thinking these of anthropological interest only), the dropsy of cardiac origin, the enlarged spleen, the cirrhosed liver, the œdema of ankles, feet and face, the small hearts found at *post-mortem* examination, the nyctalopia, the occurrence of pneumonia, the sudden onset of œdema of the lungs, the atrophy of the mucous membrane which allows of the food passing through the intestinal canal undigested, the rarity of albuminuria, the low *sp. gr.* of the urine (common in all rice-eaters), the dusky leaden hue of the face and complexion, all these are well-known familiar signs of ordinary malarial cachexia, and all these are found in cases of *kala-azar*.

We have referred to these details only to point out that the symptoms of *kala-azar* given by Dr. Rogers exactly correspond with our experience of ordinary malarial cachexia, and this fact is an important confirmation of Dr. Rogers' view that *kala-azar* is only intensified malaria. There are, however, one or two points which we are surprised to see not given in the description of *kala-azar* cases. In Bengal at least there are two very familiar symptoms of malaria which Dr. Rogers does not refer to and which we expected to find in *kala-azar* cases, *viz.*, dysentery, which all Bengal jail medical officers recognise as a frequent terminal phenomenon of malarial cachexia cases. We expected to hear more of the *kala-azar* cachexia cases dying of dysentery. Again at the end of every fever season in our Bengal jails and dispensaries we see numerous cases of spongy and ulcerated gums which, as Dr. A. Crombie pointed out years ago, are certainly not scorbutic, nor do they respond to anti scorbutic treatment, moreover they are only found in malarial years. They were conspicuously absent from the jails in Bengal during the healthy years, 1895 and 1896. The very high death-rate given by Dr. Rogers for *kala-azar*, viz., 96 *per cent.*, contrasts very strongly with any recorded death-rate from anchylostomiasis, but as *kala-azar* is *ex hypothesi* the most severe form of malarial cachexia, this high rate is scarcely surprising.

We have not much to say about the remarks on treatment in the report. It is obvious that not much can be expected. As in ordinary malaria so in *kala-azar* vigorous anti-malarial treatment in the early stages is strongly indicated, good food, plenty of milk, change of air, quinine, arsenic, and iron are the lines of treatment in both cachexias. Dr. Rogers' use of bone-marrow is suggestive; it should also be tried in ordinary cases.

We now come to an important chapter in the report—on the blood changes in *kala-azar* and anchylostomiasis. Here Dr. Rogers considers he has grounds for making a certain differential diagnosis between these two diseases. We may

* This argument cuts both ways, it is possible to say that Dr. Rogers, observing the disease during the malarial fever season, has attributed to *kala-azar* symptoms due to ordinary seasonal fever, and upon these fever-symptoms has built up his clinical picture of the disease.

sum up the differences as given in the report in the following table :—

TABLE I.

BLOOD CHANGES.

Kala-azar.	Anchylostomiasis.
1. Hæmoglobin reduced to 33 per cent. (average).	1. Hæmoglobin reduced to 15 per cent. (Rogers). 26 ,, ,, (Sandwith). 16 ,, ,, (Leichenstern).
2. Both the hæmoglobin and the number of the red corpuscles almost equally reduced.	2. Hæmoglobin reduced almost twice as much as the numbers of red corpuscles are reduced.
3. White corpuscles reduced (except during the leucocytosis of an actual fever attack).	3. White corpuscles very slightly reduced.
4. Sp. gr. of blood slightly reduced, av. 1048.	4. Sp. gr. much reduced, av. 1034.

The following table is quoted for the benefit of observers in other countries who may not see the report, more especially as much attention is sure to be paid to this point in countries like Fiji, Ceylon, Borneo, &c., where both malaria and the anchylostoma prevail. If Dr. Rogers' opinion that "the anæmia of anchylostomiasis differs from that of *kala-azar*" (and we may add ordinary malarial cachexia) "in every way it well could," be confirmed, it is clear we have here an important means of distinguishing between the two diseases.

TABLE II.

	Percentage of hæmoglobin.	Red corpuscles per cubic millimetre.	White corpuscles per cubic millimetre.	Ratio of white to red corpuscles.	Specific gravity.	Hæmoglobin value.
1. Healthy natives of Assam ...	62	4,734,000	7,325	1 : 684	1·054	·65
2. *Kala-azar* cases	33·4	2,462,000	2,600	1 : 1170	1·048	·65
3. Ordinary chronic malaria ...	31·6	2,000,000	1,600	1 : 1400	1·042	·73
4. Anchylostomiasis	15·2	1,145,000	5,338	1 : 524	1·034	·31
5. *Kala-azar* and anchylostomiasis ..	27·4	3,120,000	3,200	1 : 975	1·039	·43

In order to exclude the possibility of *kala-azar* being a new disease, Dr. Rogers made a series of examinations and cultures but failed to find any specific organism. In this point his experience exactly agrees with that of Dr. Giles.

As regards the presence of the malarial amœba or plasmodium, Dr. Rogers states that he spent much time in examining the blood in the hopes of finding some new form of the malarial parasite which would account for the virulent nature of *kala-azar* malarial fever, but though the ordinary familiar forms were regularly present, he saw nothing new nor anything which would serve to differentiate *kala-azar* from ordinary malaria.

At page 117 begins a discussion on the nature of *kala-azar* which we have already referred to. It is clear *from the report* that the parasite (anchylostoma) is not sufficiently common nor found in sufficient numbers in cases of *kala-azar* to justify the disease being even in part attributed to this parasite. It only complicates, says the report, about 6 or 7 per cent. of *kala-azar* cases just as it complicates every other disease in Assam. For a long time we confess we were strongly attracted by the view that *kala-azar* was a combination of anchylostomiasis and malarial cachexia, and even after reading this report we cannot altogether dismiss a parasitic cause. Dr. Giles' report was of extreme value in being the means of calling the attention of the profession to the widespread prevalence of this parasite in India. At present in Upper Assam there are plenty of cases of anchylostomiasis (meaning thereby a cachexia produced by the worm), but we have the recorded opinion of the planters' doctors assembled at Kukilamukh that the *kala-azar* cases shown to them "were entirely unknown in Upper Assam," and "differ in every particular from the anchylostomiasis cases with which they were familiar." *

These cases are apparently returned as anchylostomiasis but are often spoken of as "beri-beri," meaning thereby cases like the "anæmia of Ceylon." Will the misuse of this grotesque word beri-beri never die out?

Though it is perhaps outside the scope of his report we would have liked Dr. Rogers to explain how it comes that the parasite which is admittedly so widespread in Assam does not do more harm in that province to the inhabitants whose conservancy arrangements were described in Dr. Giles' report, and are certainly as bad as those of the coolies in Ceylon, Egypt, and elsewhere, where the ravages of the parasite are admitted. How is it that in the *kala-azar* affected parts of Assam the parasite

* This is not a very clearly expressed opinion. It can only mean that cases like those (of kala-azar) shown to them do not prevail in Upper Assam *with a history of infectiousness, &c.*, or in epidemic prevalence. For as clinically an individual case of *kala-azar* is *ex hypothesi* identical with a case of malarial cachexia, it cannot be meant that ordinary malarial cachexia is "unknown" in Upper Assam. What they meant to emphasise was that *kala-azar* is something quite different from anchylostomiasis.

does not spread, and is found in its hundreds and thousands in the intestines of the inhabitants as it is in Egypt of to-day? In fact, the worm being admittedly present, how is it that it does not "sap the lives" of the Assam peasantry as Dr. Sandwith tells us it does in Egypt? We know that Dr. Rogers does not say the worm is harmless, but only that it does not do the harm in *kala-azar*. Yet as this question is interesting in every country to which the useful and ubiquitous Indian coolie proceeds, we would like to have seen this point discussed, more especially as the report will be considered as a direct refutation of Dr. Giles' theory.

To return, however, to the report. It appears to be clearly established that *kala-azar* has now died out in the parts first affected,* and this fact we look upon as fatal to Dr. Giles' theory. It is strange also to find that the disappearance of the disease was recorded in a district report as far back as 1887, two years before Dr. Giles' investigations. How was such a fact not brought to his notice?

We must perforce pass over much in this interesting report as we have got to consider some of the most important points. We have shown that we are inclined to accept Dr. Rogers' arguments that *kala-azar* is an intense form of malarial cachexia, though we are not of his opinion that the disease is exceptionally rapid in course. We have certainly seen in Bengal cases of illness which terminated in profound malarial cachexia and death in five or six months from the first attack of intermittent fever; of course such a patient may have had ague several times before in other years—few escape it, and the same must be true of *kala-azar* cases.

This however is by the way. We have now to consider Dr. Rogers' views as to the origin of this epidemic malarial fever and the important question of its communicability.

As regards its communicability. This is a prevalent opinion, it was admitted by Dr. Giles, and no one after reading section 7 of this report can doubt the fact. This being granted, it undoubtedly raises the presumption that the disease must be of other than of a malarial nature. Now Dr. Rogers' view of the origin of *kala-azar* is that it arose "from an intensification of the Rangpur malarial fever of the early seventies owing to a succession of unhealthy seasons," and that it crossed over from that district along two lines of communication between Bengal and Assam. Now this involves the proposition that malarial fever may exceptionally attain to contagious properties. As no one has ever succeeded in finding the amœba of Laveran outside the blood corpuscles, it is obvious that no laboratory experiments can be made to prove an intensification of the germ possible. We must therefore fall back upon analogies in other diseases and upon history. Dr. Rogers quotes the instance of the "Burdwan fever" epidemic (1862—73), which was almost certainly purely malarial, and which was also contagious or communicable in the same way and to the same extent as *kala-azar* is shown to be. The case of the great malarial epidemic in Mauritius (1865) is not quite on all fours with the Burdwan epidemic, for it was introduced, it is said, by Indian coolies into an island which had hitherto been free from the disease, and it is well-known how terrible at times is the fatality of a disease when first introduced among a people with no ancestral experience of it, and among whom there can be no protective evolution against it,—for example, tuberculosis has caused the gradual extinction of the Maories, and it is well-known how measles ravaged the Fiji Islands when first introduced there. Dr. Rogers also quotes the case of pneumonia being at times infectious, and from accounts of its spread in outbreaks on the North-West Frontier and Biluchistan, it is difficult to deny that it was on these occasions infectious. Pestis minor is probably another example of a non-infectious type of a certainly infectious disease. Some people have even said that dysentery has been at times infectious in certain Bengal jails. There are numerous examples in history of sudden and widespread outbursts of malaria such as happened to Wellington's Army before Cuidad Rodrigo and to the British Army at Walcheren, but these are probably only examples of great prevalence and severity rather than contagion.

The malarial germs are however said to be saprophytic, having their normal habitat outside the human body, though capable of existing within it, and as they have been shown to be affected, becoming less malignant and less prevalent, by cultivation, drainage, deforestry, &c., it is, therefore, not improbable that they can likewise assume an increased malignancy under long continued favouring conditions of heat and moisture, such as Dr. Rogers has attempted to prove existed in Rangpur at that period; but becoming more intense in the sense of being more virulent or

* Yet we find Dr. Fink, Civil Surgeon of the Garo Hills, writing in this journal a few months ago, speaking of seeing cases of *kala-azar* within the present year. Some cases he describes among Native Christians with paretic symptoms are certainly not *kala-azar*, whatever else they may be. It is certain, however, from his lengthy article that the Garos themselves are not aware of their good luck in having got rid of the epidemic The fact seems to be that epidemic contagious malarial fever is absent from the Garo District, but of course plenty of cases of malarial cachexia remain which individually resemble cases of *kala-azar*. This fact of *kala-azar* having passed away from districts formerly affected is a strong argument against the worm theory, unless it can be shown that such parasitic diseases have also a tendency to die out or pass away. Can any information that anchylostomiasis has passed away from portions of Ceylon or Egypt, &c., be produced in the absence of great sanitary improvements? If so, this would be a great argument in favour of the worm theory.

more prevalent is not the same as a non-infectious disease becoming infectious. It is clear therefore that these arguments though in favour of Dr. Rogers' contention do not amount to a proof that malarial fever whether in Rangpur or not became infectious. We say "whether in Rangpur or not," because we are not convinced that *kala-azar* had its origin in that district. From what we read of the fever of Rangpur and Dinajpur from 1870 to 1875 it is certain there was an extremely bad form of malaria present, but we have no evidence produced that the fever of Rangpur was "a travelling fever" which "appears, spreads, prevails for a certain time, and disappears" as was written of the Burdwan epidemic, and as Dr. Rogers has shown *kala-azar* to be. We agree, however, with Dr. Rogers that the present Assam and the old Burdwan epidemic are essentially identical. We observe that the Sanitary Commissioner of Assam calls attention to an apparent difference between the Burdwan and the Assam fevers, in that the former was said to be frequently attended with cerebral symptoms. Certainly such cases were noted, and on account of their sudden and striking character attracted much attention, but we have before us the testimony of an able though anonymous writer who wrote a pamphlet* in 1864, criticising the opinions of a committee who had been investigating the disease. He distinctly states that he had seen cases throughout the epidemic, and that "the primary action of the fever was on the blood, and was destructive of the red corpuscles;" also "that *cerebral congestion was a very rare phenomenon of the present epidemic ..... the fever was one of constant relapses ..... the type of the disease being essentially asthenic.*" This is important contemporary confirmation of Dr. Rogers' remark that cerebral cases were the exception in the Lower Bengal epidemic.

Space forbids us to deal further with this interesting report. We have shown that we consider it a valuable one, and that we feel bound to accept Dr. Rogers' view that *kala-azar* is only an intense form of malarial cachexia, which has acquired contagious qualities in a way not certainly determined, but probably under conditions such as started the Lower Bengal epidemic in Jessore.

From the evidence given in the report, and from the fact of the epidemic having left parts formerly affected, we feel that we can no longer consider the anchylostoma theory of Dr. Giles tenable, though we confess our inability to understand why this parasite has been unable to be as destructive in Assam as it is known to be in Ceylon, Egypt and many other countries.

We have no space to discuss the recommendations in the report for mitigating this veritable plague. They appear reasonable and practical, and we trust they will speedily be put into force.

We congratulate Dr. Rogers on his able and extremely well-written report, which will remain a valuable contribution to Indian medical literature.

ANNUAL REPORT ON MEDICAL INSTITUTIONS, MADRAS CITY.

THE annual report on the working of the Medical Institutions in the city of Madras, during the past year, contains much interesting information. Fourteen institutions in all are dealt with, nine of which accommodate both in- and out-patients, whilst five are merely out-patient dispensaries. The chief of these institutions are the General Hospital with 474 beds, the Maternity Hospital with 140 beds, and the Ophthalmic Hospital with 76 beds,—all Government institutions, where clinical instruction is given to students of Medicine. The other hospitals dealt with in the report are the Native Infirmary (where clinical instruction is given to students of the Hospital-Assistant Class), the Leper Hospital, the Voluntary Venereal Hospital, the Caste and Gosha Hospital, and Sir Ramasawmy Moodaliar's Maternity Hospital.

Several of these institutions, notably the General Hospital, were full to overflowing, and the authorities were compelled to refuse admission to many patients, owing to lack of accommodation. Many of the patients who seek relief at the General Hospital, as well as the Ophthalmic and the Maternity Hospitals, are not inhabitants of the city itself, but come from the mofussil.

The number of persons treated during the year under review was 203,854, of whom 17,605 were in-patients, and 186,249 out-patients.

Diseases of the digestive system furnished the largest number of cases, and 10,580 persons were treated for intestinal worms. It may be assumed that a large proportion of these cases were due to preventable causes, particularly to defective drinking water. The water-supply of the city, although of fair natural quality, is not submitted to any process of filtration. Malarial fever appears to have been much in evidence, 17,845 persons having been under treatment for the disease. Considering that Madras is not deemed a particularly malarious spot, this seems a large number. During the hot weather a peculiar fever of a continued type, but which cannot at present be classified, is very prevalent. In some localities of the city almost every house contributes to the victims of this disease.

Turning to surgical work, we find that syphilis, and gonorrhœa and its sequelæ, including stricture of the urethra, furnished a very large quota of cases; and a large number of persons

* "The Epidemic of Fever in Lower Bengal." Calcutta: Stanhope Press, 1864, obtainable from Messrs. Thacker, Spink & Co.