REPORT ON KALA-AZAR,


सत्यमेव जयते

REPORT

OF

AN INVESTIGATION OF THE EPIDEMIC OF MALARIAL FEVER IN ASSAM,

OR

KALA-AZAR

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SECTION I.

THE HISTORY OF KALA-AZAR.

THE main facts of the origin and spread of the disease known under the name of *Kála-ázar*, as far as they can be gathered from the yearly Sanitary Reports of Assam, will be briefly given here, as far as possible in the words of those who have seen most of the disease. Further details, with statistics and map illustrating its spread, will be found in Section VI.

The disease was first described in the report for 1882 by Dr. Clarke, and was based on old records and notes of 120 cases by Mr. McNaught, the Civil Medical Officer at Tura, the headquarters of the Garo Hills district. As this report is of great interest, a few quotations will be given from it, which will show the view which was taken of the disease at this period :

"There is no registration in the Garo hills; but from information gathered in a general way, and from the depopulation of certain areas, there can be no doubt but that a very high rate of mortality exists among the Garos and other tribes, especially those tribes who occupy the low hill tracts which lie between the hills and the plains. There is a form of malarial poisoning known among the natives as *kála-ázar*, or black sickness, from the darkened colour which the skin assumes

in chronic cases. The popular idea is that the disease is contagious, and the natives have so great a horror of its appearance among them, that they frequently separate those affected from the other members of the family or community. Mr. McNaught, the Civil Medical Officer at Tura, has made some enquiries concerning the disease. He states that it begins with a high temperature, severe pains in the head and body, loss of appetite, and other symptoms of a general febrile condition: fever sometimes of an aguish form, and sometimes fever without remission for many days together. The spleen and liver enlarge, the skin becomes gradually darkened, and in advanced cases there is often hæmorrhage from the nose and gums; œdema of the feet or general dropsy is likewise common, and life ends by a combination of disordered functions known as malarial cachexia.

"This does not appear to me any new form of disease; its history, so far as I can at present gather, shows it to be produced by intense malarial poisoning. It has been known as it is now among the people for the past twenty years; and the localities chiefly in which it prevails, are those which combine all the conditions under which the most intense forms of malarial disease are produced. Further investigation is necessary to throw a clear light on this disease. It is so inimical to life, that large tracts of hill country are being depopulated in consequence."

In Appendix A to the same report, further particulars are given, in which the following statements occur:—

Ravages later on

"As far back as 1869, the attention of administrative officers in Assam became directed to a peculiar disorder called *kála-ázar*, the of which decimated, and in some instances almost depopulated numerous districts in the Garo Hills.... The disease is most intense in the low, densely-wooded Garo Hills join on to the low-lying Central Assam plain, a position *par excellence* the most favourable for malarial developments.... The Garos give definite accounts of the invasion of their villages by this epidemic at periods varying from 3 to 30 years previously."

After describing the symptoms in detail he continues—

"Nearly all the preceding symptoms seem to depend on one other symptom not yet mentioned. That symptom is an intense anæmia, which shows itself at an early period, and continues to increase in intensity as the disease progresses. The above symptoms

of intense malarial poisoning gradually culminate in a fatal issue. This usually results from progressive asthenia in from six months to three years or more. Recovery is rare."

He then mentions that most of the cases were seen once only, and that further and continuous observations are necessary, and he concludes :

"At present more cannot be said than that from the cases recorded : the opinion has been arrived at that *kála-ázar* is a cachexia produced by malarial fever, deriving its peculiar characteristics from the nature of the region where it prevails, and having a singular tendency to run rapidly into the cachexial stage."

The above extracts show that the disease was considered to be a bad form of malarial fever, which had probably been present for many years, but which at certain times became epidemic, and almost depopulated districts. The absence of registration of births and deaths makes it impossible to fix the precise date of the first increased death-rate in the Garo Hills, and this accounts for the different number of years that it is said in the above report to have been present. The highest death-rate in the selected areas of registration, which was begun in 1882, was 41·66 per thousand in 1883.

In 1883, the death-rate increased in the portion of Goalpara which lies at the foot of the Garo Hills ; and Dr. Eteson, who had succeeded Dr. Clarke as Sanitary Commissioner of Assam, writes :

"It will simplify the measures of relief, and the medical treatment also, if it is accepted, once for all, that *kála-ázar* is a marsh ague in its causation, intensified through chronic lapses into a blood taint, which passes into anæmia and organic congestions ; and that it is only especially fatal, because the sick Garos are left uncared for, unnourished, and exposed to continued relapses."

Relief work was started in the affected districts this year under the superintendence of Dr. Dobson, who reported at length on the disease in 1884. His conclusions were—

"That *kála-ázar* is no new disease, but only a local name for

malarial fever and its consequences; that it is essentially chronic in its nature, originating in ordinary fever and continued by relapses; that its most noticeable and constant complication is hypertrophy of the spleen; that darkening of the skin is not at all common; and that there is not a particle of evidence that *kála-ázar* is contagious."

Dr. Eteson entirely supported these views. It is also worth noting that out of 4,919 cases, which were treated by the relief parties, 4,191 were recorded as for fever or enlarged spleen, two-thirds of them coming under the latter heading, and only one case was returned as anæmia; and that even at this early date it was noted that—

"The area of the epidemic is extending, counterbalanced by the fact that it is now absent where it had been severe."

In 1885-86, there is nothing of importance recorded, except the steady spread of the disease. In 1887, a decrease of the disease was noted in the terai portion of South Goalpara, which had been attacked in 1883. In this year also attention was first drawn to the anchylostoma as the cause of the so-called "*beri-beri*" or anæmia of coolies, which had long been prevalent on many of the tea gardens of, more especially, the upper parts of the Brahmaputra Valley. Referring to Dr. Kynsey's pamphlet on this disease as met with in Ceylon, Dr. Eteson writes:

"There seems little doubt but that the disease described in it and called *beri-beri*, is essentially the same as that commonly called anæmia and dropsy in Assam, often prevalent and very fatal in the Immigrant class. ... Hitherto it has been attributed to malarial cachexia, and associated with bad drainage, exposure, and climatic influences. But a careful *post-mortem* enquiry has demonstrated the presence of an intestinal parasite, the anchylostomum duodenale, in all those examined, and in numbers corresponding to the severity of the symptoms during life."

After stating that water is the probable vehicle of infection, he continues that Dr. Ruddock, of Meesa, Nowgong, "by the administration of thymol in thirty-grain doses, had almost invariably succeeded in discharging the dead parasite in hundreds, followed by a rapid restoration to health, the

natural colour of the blood, and re-established nutrition"; and he adds—

"It would be interesting to prosecute this discovery in the direction of *kála-ázar*, or the ague cake of Goalpara; but I fear that is purely malarious."

In 1888, Dr. Costello, who had succeeded Dr. Eteson, records that there had been a considerable decrease in the mortality from fevers in the Goalpara district; and, after describing the very malarious character of this and the Kamrup districts, he adds—

"There can be no cause for wonder that one type should be so pronounced and of so marked a character, as well as often fatal, as to have been thought to be a distinct disease—I mean *kála-ázar*, so common still in the Garo Hills tract and in the parts of Kamrup mentioned above. The opinions of the best Civil Surgeons of the province, as well as of my predecessor—an officer of great experience—was, and is, that this apparently peculiar form of fever is nothing more than a very aggravated, and often neglected, malarious fever, and also that its so-called distinctive features, such as anæmia, enlargement of the spleen and liver, with dropsy, diarrhœa, etc., are merely ordinary results of unusually severe and neglected malarious fever. No proof of its contagiousness has been given, and the same medicines, especially quinine, which are found of use in ordinary malarial fever, are equally useful in *kála-ázar*, as far as the extent to which the disease has reached, either from its original intensity or neglect, will allow."

He appends reports by Surgeon-Major Borah, recently Civil Surgeon of Kamrup, and by Dr. Gupta, of the Garo Hills, who both agreed that *kála-ázar* is only a bad form of malarial fever. He, however, concludes by stating that he has applied for a specialist to enquire into the subject. Thus we see that up to this date every medical man, who had had any experience of the disease, considered it to be an intense form of malaria, which was, however, not contagious. The whole difficulty was in explaining its great fatality, and more still the slow, but certain, way in which it spread year by year up the valley. The anchylostomum duodenale had also been

shown, chiefly by Dr. Ruddock, to be the cause of the disease known on tea gardens as anæmia of coolies, or *beri-beri.* At this period Dr. Giles was appointed to enquire "and report on the cause or causes of *kála-ázar* and *beri-beri* of Ceylon," and he began work at Gauhati at the end of November 1889.

A preliminary Note by Dr. Giles was published in May 1890 in the Assam Sanitary Report for 1889, and his final report in October of the same year. As his results were entirely opposed to the former views of the disease, it will be necessary to briefly give them here, as far as possible in his own words, in order to prevent any possibility of misrepresentation. Fortunately, this can be done in a comparatively small space, as he expresses them very clearly in both his preliminary and complete reports.

In his full report he describes his work in Gauhati as follows :

"There were five or six in-patients in the dispensary suffering from *kála-ázar*, as well as a daily casual attendance of out-patients suffering from the disease, and the first step was clearly to make a careful examination of the cases at my disposal. The greater number certainly presented more or less the prominent symptoms of malarial poisoning, but it was equally apparent that, in by far the larger proportion, the malarial symptoms were quite inadequate to account for the gravity of the mischief. On casually enquiring the history of a case, the patient would generally say that it had commenced with "fever," and he had it off and on for months. On a more and more close interrogation, however, so as to distinguish true ague from other maladies, it generally came out that there had been comparatively little true fever, and that what the patient really meant was merely that he had been feeling ill for a long time. There is, of course, nothing new in this, because everywhere in India, nearly all disease is ascribed by natives to "fever," and it is only by the most patient enquiries as to the exact symptoms actually experienced that one can get any other history for nine diseases out of ten. Then again, most of the cases had more or less enlargement of the spleen, many of them exhibiting a regular ague cake ; but when we remember

how rare it is in *post-mortem* examinations to find an Indian with a spleen of normal weight, it will readily be seen that too much importance should not be attached to a symptom which is well nigh universal, and is by no means incompatible with fair general health."

Later on, page 50 of his report, he quotes Dr. Dobson as having found 21 out of 42 children, in a village in the Goalpara district, to have enlarged spleens, although apparently healthy; and he continues—

"This accords with my own experience in the Chaygaon district, where I examined the spleens of a large number of people reputedly healthy. The proportion was about the same; but unfortunately I have lost the slip on which the notes were made. If then half the reputedly healthy population show more or less splenic enlargement, is it surprising that it should be found also in *kála-ázar?* The reverse would indeed be astonishing; and in the face of such a fact it is obviously absurd to attach any pathogromonic importance to the symptom in connection with the etiology of *kála-ázar*."

Returning to page 11 of his report, he writes:

"Now the cases in the dispensary, though in a terrible state of cachexia, gave no other evidence of malarial poisoning than a very variable amount of splenic enlargement. The temperature charts, far from forming the well-known malarial trace, exhibited, as their most marked character, a subnormal temperature, indicative of a profound depression of the vital forces. ... I have observed the temperature as low as 94° F., and this in cases by no means actually dying. A temperature of 95° rising to 96° in the afternoon was often persistent for several days together: such low temperatures are almost unknown, except in the case of profound traumatic shock or in articulo mortis, and are certainly unknown to persist for any length of time in any other disease than anchylostomiasis. This and the profound anæmia were the most marked characteristics of the disease with which I had to deal. Putting aside cases of ordinary sickness of all sorts, which were freely brought me as *kála-ázar*, this anæmia was the one constant symptom. As has been repeatedly noted by previous observers of *kála-ázar*, it is the earliest symptom to appear, and its intensity advances *pari passu* with the disease. Its characteristics easily distinguish it from the anæmia which accompanies malarial cachexia. In the latter, anæmia is a late and

secondary symptom, whereas here it appears from the first, and nearly all the other symptoms are merely its results. Then, again in the anæmia of malarial cachexia, specially when accompanied by enlarged spleen and consequent venous and portal obstruction, the conjunctiva, although deficient in blood, is nearly always of a dirty yellow tint, often accompanied by distinct icterus. The absolute dead white, rather bluish than yellow, coloration met with in the larger proportion of *kâla-ázar* cases, is never met with in uncomplicated malaria. There is something peculiarly pathogromonic about this appearance of the conjunctiva, which, once thoroughly appreciated, is not easily forgotten."

He continues by stating, that up to this time he had no idea what might be the cause of the symptoms observed, and, while waiting for a *post-mortem*, he "inoculated a considerable series of tubes of nutrient jelly with the blood of several patients. With the exception of a few tubes which developed accidental colonies of well-known mildews, etc., such as will occur in any series of such observations, these experiments gave entirely negative results," showing, together with the absence of the least sign of paralytic symptoms, that *kála-ázar* had no connection with the endemic palsy described by Pekelharing under the name of "*beri-beri*." He then continues :

"After about ten days, the death of one of the in-patients enabled me to make an autopsy. The examination showed that the immediate cause of death was an exacerbation of a chronic state of dysentery from which she had suffered during the whole time she had been under observation. The other changes were œdema and ascites, excessive anæmia of all the tissues, and an obviously thin and watery condition of the blood. In the duodenum and upper part of the jejunum were a number of anchylostomes. In this case then the primary cause of death was plainly enough anchylostomiasis, and this led me to examine the dejecta of my other patients, and in every instance enormous numbers of the ova of the parasite were found. A rough estimate was made of the numbers passed in a few cases by diluting a known weight of fæces, and counting the number of ova in a small weighed portion of the diluted material, with the result of showing that the number passed daily must often exceed

a million. Other *post-mortems* followed, and proved incontestibly that, whatever *kála-ázar* might be elsewhere, the disease so called in Gauhati was undoubtedly anchylostomiasis."

Having thus satisfied himself as to the nature of the disease in Gauhati, he made excursions to villages near and made examinations of the fæcal matter found near the houses, with the result that—

"By examining specimens taken at hap-hazard, it was often found that in badly stricken villages, three specimens out of every four would contain the ova of the parasite (anchylostomum). Further, it was found that the severity of the outbreak and the proportion of specimens showing ova, was generally proportionate."

He continues—

"After some stay in the *kála-ázar* districts, I proceeded to Upper Assam, and visited the tea-growing districts, where anchylostomiasis has been shown to be so prevalent under the name of *beri-beri*. Nothing here struck me so strongly as the absolute identity of the clinical pictures presented by these cases of acknowledged anchylostomiasis with those I had just been seeing so much of under the name of *kála-ázar*."

With regard to treatment, he writes on page 14 of his report:

"Nothing that has hitherto been tried has had any effect on *kála-ázar*, and even thymol is, as a rule, of no use, because cases seldom, if ever, come to us sufficiently early to be relieved of the parasites before they have inflicted fatal damage on the system."

And on page 104, he urges the importance of early diagnosis and treatment; and, after remarking that he had at first hoped that the expulsion of the parasites would be sufficient to initiate a cure to be completed by proper dieting, he continues:

"This *a priori* notion would doubtless have proved correct enough if the patients had retained the power of assimilation; but a further experience of the disease has demonstrated the fact that the mere loss of nutritive matter required for the support of the parasites, is a small and quite unimportant factor in the causation of the fatal symptoms produced by them. Various calculations have been made as to the amount of blood actually withdrawn by the

parasites, and these, though differing pretty widely, agree in making the amount too small to be likely to do more than seriously weaken an adult. The real damage is mainly caused by the destruction of the digestive powers. A large share in the causation of this diminished power of assimilation, is probably to be accounted for by the constant recurrence of great numbers of small traumatic lesions of the mucous membrane. Should these lesions include boring and temporary encystment, the matter is more easily understood; but the bites themselves, in spite of their small area, can account for much."

In the clinical section of his report, he gives abstracts from the notes of cases recorded by several of the Civil Surgeons of Assam, to each of which he appends his own opinion as to the correct diagnosis in view of his discoveries, and concludes with full notes of two cases seen by himself. One of these latter, and, as far as can be judged, the other also, are simple, uncomplicated cases of anchylostomiasis. Out of the thirty cases he quotes, twenty were from notes by Dr. Dobson, who, in the Assam Sanitary Report for 1891, very truly remarks:

"With these extracts of the cases reported by me, and which Dr. Giles has undertaken to diagnose, I find that such cases as are anæmic are invariably returned by him as anchylostomiasis, unless it is that the state of the conjunctivæ is said to be yellow, in which case it is malarial. Surely it is going too far to practically say it is only anchylostomiasis when there is anæmia. On reading over the diagnosis of these cases, as arrived at by Surgeon Giles, anæmia would seem to be a symptom hardly ever met with in any other disease."

Dr. Giles gives several other arguments in favour of his view of the disease, which must be mentioned. Thus, in Section 3 he gives figures to prove that the highest mortality occurs in the smallest villages, and that several members of one family are frequently attacked before other families in the same place become infected, which, together with the popular belief to the same effect, leads him to conclude that "the distribution of *kála-ázar* is that of a communicable disease." Again, in discussing the spread of the affection, he points out that isolated villages may escape in a remarkable manner,

and that it has taken seven years to spread less than 100 miles, while on page 29 he writes:

"It is a noticeable fact that once the disease has made its appearance in a district, it never leaves it, the weekly returns of the prevalence of the disease showing it to be present to-day in every part of the country where it has been hitherto reported."

This was true of whole districts, such as the Garo Hills or Goalpara at the time it was written, but Dr. Giles seems to have overlooked the remarks quoted from some of the Assam sanitary reports earlier in this section, to the effect that the disease had died out of certain localities which had previously severely suffered from it. He also makes a strong point of the fact that no Europeans had suffered from the disease, and remarks that, although their numbers are small, yet "their escape would be indeed remarkable if malarial influences were abroad sufficient to account for the terrible ravages of *kála-ázar*;" while, on the other hand, they would not be at all likely to suffer from such a disease as anchylostomiasis, because "it is only among such Europeans whose habits assimilate them to the semi-civilised inhabitants of Assam that anchylostomiasis has ever spread." With regard to its being malarial, he writes:

"Again, if *kála-ázar* be but malarial cachexia intensified by the proximity of uncleared jungle, the habits of the people, and so forth, it is clearly incumbent on the advocates of this theory to show that these conditions and habits have been intensified in affected villages coincidently with the outbreak of the disease; but, in point of fact, no one pretends that any such change has taken place."

A very clear summary of these views is given on pages 65 and 66 of his report, which must be quoted:

"In the absence, then, of any intelligible cause for intensification of the well-known malariousness of the affected districts, it seems as great a mistake to attach any importance, in the production of the increased mortality, to such cases of malarial cachexia as one meets with, as to the cases of dysentery, pneumonia, and spinal caries. Personally I have not found cases of uncomplicated malarial cachexia at all commoner than I have in certain parts, for example, of the

Central Provinces and Punjab. What I have, however, found is that, whenever a village is seriously affected with *kála-ázar*, the larger proportion of serious illness will be found to consist of anchylostomiasis. That a very large proportion of the cases will be found to be complicated with enlargement of the spleen is quite beside the question, for, as we have seen, this condition nearly as often complicates apparent health. Malaria doubtless accounts for as much sickness and death as it always has, and that, we know well, is no small amount; but it is entirely inadequate to serve as the efficient cause of the terrible death-rate of *kála-ázar*-striken villages, and the true cause of this will be found to be neither more nor less than anchylostomiasis. Of course, it is perfectly intelligible that a man brought low by anchylostomiasis should fall a ready victim to malarial influences. In the closely analogous case of famine we know this to be the case, but it is to the shortness of food, and not to malaria, that we ascribe the high death-rate of periods of famine, in spite of the fact that many poor, half-starved wretches might have survived but for attacks of fever and enlargement of the spleen. Conversely, a man already the subject of malarial cachexia, will die much more rapidly from anchylostomiasis than an originally healthy subject.

"As will be seen, I am far from asserting that any and every case, that will be produced as *kála-ázar*, is necessarily anchylostomiasis, or that cases of malarial cachexia are otherwise than very common, for such cases are very common now, always have been, and it will be a long time, ere they cease to be so. All I wish to convey is that the increased mortality is due to the anchylostomiasis, and to no other cause, and hence the answer to the question propounded at the commencement of this section, must be that if we take *kála-ázar* to be anything brought as such, *kála-ázar* may be anything; but if we confine ourselves to the cause of the present pestilence, the reply is that it is anchylostomiasis."

Again he writes in his preliminary report:

"It will be readily seen how thoroughly this explanation solves all the difficulties that have hitherto surrounded this obscure disease. Using the term in its broad sense of communicability, there is no disease that can fairly be said to be more infectious than anchylostomiasis, always assuming the absence of proper measures of conservancy and the co-existence of suitable climatic conditions. That these conditions are most efficiently present in Assam is already

abundantly shown by the terrible mortality which, under the name of *beri-beri*, the same disease has caused amongst the labourers on the tea gardens."

I have, I think, quoted enough to show that granting the correctness of the statements on which Dr. Giles' views are based, the fact that he found anæmia to be the one constant feature of the disease, while fever and enlargement of the spleen were accidental complications, and finding the anchylostoma constantly present, which had been proved to cause anæmia in every part of the world where they had been found, he could not have come to any other conclusion than that recorded. He spent the rest of the year, over which his investigation lasted, in a study of the life history of the anchylostomum, which resulted in his coming to the conclusion that the disease is spread much more by earth and dust getting into food, etc., than by water, and consequently recommending conservancy improvements as the main way to combat the spread of the disease.

The reception of Dr. Giles' report, and the subsequent researches of the Civil Medical Officers of the province, which have led to the re-investigation of the subject, only remain to be considered.

In the report for 1890, Dr. Costello writes:

"My conclusion now is that Dr. Giles' views as to the cause of *kála-ázar* are quite true, it and *beri-beri* being due to anchylostomiasis, the apparent difference being due to *kála-ázar* being frequently complicated by malarious fever and its results, as in the districts of the Lower Assam Valley, the inhabitants are much more subject to malarious causes than those in the districts in the northern section of that valley."

He recommended a trial of the conservancy measures advised by Dr. Giles, and also directed systematic clinical and microscopic observations to be made in the affected districts for one year. As a result of this, two very valuable reports by Drs. Dobson and Campbell appeared in the 1891 Sanitary Report, a summary of which will be given here. Dr. Neil

Campbell gives notes of 88 cases, which he classes in three divisions, thus—(1) Malarial cachexia, 7 cases, in which no ova were found in the stools, or no anchylostoma after the administration of thymol. (2) Anchylostomiasis, 15 cases. (3) Anchylostomiasis *plus* malaria, 65 cases, which he subdivides into—(1) where the malarial complication is slight, 15 cases, and (2) where it is of a serious nature, 50 cases. The anchylostomi were noted either as simply being found in about two-thirds of the cases, or as having been present in "numbers" in about one-third of them, while in two they were found in large numbers. The finding of ova in the stools, or of any of the parasites after the use of thymol, is taken as a proof of the presence of anchylostomiasis. He sums up his results as follows :

"I believe that in *kála-ázar* the starting or exciting cause is infection by dochmius duodenalis, hence the number attacked and the rapidity with which it spreads ; that it spreads in various ways by air getting into food, etc., but also by contamination of water, though Dr. Giles denies the latter. After the patient has become infected with the dochmias, he, as parasites are added by fresh infections, developes a lower vitality, and is liable to other diseases; and as Goalpara and Kamrup are notoriously malarial districts, the anchylostomiasis is complicated by malarial cachexia. Hence, I believe, *kála-ázar*, as so called by the natives of Lower Assam, is in 75 per cent. anchylostomiasis *plus* malarial cachexia, and in the remaining 25 per cent. either anchylostomiasis, pure and simple (in 10 to 15 per cent.), or pure malarial cachexia (in 5 to 10 per cent.)."

Moreover, in discussing the purely malarial class, he says :

"This division might be omitted, as it is not really *kála-ázar*, but for the fact that the patients, their friends, and their native doctors call it so."

It will be seen that his view is substantially a strong confirmation of the results arrived at by Dr. Giles, and if it be granted that the presence of the anchylostoma or their ova is evidence of anchylostomiasis, that is, of a diseased condition brought about by this parasite, the opinion expressed is a perfectly logical one. In the report of 1894,

however, that is, after the publication of Dr. Dobson's discoveries, to be immediately described, Dr. Campbell writes :

"I believe it to be a severe form of paludal fever, complicated in 70 to 80 per cent. of the cases with anchylo-tomiasis."

Coming to Dr. Dobson's work, it may be summed up in a few words. In the course of some four years he examined over 1,000 persons for the presence of anchylostoma, and in all, except those examined in his first year's work, 1891, he has recorded the exact number of the various intestinal parasites passed after the administration of thymol, with the exception of thread worms. I find an analysis of his tables shows that out of 797 healthy people, no less than 620, or 77·78 per cent., harboured anchylostoma in numbers ranging from 1 to 230. Of the total number, 651 were coolies imported from all parts of India, and 80·18 per cent. of them carried this parasite into Assam with them; while 146 were healthy men in some of the jails of Assam (and so the great majority of them were either inhabitants of Assam, or had been resident in this province for some time), and 67·12 per cent. had anchylostoma in them. These figures point to the conclusion that the worm in question is more commonly present in natives of Bengal, Chota Nagpur, the Central and North-Western Provinces, and other parts from which coolies are imported, than it is in those of Assam. In 116 cases of *kála-ázar*, Dr. Dobson found the worms in exactly 75 per cent., and in 212 cases of other illnesses he found them in 73·20 per cent. Moreover, taking the cases in which the exact numbers present are stated, they average much the same in the *kála-ázar* cases as they do in the healthy natives. It will be at once seen that these observations are of the utmost importance, as, in the absence of any figures to show in what numbers the anchylostoma were found to be present in the cases of *kála-ázar* investigated by Dr. Giles, they throw great doubt on the correctness of the view taken by that observer, that this parasite is the cause of *kála-*

ázar. In fact, it is not too much to say that it is due mainly to these observations, that the question has been re-opened.

The gradual changes of the views of the medical men in Assam with regard to *kála-ázar* during the five years that elapsed between the publication of Dr. Giles' report and the determination to have the question re-investigated, can be best illustrated from the remarks of the successive Sanitary Commissioners in the Assam reports. Thus, in 1891 report Dr. Costello writes :

" After the most careful enquiry in each of the affected districts of the Garo Hills, Goalpara, Kamrup, and Nowgong, where I visited, with the Civil Surgeons, all the notoriously affected centres, my opinion now is, as regards *kála-ázar*, that Dr. Giles discovered and hit the true cause which makes that disease contagious or carriageable from place to place and from person to person by infected persons disseminating the embryoes of the dochmius in the soil through their fæces, and which is also the main factor of its remarkable fatality, *i.e.*, that he was the first person to discover and prove that the dochmius or anchylostomum parasite was an important and distinctive factor in this disease, and that which completely distinguished it from ordinary "malarious" disease.... However, in comparing *kála-ázar* with anæmia or *beri-beri* of Ceylon, and in deciding that they were, in all their main points, essentially the same, Dr. Giles, in my opinion and in those of the officers most competent to judge, (namely, the Civil Surgeons who had seen most of the disease) overlooked the very important malarious element in *kála-ázar*. It is our opinion that the difference between the two diseases is altogether and only the result of the effects of intense malarial poisoning, arising from the topographical peculiarities of the localities made notorious by *kála-ázar*, which do not exist in anything like the same intensity in Upper Assam, where the anæmia or *beri-beri* form of anchylostomiasis has been known for years to be common, and where such remarkable and deadly terais, etc., as exist in Lower Assam, are rare, or almost absent."

A little further on he writes :

" From Dr. Dobson's report it is clearly proved that *kála-ázar* is not mere anæmia of coolies or *beri-beri* of Ceylon, but that, on the contrary, malaria plays a chief part in this disease, and so complicates it as to render it much more fatal and serious."

In the 1892 report the same officer, after recording that the mortality from *kála-ázar* had much decreased in the Goalpara and Kamrup districts, while it had increased in the Nowgong and Mangaldai subdivisions, writes:

"As regards *kála-ázar*, the further observations, as far as they have gone, go towards throwing doubt on Dr. Giles' conclusions, and seem rather to point, as was previously supposed, towards malaria in some form being the main factor in causing that disease. In fact, no satisfactory conclusion as regards its cause has, in my present opinion drawn from the latest evidence at my disposal, been arrived at, and, except, that Dr. Giles wrote a most interesting life history of the parasite, and also confirmed our previous views as to *beri-beri* of Ceylon (anæmia of coolies) being a form of anchylostomiasis, I cannot now, with the further light thrown on the subject, see that we have as yet discovered the undoubted cause of this disease."

He, therefore, applied for the services of a fully competent pathologist and bacteriologist to re-investigate the subject. The results of some enquiries from tea garden doctors on the subject of anæmia of coolies, are also given in this report, which show that they unanimously attribute the disease known as anæmia of coolies to anchylostomiasis; but most of them look on such predisposing causes as malaria, bad feeding, and other causes of debility as being necessary factors in its production, in addition to the presence of the parasite, and regard it as being very uncommon among the indigenous inhabitants of Assam, who have not worked on tea gardens.

In the 1893 report, Dr. Wiburton, who had succeeded Dr. Costello, after reviewing the symptoms of the disease, writes:

"It will be seen that many of the symptoms that I have named are also characteristic signs of malarial poisoning, while others are not inconsistent with it;" and again "its unequal incidence where sanitary conditions appear equally bad, the gradual and steady progress downhill unaffected by treatment, the statement that it is slowly, but steadily, extending up the valley, seem to point to something more than malarial cachexia, and that something is, in my opinion not the dochmius duodenalis. I have seen cases in Northern

India with symptoms so like those seen in cases of *kála-ázar* in Nowgong, that they would most certainly have been returned as such, had they occurred there, and there is at present in the Golaghat dispensary a case entered as splenitis, which, if seen at Nowgong, would be described as a typical case of *kála-ázar*. Yet this man, an ex-garden coolie from the neighbourhood, has never been in the Nowgong district. Looking at all the circumstances connected with the so-called *kála-ázar* epidemic, I am convinced that, if malarial conditions could be completely removed, *kála-ázar* would cease to exist."

He, therefore, recommends improved sanitation, rather than a re-investigation of the disease, as a better way of spending whatever money might be available.

In the report for 1894, Dr. Stephen, who had succeeded Dr. Warburton as Sanitary Commissioner of Assam, gives a very clear summary of the reasons for and against *kála-ázar* being a form of malarial fever. In the former list he gives—(1) the symptoms in the first instance cannot be distinguished from those of malarial fever; (2) the disease never makes its appearance except in very malarious places; (3) except that the liver is much more enlarged in *kála-ázar*, to the naked eye pathological changes are the same as in chronic malarial; (4) persons suffering from *kála-ázar* and chronic malarial fevers, are frequently carried off by the same secondary diseases, *viz.*, dysentery and diarrhœa, and, in the cold-weather months, pneumonia. On the other hand, the main differences are:—(1) the distribution and way in which the disease has spread, is very different from that of malarial fevers, and (2) several members of one house, or part of a village, may die of the disease before other families or houses suffer, while malaria will equally attack all parts of a village which are in the same sanitary condition; (3) the liver is more enlarged in *kála-ázar*; (4) quinine and arsenic seem to have no effect in advanced cases of *kála-ázar*, but he also remarks that quinine is not so useful in cases of chronic malarial fevers as it is in acute cases of quotidian fever.

He gives the following summary of the opinions of the various Civil Surgeons who had seen the disease:

"Surgeon-Major Borah is of the opinion that *kála-ázar* is something more than malarial cachexia. Surgeon-Major Mullane believes that the disease is nothing more than an aggravated form of malarial fever. Surgeon-Major Campbell is under the impression that the disease is malarial fever complicated in 75 to 80 per cent. of the cases with anchylostomiasis. He, however, is not quite certain if some other cause does not come into play, as later on in his letter he states that, were a bacteriologist appointed to investigate the disease, he might discover it to be due to a micro-organism, differing from the plasmodium malaria. ... Mr. McNaught believes that it is a specific disease distinct from malarial fever. The medical officers of tea gardens, who met at Kokilamukh on the 30th October 1894, after examining persons suffering from *kála-ázar* brought before them by Drs. Price and Lavertine of Nowgong, came to the conclusion that the disease was distinct from malarial fever, as they remark that it appears to be entirely unknown in Upper Assam. All medical officers whom I have consulted, with the exception of Surgeon-Major Campbell, are of the opinion that the disease is in no way dependent on anchylostomiasis."

The views of Dr. Macnamara only remain to be recorded as set forth very succinctly in an appendix to this report. He writes:

"There can be no doubt that we have some disease in Assam which appears to be going up the valley of the Brahmaputra. ... There seems to be no record of similar outbreaks in Assam in former times, but I have known epidemics of what was believed to be malarial fever in the Surma Valley. I confess my inability to distinguish *kála-ázar* from malarial fever by any physical sign or subjective symptom apart from a history of residence and the downward tendency of its course. I am satisfied the dochmius duodenalis is not the cause of *kála-ázar*, because I found abundance of the parasites where there was no *kála-ázar*, and, conversely, *kála-ázar* in cases where no parasites were present. I have no evidence that *kála-ázar* is contagious. It does, however, appear to spread as diseases, which are known loosely as contagious, spread."

All the Civil Surgeons above mentioned agreed in recom-

mending that the services of a specialist should be obtained, who would be able to devote his whole time to investigating the disease, as they had no time for systematic observations of a scientific kind. Surgeon-Colonel Stephen gives it as his opinion:

"From what I have seen of the disease, and from what I have been told of it by those who have had the opportunity of studying it, I am under the impression that, though allied to malarial fever, it is probably a separate disease, and as none of the remedies used seem to have had any effect in stopping its ravages, and as it is steadily spreading eastwards up the Assam Valley on both sides of the river, I would recommend that a specialist be appointed to enquire into its cause."

And he suggests either Dr. Hankin, the North-West Provinces Bacteriologist, or Dr. Haffkine, who was at this time in Assam carrying out his inoculations against cholera, as suitable a man for the work.

In the report for 1895, there is nothing new recorded, except that the disease was beginning to die out of the Nowgong district.

Space does not permit of any reference to the views of those who have written on the subject without any practical acquaintance with the disease; but enough has been said to show that, until the time of Dr. Giles' investigation, everyone who had studied the disease believed it to be malarial in its nature, although no one could satisfactorily explain its steady spread up the valley. Dr. Giles' theory furnished an explanation of this difficulty, and was accepted, with the reservation that he had under-estimated the malarial element, until Dr. Dobson's laborious researches threw doubt upon it, since which time the pendulum of the united professional opinion of the province has swung back again to the belief, that the essential element of the disease is malarial, while the old difficulty of its epidemic nature leads some to think that, either there is some other factor in it besides malaria which accounts for this feature, or it is an entirely different and new disease.

SECTION II.

THE NATURE OF THE PROBLEM, PLAN OF WORK, AND COURSE OF THE INVESTIGATION.

THE NATURE OF THE PROBLEM.

From the history of the epidemic, as recorded in the last section, it will be seen that the problem at the time of the re-opening of the question, may be stated thus. The disease might be—

(1) Anchylostomiasis, pure and simple, or, in the words of Dr. Giles, "The increased mortality is due to anchylostomiasis, and to no other cause," although he also writes—"The disease is due to anchylostomiasis acting in people already worn down by malaria," and says that the only difference between the disease as met with in the *kála ázar* districts in the lower part of the Assam valley, and anchylostomiasis as seen on tea gardens in the upper districts, is that in the former malarial complications are much more common.

(2) Malarial fever of a very intense form spreading in a wave of increased mortality up the valley. This view necessitates the admission that malaria may, under exceptional circumstances, become communicable, which has always appeared to be an insuperable objection to it.

(3) That it is due to a combination of both anchylostomiasis and malaria acting in varying proportions in different cases, the former factor accounting for the spread, and the latter for the much greater mortality than is usually the case in simple anchylostomiasis. This view seems to be that which is most commonly held outside Assam by nearly all who have written on the subject without any close personal acquaintance with the disease. On the other hand, it must be admitted that of those who have had much experience of

the disease, only one, namely, Dr. Campbell, inclines to this view, and even he now looks on malaria as the essential part of the disease, and states that it is complicated by anchylostomiasis in about 75 per cent. of the cases.

(4) That it is a new and distinct disease. The only advocate of this view that I know of, is Mr. McNaught, of Nowgong, and it must be admitted that he has had exceptional opportunities of seeing the disease. He was, however, when in the Garo Hills, an equally strong supporter of the malarial view of the affection, but the steady spread of the disease has caused him to alter his opinions.

PLAN OF WORK.

Having handed over medical charge of the 11th Bengal Infantry on April 3rd, 1896, work was commenced in Calcutta on the 6th by a study of the literature of *kála-ázar* and allied diseases. Leaving Calcutta on the 16th, two days were spent at Dhubri, where Dr. Dobson kindly gave me an opportunity of seeing some coolies examined for anchylostoma. Brief visits were paid to Gauhati, Shillong, and Tezpur, and my headquarters were reached on April 27th at Nowgong. During these visits, it became apparent that one of the main causes of the absence of any very precise knowledge of the disease lay in the fact that, owing to its chronic nature, Civil Surgeons are unable to follow cases from the beginning to the end of the attacks, and in fact rarely see them until they have arrived at a late stage, and then only too often they remain but a few days in hospital. In order to try and settle the vexed question of the true nature of *kála ázar*, the following plan of work was decided on, and only slight additions to, and modifications of, it have been found necessary.

(1) A careful clinical study of the disease, the cases being followed up as long as possible, especial care being taken to determine the frequency of the presence of anchy-

lostoma and the number found in each case, for in view of Dr. Dobson's work (Section I), it is obvious that these worms must be present in the great majority of *kála-ázar* cases in considerably larger numbers than they are to be found in the ordinary healthy people of Assam, if they are to be considered an important factor in the causation of the disease.

(2) An examination of a number of healthy Assamese for anchylostoma by the thymol method, controlled by a quantitative examination of the blood, by which means the effect of such numbers as might be found on the composition of the blood of apparently healthy people, could be ascertained. These observations were carried out in precisely the same way as those of Dr. Dobson, in order that they might be strictly comparable with his. The amount of fever from which each man had suffered within the last two years, was also noted to see what influence this had on the production of anæmia. It was also expected that an indication might be obtained as to whether the inhabitants of Assam had larger or smaller numbers of anchylostoma than the imported coolies had been shown to have by Dr. Dobson.

(3) An examination of the blood in both *kála-ázar* and anchylostomiasis cases, in order to find out if the anæmia, which is such a marked feature of each disease, is of the same nature or not ; for it appeared to me to be very probable that the changes brought about by the action of the anchylostoma, would differ materially from those produced by a persistent and relapsing form of fever, such as *kála-ázar* has always been described as being by all observers, with the solitary exception of Dr. Giles.

(4) These observations to be controlled by a similar examination of the blood in cases of both anchylostomiasis and chronic malaria as met with in districts where *kála-ázar* was not present ; and it may be said here that this was always looked on as the most hopeful method of solving the complicated problem before me.

(5) Through the kindness of Dr. Waddell, Chemical Examiner, Medical College, Calcutta, arrangements were made for an analysis of the amount of iron in the liver after death in some cases of *kála-ázar*, as it has been recently shown that this is much reduced in cases of anchylostomiasis, while it is increased in chronic malaria.

(6) A bacteriological examination of the blood was also necessary, and Professor D. D. Cunningham very kindly promised to supply me with culture tubes, and to examine any organisms which might be obtained, as for this purpose a fully equipped laboratory is required. A search for the plasmodium malariæ, and, if found, a study of its forms in order to determine, if it differed from that met with in ordinary malarial fevers, in some such way for instance, as the form met with in the summer-autumn fevers of Italian authors does, constituted a very important and hopeful part of this section of the investigation.

(7) The performance of as many *post-mortems* as possible on both *kála-ázar* and anchylostomiasis cases, with control observations on medico-legal ones, and with the complimentary microscopical examination of sections of the various organs; for, although Dr. Giles tells us in his report that he spent over two months on this part of the work, he unfortunately appears to have only examined his sections for bacteria, and makes no mention of any pathological changes being found, except in the intestines.

In this part of the work especial attention to be paid to the presence of pigmentation of the organs, as this has been shown by two French observers to be constantly met with in chronic malaria, and is indeed a much more easily demonstrated and universally accepted sign of the presence of malaria in chronic fever cases than the presence of the plasmodium malaria itself, owing to the great changes in the blood rendering it unusually difficult to demonstrate the presence of the organism in such cases.

In these various ways it was expected that it might be determined if the disease was either anchylostomiasis or malaria, or a combination of the two; and in the event of neither of these alternatives proving to be in accordance with the facts met with, the ground would be cleared for striking out on a new line of work. If, on the other hand, any of these explanations sufficed, then it would only be necessary to study the exact present limitations and manner of spread of the disease, in order to see if anything could be done to check it, and the origin of the epidemic would also have to be investigated in the light of any new facts that might be brought to light.

Arrangements were also made for observations of the variations in the distance of the water from the surface of the ground to be recorded in the wells of the jails of the different districts, and similar observations were made in such other places as were visited from time to time, in order to determine if the distribution of the disease had any relationship to the depth and variations of the ground water-level, and if the places that suffered most severely were water-logged. The results obtained will be found in Section VI.

THE GENERAL COURSE OF THE INVESTIGATION.

The progress of the investigation will very briefly be described here, so that the way in which the facts on which the following description of the disease is based, have been accumulated, and the gradual steps by which I have been led to my final conclusions as to its nature, may be apparent.

Active work was commenced at the beginning of May 1896 by an examination of all the cases in the Nowgong dispensary and jail, minute notes in shorthand, and a full examination of the blood being made in each case. A month was spent in this way, including a good many *post-mortems*, in which respect I was particularly fortunate. As many as possible of these cases were kept under observation, and followed up in the intervals of my tours when I returned to Nowgong, for

many months, a matter of great importance, and one, the neglect of which was responsible for much of Dr. Giles' misfortune. The main impressions I derived from this first month's work, were the constancy of marked fever and signs of chronic malaria, and the difficulty of finding anything like a typical case of anchylostomiasis in other than ex-tea garden coolies. These cases were nearly all in an advanced stage of the disease, only such being usually seen in a civil hospital, so I determined to visit some of the affected tea gardens, where I hoped to be able to find early cases, and to follow them up by returning to visit them at intervals. I received most ready assistance from Drs. Price and Lavertine, who hold medical charge of the tea gardens of this district, the former in particular having had a very large experience of the disease, which he freely gave me the benefit of. A great difficulty, however, arose, for, although all were agreed that *kála-ázar* began with fever, which continued on and off until the spleen and liver became enlarged, yet no one even pretended to be able to diagnose the fever of *kála-ázar* from that due to simple malaria in the early stages, and only designated cases as *kála-ázar* when fever had persisted for a long time, and symptoms of chronic malaria had become developed.

I therefore decided to examine a series of cases of early fever, of such a nature that the patients could not be said to be suffering from *kála-ázar*, but some of whom had lost relatives of the disease, and were therefore likely to be early cases of it. Notes and blood examinations were made as before, and temperatures taken four times a day. About a month or six weeks later, I went around again and re-examined all the cases. Some of them had recovered, and had apparently only suffered from ordinary malarial fever, while others had continued to be affected by it, and now presented the typical appearance of *kála-ázar*. On comparing the notes and the blood examinations, including the search for the plasmodium malaria, no difference could be found in the early stage

between those who had become *kála-ázar* cases and those who had ordinary malarial fever, nor could any difference be found in the sketches of the plasmodia, which had been found alike in both classes of cases, nor did these differ in any way from those which I had found as a result of six months' continuous work at Doranda in Chota Nagpur in one hundred cases of quotidian and tertian ague. This was very disappointing, as I had hoped by these means to be able to differentiate the early stages of *kála-ázar* from simple malaria, a point of the first importance in view of the general opinion, that the former disease is in some way or other infectious, for if they could be isolated in the early stage, the disease might be prevented from spreading on a tea garden or elsewhere. On the other hand, I recognised that my failure in this respect was in accordance with the experience of everyone else who had studied the disease, with the exception once more of Dr. Giles, who unfortunately began work at the end of November, at which time of the year chronic cases of *kála-ázar*, such as he must have met with in the Gauhati dispensary, often lose their fever for even months at a time; and as he admits that the longest time he had any one case under observation, was one month, while the great majority of his cases he probably only saw once, it is not surprising that he denied that fever is a marked symptom of the disease, especially as he attributed any history of it which he acknowledges, he usually obtained, to meaning simple, that the patient had been feeling ill. It must then be admitted that the fever of *kála-ázar* is in its earlier stages indistinguishable from ordinary malarial fever.

At the same time, by means of re-examining the blood of cases at intervals of from two to four weeks, I found that the anæmia was in direct proportion to the amount, and more especially to the height of the fever; and that if fever was absent for a few weeks, the blood immediately improved, only to fall again if the fever returned. It was then evident that

the fever was the direct cause of the anæmia. Moreover, when I visited the tea gardens in June, I was able to get sufficient cases of pure anchylostomiasis to study the blood changes, and to compare them with those found in *kála-ázar*. The result exceeded my most sanguine expectations, for it became apparent that the anæmia of the two diseases was absolutely distinct, so that given a drop of blood from a typical case, I could, without fail, tell whether it was from a case of *kála-ázar* or from one of anchylostomiasis, without seeing the patient at all. Moreover, in the very small percentage of *kála-ázar* cases, which were complicated by anchylostomiasis (by which term I mean disease produced by anchylostoma, and not merely the presence of the parasites in small numbers), the blood changes were intermediate between those of the two primary conditions. I also found that cases did occasionally occur in which the two diseases so closely resembled each other, that they could not be distinguished clinically, but could be immediately diagnosed correctly by the blood examination. Further, my experience shows that anchylostoma are not present in quite as large numbers in cases of *kála-ázar* as they are in the healthy people of this district, the small difference being doubtless owing to some of the former having been given thymol. I have also found that the numbers in which they are present in *kála-ázar*, are too small to have any appreciable effect on the blood, for in the healthy jail-people larger numbers were present without the slightest alteration of the blood; but that those of the jail population who had had more than one week's fever in the last two years, had distinctly poorer blood than those who had had no fever in the same time. It is evident then that *kála-ázar* is quite distinct from anchylostomiasis, and that only in a very small proportion of the cases is it complicated by the latter disease.

It now became necessary to take in hand the section-cutting, and as ice could not be obtained in Nowgong, and it was

PLATE I

GROUP OF ORDINARY CHUCHU MALAKA IN SYLHET

much too hot to freeze with an ether spray, an adjournment was made to Shillong, where the greater part of October was spent with this and other work, with the result of showing that marked pigmentation of the liver, spleen, and kidneys, was a constant pathological feature of the disease, and was not found in cases of anchylostomiasis, or in persons who had died of snake-bite or from accidents, provided no malarial complications were present. These observations then furnished absolute proof of the malarious nature of the disease, and it now remained to be determined if the disease could be differentiated from ordinary chronic malaria.

For this purpose it was necessary to examine cases of chronic malaria in some place where there was no *kála-ázar*, for they certainly cannot be clinically distinguished in such a place as Nowgong at the present time. Sylhet, which had never been affected by the epidemic, was therefore visited, and some cases of advanced chronic malaria were kindly obtained for me by Dr. Banerjee. As will be seen by comparing the photograph opposite this page with those opposite pages 33 and 57, they were clinically the same as *kála-ázar*, and, in a district affected by the latter disease, would inevitably be returned as such. Nor did the blood changes show any points by which they might be distinguished. The symptoms were also identical, yet there was this very marked difference between the two classes of cases, for on enquiring into the history of them, it was found that, roughly speaking, they had suffered from fever on and off for about as many years before they developed this extreme form of malarial cachexia, as *kála-ázar* cases take months to reach the same stage, and moreover they were widely scattered in their distribution, it having taken two or three days to collect six cases, and no two cases occurred in the same family; while, on the other hand, in the case of *kála-ázar*, the course of the disease is very rapid, and the fever very intense, and it has a very great tendency to run in families, and even to kill out whole

villages, so much so that I know of one educated native of Nowgong, who has lost 39 relatives in six years, and has only four near ones now living. In short, the Sylhet cases are ordinary endemic cases of chronic malaria, such as are to be met with in any highly malarious district, while *kála-ázar* is equally malarial in its nature, but it is of a peculiarly intense form, which has the power of spreading slowly through districts, and killing off the majority of the inhabitants of villages, and more especially of families, to such an extent that it must be in some way communicable: in a word it is epidemic.

It now became necessary to study the origin, spread, and present limitations of the disease. For this purpose the Garo Hills were first visited, and an examination of the records, as far back as 1870, yielded much interesting information as to the exact time and places where the disease first appeared in that district, and afforded a clue as to real origin of the disease, which was subsequently successfully followed up. Tours in the Nowgong, Mangaldai, Tezpur, and Golaghat divisions, were then carried out, and evidence as to the mode of spread and limits of the disease was obtained. These tours, together with the writing of this report, occupied the whole of the cold-weather months. The results of this latter part of the work will be found recorded in Sections VI—VIII.

SECTION III.

CLINICAL DESCRIPTION OF *KÂLA-ÂZAR*.

Although *kála-ázar* has been briefly described by many writers, yet doubtless owing to the difficulty of following up cases and the impossibility of diagnosing them from ordinary malarial fever in their early stages, I have not been able to find any complete clinical account of the disease, such important points as the cardiac and retinal changes being scarcely mentioned. The following attempt to supply this deficiency is based on short-hand notes of nearly 100 cases, a great many of which have been repeatedly examined and followed up for months, and also on details of the exact admissions and dates of the deaths or recoveries of 200 cases, which occurred on a tea estate in the Nowgong district, which were taken direct from the hospital books by the kind permission of Dr. Dodds Price, who had seen all the cases during life, and could, therefore, vouch for the substantial accuracy of the diagnosis, and who has given me invaluable assistance in this and other work on the gardens under his medical care. This laborious part of the research has presented opportunities as unique as they were invaluable in solving several of the problems connected with the disease.

Kála-ázar may be defined as being a chronic and relapsing form of fever of an intermittent or irregularly remittent type, which is very resistent to treatment, and produces progressive anæmia, great wasting, and in many cases dropsy, and terminates either in a final attack of fever or in asthenia, often accompanied by diarrhœa or lung complications; it attacks usually several members of a household, and spreads slowly through districts in a wave of increased fever mortality, and after about six years dies out again.

The diagnosis of the cases is obviously of the first importance, as if it is incorrect, the results based on it will also be wrong. Unfortunately, it is quite impossible to tell early cases

of *kála-ázar* from ordinary malarial fever, and I find that it is not until fever has persisted steadily on and off for some months, and signs of cachexia, such as anæmia, enlarged spleen and liver, etc., have supervened, that cases are called *kála-ázar*. If, in addition, there is a history of other cases in the family, or in the house in which the patient has lived, then the diagnosis may be made with confidence. Doubtless, cases of ordinary chronic malaria and of anchylostomiasis and other forms of anæmia, which happen to be complicated by enlarged spleen, are not unfrequently returned as *kála-ázar*, and I have met with such cases which could not be diagnosed correctly by ordinary clinical methods, even after I had had considerable experience of the diseases. Fortunately, a method has been found of differentiating anchylostomiasis even when complicated by enlarged spleen from *kála-ázar*, namely, by the blood changes, which will be described in detail in the next section, and it will suffice to say here that no doubtful case has been included in the series on which the following clinical description is based, that has not been submitted to this test, with the exception of those on the tea garden, which have only been used to get some idea of the duration and seasonal distribution of the disease, and the number of which, all of whom have been under the care of Dr. Price, is sufficient to do away with all chance of appreciable error in the limited deductions drawn from them.

The distribution of the disease.

The spread of the disease will be described in detail as far as it can now be traced in Section VI, and only the incidence in families will here be dwelt upon. It has already been mentioned that the disease has a marked tendency to affect several members of the same family, and, as was pointed out by Dr. Giles, several members of one household are often attacked before any other family of the same village suffers, although they are all living under the same meteorological and sanitary conditions.

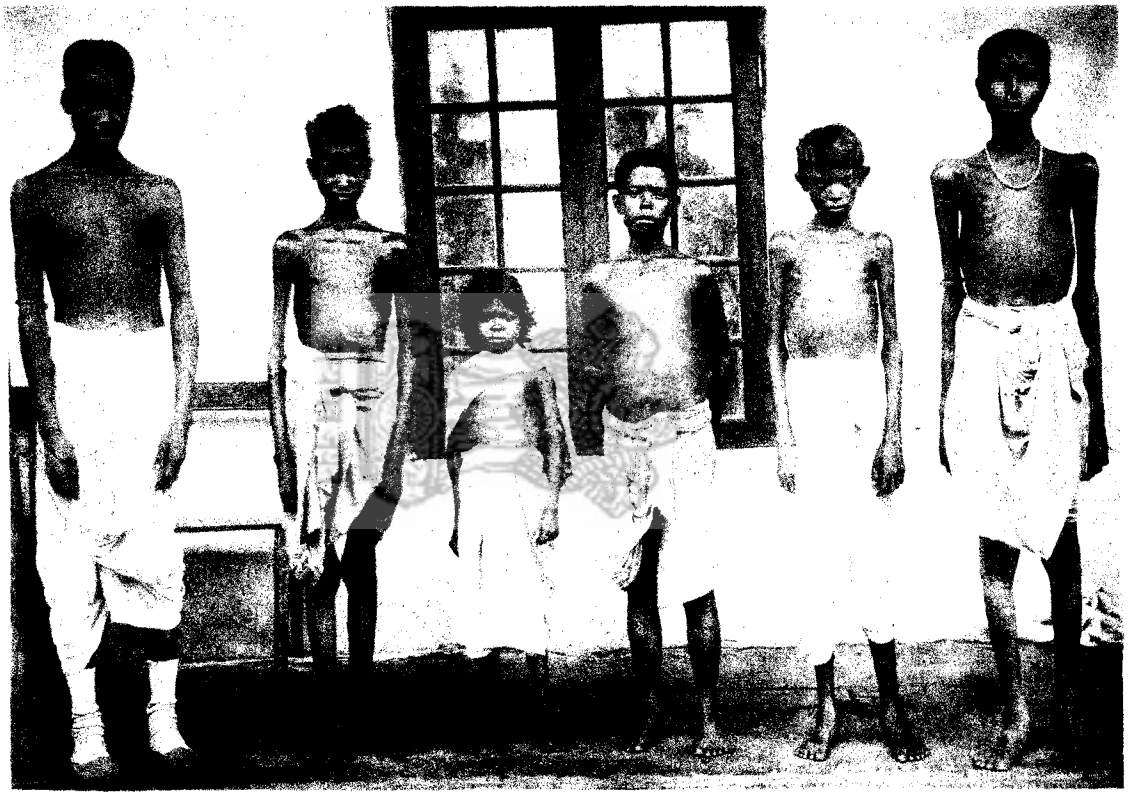

Photo-etching. Survey of India Offices, Calcutta. April 1887.

GROUP OF CASES OF KALA AZAR AT MANGALDAI.

The exact number of persons in each family of my cases was noted, both as to how many had suffered from, or died of, *kála-ázar* and how many had escaped the infection. Taking only the Nowgong dispensary cases, the following very remarkable figures were obtained. Among the relatives of 20 cases, 123 had suffered from *kála-ázar*, all but 2 dying of the disease, while only 44 had escaped; or to put it in another way, no less than four-fifths of the cases had lost half or more than half their relatives from the disease in the course of from three to five years. Cases, who had no relatives in Assam, as was the case with some imported coolies, were omitted from this calculation. This, taken with the facts to be mentioned in Section VII, is sufficient to prove that the disease is capable of being contracted in some way or other by living in a house in which infected persons reside. This was also Dr. Giles' opinion, although the explanation of the fact, I believe, to be very different from that which he propounded. It should be mentioned that the dispensary cases show a stronger family history than the general average, owing to the presence of some children who had lost their parents and other relatives from the disease.

General clinical description of *kála-ázar*.

The disease has been described in its general features so often, that only a few words need be written here on this part of the subject, especially as it will be necessary to go into considerable detail on those symptoms of the disease regarding which my observations differ very widely from those of Dr. Giles. A typical group of cases at the Mangaldai dispensary is shown in the photograph opposite this page. The man on the left had only suffered from fever for a short time, but it was thought that he might be a very early case of *kála-ázar*. However, he soon recovered, so probable had only ordinary fever, but he serves as a good contrast to the other definite cases of the disease. As already mentioned, the early stages of the disease cannot be distinguished from ordinary malarial fevers, while the latter stages resemble in every respect very

advanced cases of malarial cachexia. Two very different clinical types, however, occur, which may best be distinguished by wasting of the body, accompanied by great enlargement of the spleen and liver, although to a variable degree in different cases in one type, and in the other by the same symptoms complicated by dropsy of the legs and abdomen, and in the very last stage, only in rare cases, of the face also, which imparts a very different aspect from that met with in the first class. The latter may easily be mistaken for anchylostomiasis, and, indeed, can sometimes only be diagnosed from it by an examination of the blood. The dropsical type is much more rare than the other, and its pathology will be explained presently. Taking then the common type, a case will show great wasting of the whole muscular system, best seen in the thinness of the arms and face, and the prominence of the ribs, and even of the sternum (breast-bone), which contrasted with the enlarged abdomen in which the outline of the enormous spleen and liver may sometimes be dimly discerned, forms a striking clinical picture which is well illustrated in the photographs opposite pages 33 and 57. If to these prominent features be added anæmia of a more or less marked character, usually, but not necessarily, accompanied by a yellowness of the conjunctiva, and more or less marked weakness and fever, and in a minority of the cases by slight œdema of the feet, the picture will be fairly complete. The skin presents a leaden hue, which is said to be characteristic of the disease, but which is difficult to appreciate if the patient is seen but once, although those, who see people whom they have known well in health gradually waste away from the disease, are unanimous in looking on it as a constant feature of the affection. I can, however, confirm the fact from my own experience, as I was much struck on my return to Nowgong after two months' absence on tour, by the darkening of the face in several cases in the Nowgong dispensary, which had taken place during my absence. Its pathology will be discussed in Section V.

The duration of the disease.

The average duration from the first admission for fever to the date of death, taken from 193 cases on a Nowgong tea garden, was 7·4 months. The cases at the beginning of the epidemic, however, averaged only 4·5 months, and during the last year they averaged 10 months, the intermediate two years being 5·5 and 7 months, respectively, showing a progressive increase in the duration of the disease each year, proving that the disease was most virulent at the beginning of the outbreak, and diminished in intensity each year. Moreover, recoveries were more common during the last year, pointing to the same conclusion. A very few cases died within two months of their first admission to hospital, but they had probably suffered from fever for some considerable time before admission, as I have never seen any case which I have been able to follow through the greater part of the illness, that has been fatal in less than three months; while, on the other hand, they may extend, with remissions of some months' duration, over three years. From two months to three years may be taken as the extreme limits, while the usual duration is from four to nine months.

The seasonal incidence.

This point has also been worked out from the tea garden cases, which show that, although they begin in all months of the year, yet there is an increase in the number commencing in April, they are highest during May, June, and July, decline again in September, and reach the minimum during December, January, and February. This seasonal incidence agrees very closely with that of ordinary malarial fevers in the Nowgong district, except that it is a more extended one, and is of considerable importance, as will be shown in a later section. The death-rate, on the other hand, showed two maxima, one in April and the other in October and November, and this again agrees with the monthly death-rate from *kála-ázar* and ordinary fevers combined during the epidemic years in this district, as is shown in the chart given in Section VI, for it is certain that the illiterate agents who register the deaths, do not accurately distinguish

between those from the two kinds of fever. Cases then begin chiefly during the rainy season, for in each of the years during which this tea garden outbreak has lasted, namely, from 1893 to 1896, there was a considerable amount of rain in the months of April and May, while the main death-rate occurs at the end of the rains, and at the beginning of those of the ensuing year, which is accounted for by the fact that most of those cases, whose fever begins during the rains, die at the end of them, or at the onset of the cold weather, while those which survive that period, usually lose their fever for a time during the cold months, but relapse and die as soon as the rains begin in the next year.

Age and sex.

The age of the patients is of importance, as it may be compared with that of anchylostoma cases as noted by other observers. The disease attacks people of all ages, from children one year old to quite old people. My cases vary from a baby of one year, who was still being suckled, and whose mother and father had the disease, to a man of 67. There is, however, a very marked tendency for it to attack children. Thus, of the cases of which I have notes, 25·6 per cent. were under 10, 24·4 per cent. were between 10 and 20, 34·15 per cent. were between 20 and 30, 10·97 per cent. were between 30 and 40, while 4·87 per cent. were 40 or over. Exactly half then were under 20 years old, while the average age of the whole was only 14·84 per cent. The ages of 402 cases of anchylostomiasis, as given by Dr. Sandwith of Cairo, are as follows: under 10, 0·25 per cent., between 10 and 20, 13·94 per cent., between 20 and 30, 42·28 per cent., between 30 and 40, 34 per cent., 40 or over, 10·67 per cent. Thus, only 12·25 per cent. were under 20 years old, as compared with 50 per cent. of *kalá-ázar* cases. With regard to the sex, the tea garden cases show that both sexes suffer equally, and, moreover, the occupation of the coolies does not affect their liability to the disease, for those who work in the tea-house suffered just as much as those who did hoeing and other outdoor work.

Predisposing and opposing causes.

Although all classes suffer from the disease when once it has been introduced amongst them, yet the poor, I think, suffer in a disproportionate degree, and have less chance of recovery, owing to their living in more crowded dwellings, and to deficiency of nourishing diet, especially of a nitrogenous nature. On the other hand, I have known a Babu in very good Government employ, who lost 39 relatives of the disease. Again, Europeans who were thought by Dr. Giles to escape the disease, have, since his report appeared, been proved to be liable to it. I have reliable information of seven certain cases, about most of which I have obtained details from the Medical Officers who attended them, while in the Nowgong district alone, there have been five cases with one recovery, and two of the deaths occurred in one household. I find, too, that in the tea garden cases, a good many had suffered from such depressing affections, as dysentery and diarrhœa, etc., shortly before they were first attacked by *kála-ázar*, while two cases had previously had anchylostomiasis, for which they had been successfully treated a few months before they got *kála-ázar*, but were still anæmic.

On the other hand, there is evidence to show that opium-eaters suffer less from the disease than those who do not take it. Thus, although it appears from the evidence given before the Opium Commission that a considerable percentage of the natives of Assam habitually consume this stimulant, yet out of 51 adults, who suffered from *kála-ázar*, only one was an opium-eater before he was attacked by the disease, and he recovered from it, while two others took to it after they got ill, and one of these recovered, and the other died of lung complications, some weeks after he had lost all fever. Again, it was given in evidence before the Opium Commission that a village of opium-eaters in the Kamrup district escaped the epidemic, although others around them were depopulated by *kála-ázar*. This opinion was also confirmed by my enquiries in some of

the villages of the Nowgong district, and it was found in one outlying part of this district that the villagers had become so convinced of both the prophylactic and curative action of opium, that its consumption had greatly increased among them, as will be shown in further detail in Section VII.

Fever.

This is the most essential and constant feature of the disease. True cases of *kála-ázar* invariably begin with fever, which may be of an irregularly remittent or of an intermittent type. The former is most common in the first attacks, and is also met with not unfrequently in the final fatal relapse. In the majority of cases, which I have been able to follow up for some months, both types occur at different stages of the illness, as is well shown in the temperature chart opposite page 73, while in a few cases only occasional attacks of a remittent nature are seen, and in a fair proportion only intermittent fever occurs, and in these the fever is of a much more constant nature than in the former instance, and sometimes lasts for two or three months at a stretch, but then usually the temperature does not rise above 101° or 102°F. In the remittent attacks, it may reach 105° or more, and I have seen a temperature as high as this the day before death. I have seen the temperature fall two degrees below normal, but have not met with the persistent low temperatures described by Dr. Giles, except in cases of pure anchylostomiasis. In the great majority of the cases which succumb during the rains, death takes place during a final paroxism of fever, which is often more severe than has occurred for some time previously. I have notes of three cases in which the first attack of fever was said to have been of a tertian character, in one of which the patient was positive that it persisted for two months. Dr. Campbell also mentions that the fever may be tertian in *kála-ázar*, and this is a point of considerable importance, for it is obvious that if one case may begin as a tertian, and another as a quotidian, while a third starts as a remittent fever, then the fever has no characteristic

and constant type such as would constitute it a special form, malarial or otherwise, such as the summer-autumn type of malarial fever, described by Marchiafava and Bignami, and this accounts for the impossibility of distinguishing it from ordinary malarial fevers. The temperature was taken four times a day in a series of cases; but this only served to reveal the extreme variability and irregularity of the course of the fever. As regards the constancy of the occurrence of fever in true *kála-ázar*, not only was a clear history obtained in every case, but an analysis of the tea garden cases shows that each was admitted on the average 3·5 separate times for fever, in addition to other admissions under such headings as anæmia, rheumatism, diarrhœa or dysentery, and dropsy, during many of which they doubtless also suffered from fever. About three-quarters of the admissions were for less than one week, while the intervals between the admissions vary from a few days to two months or more, the commonest interval being from two to four weeks. Many of the cases were admitted for debility, anæmia, diarrhœa, or even dropsy, within a very short time of their first admission for fever, showing that they had suffered from the latter in a mild form for some time before coming to hospital, as, indeed, the history of the cases shows to be very commonly the case. In fact, I should say that about half of the cases begin in this way, while the rest begin with a sharp attack of remittent fever. The fact that the earliest cases which I could find showed in almost every instance marked changes in the blood, such as could only be produced by fever of some duration, also strongly supports this opinion. One other very marked characteristic of the fever in this disease remains to be mentioned, and that is, that after two or three attacks the patients seem to lose all reaction to the fever. Thus it is a well-known fact that patients, who have a temperature of 103°F. or over, will sometimes deny that they have any fever at all, and will, moreover, eat a very fair meal without any apparent discomfort. I have even seen a coolie on a tea garden get most indignant at

being kept in hospital, and say he had no fever, although his temperature had been between 102° and 104°F. for several days. In such cases, the white corpuscles will be markedly reduced, even to only 1 to 2,000 red, so that leucocytosis cannot occur to any extent, and this is very likely the explanation of the loss of reaction to the fever. This point also explains the short stay of the majority of the patients in the tea garden hospital, especially as most of the cases occur during the busy season, when the coolies can make a great deal of extra money, and are correspondingly loath to stop in hospital. It is also a fact that these cases will work up to within a few days of their death, for, as long as they are free from fever, they will work unless prevented from doing so, although they can do but little, and then they will get a relapse of the fever, which will carry them off in a very few days. Thus, I find that about half the tea garden cases died within a week of their last admission to hospital. This loss of reaction to the fever results in the patients working when they have got fever upon them; and as they are also for the most part anæmic as well, they are thus deprived of the rest which is absolutely essential to their having any chance of recovery—a fact that, together with their not applying for treatment, or, if they do come to hospital, their discontinuance of it after a very few days, partly accounts for the fatality of the disease. It is also of great importance in connection with Dr. Giles' statement that, "on a more and more close interrogation, however" (which, by the way, generally results, in the case of a native, in getting him to say whatever the questioner has in his mind), "it generally came out that there had been comparatively little fever, and that what the patient really meant was merely that he had been feeling ill for a long time," for it is obvious that patients suffering from *kála-ázar* will under-estimate, rather than over-estimate, the amount of fever from which they have suffered. Thus there is no definite type of fever in the disease, while its duration, and that of the intervals between the attacks differ very widely, the only constant characteristic being

the inveterate tendency to recur again and again until a fatal issue results, and the rapidity with which the liver and spleen become greatly enlarged, and the extremities and body waste.

Enlargement of the liver and spleen.

The spleen is always enlarged in some stage or other of cases of *kála-ázar*, but it is not so much the frequency of its occurrence as the constancy with which it attains to a very considerable size, that is the most remarkable feature of the disease. Thus, in a series of 70 cases, it was found to be enlarged in all, while in 94·5 per cent. of them it reached at least three fingers' breadth below the margin of the ribs, while in 56·5 per cent. it reached to or below the navel, and in 27·7 per cent. it extended as far as the level of the anterior superior spine of the ilium. In the more chronic cases it was hard, but in other cases it was soft, and sometimes varied in size from week to week in a remarkable manner. In cases of recovery it not unfrequently disappears entirely beneath the ribs after some months, even when it has been very large during the illness. The liver was enlarged in 93 per cent. of the cases, including a few in which it extended upwards only, as revealed by a higher level of its dulness than normal. Here again, the size which it often attained to, was remarkable, and has, indeed, been thought by some to be a characteristic feature of the disease in which it differs from ordinary malarial fevers; but, as a matter of fact, as will be shown directly, it is not more enlarged in *kála-ázar* in proportion to the enlargement of the spleen, than it is in the ordinary malarial fevers of Assam. It extended to two fingers' breadth below the ribs in 64 per cent., and to four fingers below in 24 per cent. The edge could usually be easily felt, and in the more chronic cases it was quite hard. Pain over the liver and spleen is of frequent occurrence, and may be very severe, being due to peri-splenitis and peri-hepatitis. Dr. Giles was of the opinion that the spleen was not more often enlarged in *kála-ázar* than it was in the "reputedly healthy

population," but he produces no evidence in favour of this view beyond quoting Dr. Dobson, as having found 21 out of 42 children in a Goalpara village to have enlarged spleens, and this in a district which was at the time affected by *kála-ázar*, and that he had found about the same proportion in the Chaygaon district (also affected by the disease), but he could not give the figures, as he had lost his notes on the subject. On the other hand, he does not state in what proportion he found the spleen enlarged in *kála-ázar*, although he admits that it is so in "most of the cases;" but as he included pure cases of anchylostomiasis among them, the absence of the exact figures is of no account, for, as I shall prove in the next section, this disease is quite distinct from *kála-ázar*. In order to estimate the importance of the enlargement of these organs, it is necessary to know in what proportion of apparently healthy inhabitants of the district they are enlarged, and to what size. To determine this point, I examined 65 men in the Nowgong jail in the month of June, with the result that 12 of them, or 18·46 per cent., had some enlargement of the spleen; but of these, in only one was it three fingers or more below the edge of the ribs, and this man died of *kála-ázar* very shortly afterwards. Again, I examined 200 coolies in the lines of a tea garden, where there were no cases of *kála-ázar*, except the Assamese wife of one of the sirdars, who came from an infected village. The size of the spleen and liver, as well as the presence of anæmia, pigmentation of the mucous membrane of the mouth, and the amount of fever from which each coolie had suffered during the previous two years, were noted. The results, which are very instructive, were as follows: The spleen was enlarged sufficiently to be felt in 25 per cent., but in only 6 per cent. did it reach to three or more fingers' breadth below the ribs, and in only 1 per cent. did it extend below the navel. What was more remarkable was the fact that in 19 per cent. of the coolies the liver was enlarged, and the enlargement was in proportion to that of

the spleen. Thus, in the 150 cases in which the spleen was not enlarged, only 3·33 per cent. had any enlargement of the liver, while in the cases in which the spleen could be felt, but did not extend as much as three fingers' breadth below the ribs, 57·89 per cent. had some enlargement of the liver, though in the majority of them it was not as much as one finger's breadth below the ribs; but in the cases in which the spleen reached to three fingers or more below the ribs, in 91·65 per cent. the liver was enlarged, and in more than four-fifths of these it extended one or more fingers' breadth below the costal margin. This, it will be observed, is almost exactly the same proportion as the liver is enlarged in *kála-ázar*, so that it is evident that in this disease it is only enlarged in proportion to the enlargement of the spleen, exactly as it is in ordinary cases of malaria in Assam. Once more, there is a very definite relation between the amount of fever that the coolies had suffered from and the enlargement of the spleen and liver, and also between these and the frequency of anæmia amongst them. Thus, of the cases in which the spleen was not enlarged, only 10 per cent. had suffered from fever to any degree within the last two years, and two-thirds of these had not suffered within the last year, while 27·33 per cent. of them showed more or less anæmia, but in two-thirds of these it was not marked. On the other hand, of those who had slight enlargement of the organ (less than three fingers' breadth below the ribs), 61·16 per cent. had suffered from fever, and 55·33 were anæmic, and of those who had marked enlargement of the organ, 75 per cent. were anæmic, the majority of them being markedly so. It is also worthy of note that in a good many of the anæmic cases with enlargement of the spleen, the conjunctiva was white, so that the conjunctiva need not necessarily be yellow in cases of anæmia due purely to malarial fever; and the main point which Dr. Giles relied on to differentiate anchylostomiasis from the anæmia due to malaria, is based on false premises, and the great majority of the cases which

he quotes from other writers, and states to be "clearly cases of anchylostomiasis," are not necessarily so, while the two cases which he details as having been seen by himself, are not cases of *kála-ázar* at all, but simple anchylostomiasis. Hence the necessity of this full description of true *kála-ázar*. With regard to the pigmentation of the buccal mucous membrane, it may be mentioned here that I have been able to distinguish two kinds, one of which is normal to many of the darker skin races of India, and is most frequently seen in the indigenous inhabitants of Chota Nagpur, namely, the Kols and Mundas, while the other is, I believe, a pathological result of malarial fever, and hence is of diagnostic value. The former presents itself as black patches on the tip or sides of the tongue, and in extreme instances on the floor of the mouth also, and as a diffuse slate-coloured hue on the hard palate, only rarely extending back to the soft palate. In fairer natives only a little light-brown colour may be seen along the central line of the hard palate, and this, as well as the darker colour, is of no clinical or pathological significance. On the other hand, small dots of a brown colour, usually of a lighter tint than the above described, and often best marked on the soft palate, and not seldom of a slightly raised character, are very commonly seen in persons who have recently suffered from malarial fever, especially if it has been of a severe or continued type. Thus the former diffuse form was met with in from 50 per cent. to 66 per cent. of the different coolies classified according to the enlargement or not of the spleen as above, but the dotted form was seen in only 24 per cent. of those who had no enlargement of this organ, but was present in 76 per cent. of those who had large spleens. The point is of some importance, as this form of pigmentation is met with very commonly in *kála-ázar*, and I am not aware that the distinction between the normal and the pathological forms of pigmentation met with in the mouth, has been pointed out before.

It is then evident that the spleen is enlarged four times

as frequently in *kála-ázar*, as it is in healthy persons in Nowgong; while if the size be taken into account, as, of course, it should be, in order to obtain a correct comparison, then it may be said that this organ is markedly enlarged in *kála-ázar* 15 times as often as it is in the ordinary inhabitants of Nowgong, for the tea garden coolies who were examined lived in lines near jungle, and with a *beel* close by, so that the site was quite typical of Assam, and a large proportion of them had suffered from malarial fever at one time or other in the course of the last two years. The great majority of the men in jail were Assamese.

Anæmia.

This symptom will be only briefly discussed here, as it will be more fully treated of in the next section. It was present, as shown by pallor of the conjunctiva or buccal mucous membrane, in 93 per cent. of the cases, being slight in 31 per cent. and marked in 62 per cent. Moreover, in the cases in which the conjunctiva was of a good colour, the hæmoglobinometer showed distinct evidence of anæmia being actually present, and it is worthy of note that the hæmoglobin may in some cases be reduced by one-half without anæmia being visible in the mucous membranes, and the use of the instrument has proved that anæmia is really an absolutely constant symptom of the disease. As already mentioned, however, the conjunctiva is not always of a yellow colour, but in very nearly one-third of the cases in which this point was noted, it was white, and in the other two-thirds yellow. It was more frequently yellow in those cases which showed marked enlargement of the liver. In those cases who had not suffered from fever for a long time, the blood improved markedly, and this often occurred while the patient was still very weak and thin. Such cases were very liable to be carried off by diarrhœa, pneumonia, etc. It may also be noted here that the anæmia was in proportion to the fever, and if fever was slight or absent for a few weeks, an immediate improvement in the blood took place, and, as soon as fever recurred, the blood ran down again,

proving that the anæmia is directly due to the fever, and to that alone. Moreover, if the temperature only rose to 100°F., or so, even if it was fairly persistent, the blood maintained its state without gaining or losing in any degrees; but if the temperature rose to 103° or 104°F., it rapidly became worse—a point of practical importance. I have seen the hæmoglobin run down, in a final fatal attack of fever, to 12 and even to 8 per cent. of Gower's standard; but such extreme anæmia is rare in *kála-ázar*, although common enough in anchylostomiasis.

Dropsy.

This symptom appears either as a slight œdema of the feet, which is of little significance, or as well marked swelling of the lower extremities, which may in rare cases involve the abdominal cavity, and even the pleura and pericardium. The slight form was present in 12·5 per cent. of the cases, and had been present at some previous time in 19·5 per cent. more, or altogether in about one-third, while the more severe form was met with in 12·5 per cent., in one-third of which it was present in the abdominal cavity. Œdema of the face is very rare, and only occurs in the very last stage in that class of cases in which œdema of the serous cavities is a marked feature. This is in marked contrast to the œdema of anchylostomiasis, which Lutz describes as being most frequently seen in the eyelids and cheeks, and as appearing early and giving to the patient a characteristic look. The dropsy of *kála-ázar*, which always begins in the feet, is of cardiac origin, while cirrhosis of the liver, perihepatitis and periplenitis, and possibly, but I think rarely, pressure of the enlarged abdominal organs on the inferior vena cava may assist in its causation. In anchylostomiasis it is due to the poverty of the blood, more especially to its low specific gravity, as will be shown later on. A very considerable proportion of *kála-ázar* cases, over one-half, go through their whole course to a fatal termination without any œdema whatever, while it always occurs in fatal cases of anchylos-

tomiasis, except, perhaps, in some of the very rare so-called acute cases.

In most of the more chronic cases of *kála-ázar*, the veins on the surface of the abdominal wall become enlarged, so as to stand out very prominently, and many of these cases also have slight œdema of the feet or legs. This enlargement of abdominal veins is only met with in cases where the spleen and liver are much enlarged, and is due to the obstruction of the portal circulation. They disappear completely in cases of recovery, even when they have previously been well marked.

Circulatory system.

The changes in the circulatory system are mainly dependent on the changes produced in the heart itself, so these will be first described. I was much struck by the small size of the heart *post-mortem*, especially in the more chronic cases, but subsequently came to the conclusion that this was, to a great extent, accounted for by the general wasting of the whole body, although not entirely so. Clinically, the heart is often smaller than normal, as shown by the apex beat being usually in the fourth space, and just inside and below the left nipple. The diminution of cardiac dulness was also common, it being very frequently limited by the left edge of the sternum, the fourth rib, and only extending outwards to a finger's breadth inside the nipple line. The apex beat was usually invisible, and very often could not be detected by palpation in spite of the thinness of the chest-wall. If felt, it was usually feeble, and with a tendency to diffuseness, which made it difficult to accurately localise it.

The heart sounds were usually feeble at the apex and over the aortic cartilage, but the pulmonary second sound was sometimes noted to be louder, but probably this was only of a relative nature, except in a few cases where there were signs of mitral regurgitation. Murmurs were of surprising rareness, considering the marked nature of the anæmia, as they were only occasionally met with, most frequently being heard over the pulmonary cartilage.

Venous murmurs over the neck were not uncommonly present.

Enlargement of the heart was only met with in very chronic cases, and in those in which mitral regurgitation was present.

The tendency to diminution of the size and power of the heart, is no doubt due in great part to the deteriorating effect of the prolonged fever, which produces granular degeneration of the heart muscle (which I found in a marked degree in the case of a man who died within a few hours of having a temperature of 105·4°F.) followed by fatty degeneration.

The anæmia also aids in producing the fatty changes. The dropsy of the lower extremities is a result of the cardiac weakness. The pulse remains to be described, and it will be noted that it is in accordance with what might be expected from the above-described cardiac changes. Thus, the artery is usually small in the latter stages, and the tension is nearly always low, the high tension pulse of anæmia being quite the exception, although it is met with in some cases where the power of the heart is fairly well maintained, while, presumably from the diminution of the quantity of the blood in the body, the arteries are small, having contracted down on the blood contained in them as it were. When there is fever, of course, the pulse is increased in frequency, and in the last stages, especially when dropsy has set in, it is permanently accelerated. It is usually quite regular both in force and frequency, unless marked fatty changes have taken place in the heart.

The changes in the heart are of considerable diagnostic and prognostic importance, especially in differentiating the disease from anchylostomiasis, for in this latter affection the heart is very commonly enlarged from dilatation, and often also subsequent hypertrophy, and well-marked and visible palpitation, is a well-known feature. Moreover, hæmic murmurs are of very frequent occurrence, accompanied by venous murmurs in the veins of the neck in anæmia of coolies, doubtless due to the alterations in the specific gravity of the blood

in this disease, while in the anæmia of *kála-ázar*, as will be shown in detail in Section IV, the most important elements of the blood are reduced in much more equal proportion than they are in anchylostomiasis.

Nervous system.

The changes in the nervous system are very slight, as might be expected in a slow-wasting disease of this nature, for this system has been proved to be the last to lose weight in starvation. Sensation does not appear to be ever affected in the way it is in the true form of *beri-beri*, which is met with in the Straits Settlements (but has never been found in Assam, so that the use of this name for anæmic conditions is incorrect), nor is the knee-jerk lost, although it may be somewhat difficult to elicit it in the latter stages of the disease.

The retina was examined in a good many cases in the latter stages, and showed some changes of interest which account for the diminution of vision, which is not unfrequently met with, and which usually takes the form of night blindness, or inability to see after sunset, when the light is fading, and is accompanied by diminution of the field of vision. The change most commonly seen was a remarkable diminution of the size of the retinal arteries, while the veins remain of nearly full size. This was usually most marked in those cases in which the pulse was also noted to be small and of low tension. The disk was also pale, and probably the retina shared this change; but owing to the great variation in the shade in different natives, this cannot definitely be stated to be the case. Hæmorrhages were very rarely, if ever, present, although carefully looked for, especially in view of the fact to be presently mentioned, that hæmorrhage from the nose was frequently noted to have occurred. Their absence seems to be partly due to the contraction of the retinal arteries, and partly that these vessels were found to be unaffected by the fatty degeneration, even in a case in which the cerebral arteries were so affected. The anæmia of *kála-ázar* is moreover rarely so great, as that seen in so-called pernicious anæmia. The

impairment of vision is evidently due to loss of sensibility of the retina from diminution of its blood-supply.

Respiratory system.

There are no primary changes in the respiratory system, with the exception of the increase of the frequency of respiration during fever. Congestion, especially of the bases of the lungs, and more rarely actual pneumonia, usually of the lobar type, or pleurisy, are not an unfrequent complication in the last stages, especially during the cold-weather months, and they often play an important part in bringing about a fatal termination of the case. Acute œdema of the lungs may rapidly cause death in cases which are already suffering from abdominal or pleural effusion.

Digestive system.

The pallor and pigmentation of the mucous membrane of the mouth, have already been described. The appetite is reduced in two-thirds of the cases, but there is sometimes a very remarkable desire for animal food, which will even lead those who have never partaken of it before, owing to caste reasons, actually to ask for it. This appears to be due to the loss of the power of digesting vegetable foods, as is shown by the fact that when looking for anchylostom ova, I have frequently seen starch granules, which have passed through the whole intestinal tract untouched. Sickness is very rare, being only noted once, but nausea after food is more common. Diarrhœa is very common, and was noted as having been present at some period or other in three-fourths of the cases. It is most commonly met with in the latter stages, when the patient is very emaciated, and is due to either inflammation of the small intestine accompanying high fever or to the resulting atrophy of its mucous membrane which allows of the food passing through the intestinal canal undigested, and so causing irritation of the bowel. It is a very bad sign if persistent, such cases showing no recuperative power, even if the fever ceases, and die of asthenia or a relapse of the fever. They seem to have lost all power of assimilating vegetable food, and the only chance for them is a diet of milk and animal

food, and above all, treatment by bone marrow tabloids, as will be described further on. Constipation is uncommon, and was only met with, to a slight degree, in 5 per cent. of the cases. This is a very different state of affairs to that found in anchylostomiasis, in which Dr. Sandwith noted constipation of an obstinate nature in 60 per cent., and diarrhœa in only 28 per cent., in some of which it was due to Bilharzia ulceration in the rectum. Cancrum oris not unfrequently appears in the later stages of the disease, and is almost invariably fatal, although I have seen one case recover from it with much resulting deformity.

Genito-urinary system.

As soon as cachexia and anæmia become marked symptoms, sexual power and desire is lost, and menstruation ceases. The urine varies according as fever is present or not. In the former case, it is scanty and high coloured, and with a fairly high specific gravity, while in the absence of fever in advanced cases, it is normal, or of rather a high colour (in which respect it differs from that of anchylostomiasis, which is light coloured), neutral or faintly acid, and of a low specific gravity, usually from 1,006 to 1,012. A trace of albumen is met with in a small proportion of the cases, and in a few it is present in larger quantities, usually in the dropsical class. Phosphates are also often present, and mucous in small quantities. The comparatively high colour of the urine in *kála-ázar* is doubtless derived from the hæmoglobin of the red blood corpuscles, which are constantly being broken down.

Pain in the extremities.

This symptom must not be omitted, as apart from any dropsy, it is very commonly met with. It may be so marked as to lead to the admission of the patient under the heading of rheumatism, and is due to the porosis of the bones brought about by the changes in the bone marrow, which will be described in Section V. It is more common in the lower extremities, and may be most severe in either the shafts or the ends of the long bones.

Progress and termination.

As the disease progresses, the appearance of the patient becomes more and more typical, and, although individual cases cannot be distinguished from ordinary chronic malaria, yet when several members of a family or small village are affected by the disease, the clinical picture is quite characteristic of *kála-ázar*. The fever relapses again and again, the spleen and liver become more and more enlarged, until the abdomen stands out in marked contrast to the emaciated face, chest-walls, and extremities, in which the outline of every bone is easily seen beneath the integument, which loosely covers it, while the leaden hue of the skin contrasts with the pallor of the mucous membranes, the feet often become swollen with œdema, and the patient drags on a wretched existence until a fresh attack of the fever, or less commonly diarrhœa, congestion or inflammation of the lungs, or cancrum oris brings him a merciful release from his miseries.

It is evident that the last stages of *kála-ázar* may closely resemble those of anchylostomiasis, and especially if the patient is seen during a prolonged remission of the fever, which so often occurs in the cold-weather months, and if in addition any enlargement of the spleen and liver is regarded as an accidental feature, and the history of previous fever is taken to mean only that the patient "had been feeling ill for a long time," then it will not only be easy, but quite natural, to look on the case as one of anchylostomiasis. If, then, the ova of this parasite be found in the fæces, and it is not recognised that they are present in from 70 to 80 per cent. of the ordinary inhabitants of Assam (and it must be borne in mind that this fact was not known at the time of Dr. Giles' investigation), then the diagnosis will very naturally be considered a certain one. The fallacies involved in this argument will be evident from what has already been written, for apart from the fact that fever and enlarged spleen are constant symptoms of true *kála-ázar*, it is now obvious that in order to constitute anchylostoma a

factor in the causation of *kála-ázar*, it will be necessary to show that they are present in all, or at least a great majority of, the cases in considerably larger numbers than they are in ordinary healthy people of the infected districts. Let us see if this is the case or not.

The frequency and number of anchylostoma present in *kala-ázar* cases.

In deciding whether anchylostoma play any part in the causation of *kála-ázar*, it is necessary to determine in what numbers they are present in both healthy people and in cases of the disease. Dr. Dobson's work has proved that they are present, for the most part in small numbers, in over 80 per cent. of all imported coolies, and in nearly 70 per cent. of persons resident in Assam. I have examined 50 men in the Nowgong jail in precisely the same way that Dr. Dobson examined his cases, and found the worms present in numbers varying from 1 to 293 in 66 per cent., but in only two cases were over 100 found. The average number met with in those men who were infected by this parasite, was 18·8. Again the average number met with in 7 medico-legal *post-mortems* was 32.

Thus it will be seen that, although they are very frequently present in healthy people, they are usually found in quite small numbers, which, in view of the opinion of all who have had much experience of anchylostomiasis, that 500 of these worms must be present for from six months to one year in order to cause anæmia in a healthy person, may be expected to be absolutely harmless in healthy well-fed people. That, as a matter of fact, they do not cause any injury to the blood when present in these small numbers, will be proved in the next section. On the other hand, in 4 cases of anchylostomiasis examined *post-mortem*, I found the worms to average 200.

Let us now see in what numbers they are present in cases of *kála-ázar*. In 1891, Dr. Dobson found them present in 75 out of 99 cases of this disease, but the numbers present were not given. In his report in 1892, however, he

records that he found anchylostoma in 11 out of 15 cases, the average number being 10·4.

Dr. Campbell also found them to be present in 75 per cent. of 88 cases; but among this number he included 15 cases of pure anchylostomiasis, and he does not give the numbers in which they were found. Coming to my own researches, I examined for the ova of the worms in as many cases as possible, and to some, who were in a fair state of health, I gave thymol, and counted the worms which were passed. In searching for the ova, a small piece of fæces was spread out under a cover slip, with a drop of carbolic lotion, and the whole of the specimen carefully and systematically examined under the microscope. I found this a much surer way of finding the ova than that described by Dr. Giles, and by this means I was able to detect the ova in three successive cases, in which, after giving thymol, only 2, 14, and 8 worms, respectively, were found, so that it is evident that if only from ten to twenty were present, they would thus be detected. In this way it was found that there were either no ova detected or less than 20 anchylostoma were passed after thymol in 83·33 per cent. of the *kála-ázar* cases, while in 16·66 per cent. 50 or more were present; but in only 10 per cent. did the blood changes show that these had affected its composition. Again, in 25 *post-mortems* on *kála-ázar* cases, an average of 21·48 worms were found, including three mixed cases, in which the disease was complicated by 50, 80, and 103 worms, respectively. Thus it appears that in the great majority of *kála-ázar* cases, anchylostoma are not present in sufficiently large numbers to affect the patient in the least degree. In view, however, of the argument, that when the worms are only found in such small numbers during life or after death, they may have been present in much larger numbers at some previous stage of the disease, which is true in exceptional cases, it is difficult to absolutely exclude their action in this way. A means of doing so has, however, been found in the blood changes, as will be shown in the next section.

The death-rate.

This only remains to be discussed, and it may at once be said that it is so high that some have even doubted if it is ever recovered from. The most reliable evidence has been obtained from the study of the tea garden outbreak, in which the mortality was 96 per cent. This contrasts very markedly with the death-rate from anchylostomiasis, which, in the 402 cases of Dr. Sandwith, was 8 per cent., while Lutz lost 5 out of 150, 3 of which died from other complications, and he quotes two Italian observers as treating 787 successive cases without a death. Moreover, Dr. Dodds Price tells me he now very rarely loses a coolie from anchylostomiasis. Again the great majority of the tea garden *kála-ázar* cases were treated with thymol in an early stage of the disease, not because they were thought to be cases of anchylostomiasis, for they were looked on by Dr. Price as a bad type of malarial fever, unlike anything formerly met with in the district, but because in view of Dr. Giles' report it was thought well to exclude possible complication by anchylostomiasis. This series of cases may, therefore, be taken alone as furnishing conclusive proof that, whatever *kála-ázar* may have been in the Gauhati district in 1889, the increased mortality on this tea estate was not due to anchylostomiasis. This mortality agrees closely with that in villages in the Nowgong and Mangaldai districts, where it is nearly always over 90 per cent., but I have met with a somewhat larger proportion of recoveries among patients treated in the Nowgong dispensary; but, on the other hand, it must be admitted that I worked at a time when the epidemic was subsiding, at which time, as already pointed out, the disease is less intense, and will accordingly be less fatal.

In cases of recovery, the first change is a prolonged cessation of fever accompanied by a marked diminution of the anæmia, and subsequently an improvement in nutrition, and a reduction in the size of the spleen, which, however, usually takes a many months to reach its normal size, but it

certainly does do so in time in some cases. There is also a tendency for persons who have recovered from the disease to grow fatter than they were before their illness—a sign of the great interference with metabolism. That more cases do not recover is, I feel sure, due largely to the fact that, so few of them remain under treatment long enough to allow of the damage done by the fever to be repaired, so that when a relapse comes, they are not in a position to meet it, and this view will be further illustrated later in this section.

The high rate of mortality above given applies to cases in which well-marked signs of chronic malaria have become developed, but there is good evidence to show that, when *kála-ázar* is prevalent in a place, there are a greater number of cases of apparently simple malarial fever, which are recovered from, some of which must really be mild cases of *kála-ázar*. Thus, on the tea garden, which has been mentioned as having suffered so severely, many cases of apparently ordinary malarial fever, which were more amenable to treatment than typical *kála-ázar* cases, were met with during the early cold-weather months, when other neighbouring gardens, which had not been attacked by *kála-ázar*, had very few or no fever cases. Some of these are nearly sure to relapse next rains and become typical cases of *kála-ázar*, but others will probably be protected by the slight attack against a more severe one.

ILLUSTRATIVE CASES OF KÂLA-ÂZAR.

Illustrative cases of *kála-ázar*.

The fulness of the previous clinical description of the disease renders it unnecessary to record the details of many cases, especially as Dr. Giles gives in his report extracts of the notes of thirty, which had been recorded by several of the Civil Surgeons of the province, and two, which he himself diagnosed as *kála-ázar*. A fairly accurate notion of the disease will, I think, be best conveyed by brief notes of a typical group of

cases, such as that depicted in the photograph opposite this page, which included all the cases in the Nowgong dispensary at the time it was taken, namely, early in August 1896. These cases were followed up, as long as possible, usually till death put an end to their miserable existence. It must be remembered that the class of cases seen in a dispensary consists, for the most part, of the more chronic type.

Case 1.—(First from the left in the group). Nankee, male, aged 30, a native of Bhagalpur, Bengal, but has been in Assam for the last seven years. He came to live in Nowgong in June 1895.

Family history.—He has no relatives in Assam, but one of his companions died of *kála-ázar*. He began to suffer from fever in December last, at first every day, and with a few days' remission now-and-then it lasted up to the time of his admission on 28th April 1896. He had some hæmorrhage from the nose last December; has never taken opium, and takes very little alcohol. Diet good.

History of illness.

(Notes taken on June 2nd, 1896).—He is thinner than he used to be; conjunctiva pale and slightly yellow, but the vessels are still visible; mucous membrane of the mouth pale; palate pigmented; vision good; retinæ normal; sensation normal; no pains in limbs; appetite not good; bowels irregular; has never suffered from sickness. Temperature 104·8°F. at 8 a.m. During May it had been normal, except in the last week, when it varied between 101° and 102°. Pulse 128 a minute; artery full, but very soft. Heart dulness normal; apex beat feeble; loud systolic murmur at the pulmonary cartilage, and a soft one at the apex. Lungs normal. Liver dulness extends from the fifth rib to the costal margin; its edge can be felt, and is rather hard. Spleen extends to one finger's breadth below the navel and to the middle line; over its upper anterior border is a prominent rounded swelling, which is well seen in the photograph, and which I thought might be an abscess in the organ. No dropsy of abdomen, legs, or face. Urine:—sp. gr. 1,006;

Present condition.

colour dark straw ; no albumen ; a little phosphates ; reaction neutral. Thymol had been given, but no worms were found, and, on examination under the microscope, no ova of anchylostoma, or of other worms, were seen, but some undigested starch granules were detected. The plasmodia malariæ were found in his blood, taken when his temperature was high. Examination on June 2nd showed hæmoglobin 30 per cent., red corpuscles 2,240,000 per cubic millimetre ; white corpuscles 1,000 per cubic millimetre, or 1 to 2,240 red, and the hæmoglobin value, or amount of hæmoglobin in each red corpuscle, as compared with the normal,. which is taken as being unity, was ·67.

Sub-sequent course of the case.

His temperature continued to vary between normal and 105°F. in a very irregular way until June 27th, when it became normal for a week. On July 1st, he was noticed to be more anæmic, and his feet were swollen. At this date his hæmoglobin was found to be only 22 per cent., and his red corpuscles numbered 1,700,000, showing a considerable decrease since the last examination. During the next fortnight, there was very little fever, the temperature having only twice ran up to 101°F., and on the 20th of July, the hæmoglobin was 32 per cent., and the red corpuscles 2,770,000—a decided increase in correspondence with the decrease of the fever. He again relapsed, the temperature frequently running up to 103°F., and he got steadily worse, and his feet became swollen. The swelling over the spleen increased in size, and became very painful, so on August the 12th, I passed the middle of an aspirating syringe into the prominence, but did not obtain any pus. The blood was used to inoculate some *agar agar* tubes kindly supplied me by Professor D. D. Cunningham, but with negative results. He died of fever on the 9th of September.

Treatment.

In this and the following cases, except where otherwise stated, the treatment was carried on by the civil hospital authorities, as I desired to study the disease and to see the effects of the ordinary treat-

ment in which I seldom interfered in the earlier stages of my investigation, although I wish to acknowledge that I am much indebted to the Civil Medical Officer of Nowgong, Mr. McNaught, for kindly allowing me a perfectly free hand in trying any treatment I might desire. Unfortunately, my frequent and prolonged absence on tour prevented me from doing much in this line. In this case quinine in small doses, usually combined with arsenic, was the chief medicine given.

Post-mortem examination.

Body much wasted. Feet and legs œdematous. Pericardium contained 3½ oz. of clear fluid. Heart weighed 6 oz. 3 drm. Heart muscle somewhat pale, and showed signs of fatty degeneration. Cavities and valves normal. Left pleural cavity contained 3 oz. of fluid, and the right 1 oz. Both lungs were emphysematous at the edges, and congested and œdematous at the bases. The abdominal cavity contained 64 oz. of fluid. Stomach normal; duodenum normal, and without any anchylostoma; small intestine contained 8 anchylostoma, but its mucous membrane was normal in appearance, and presented no signs of cicatrices or of pigmentation. Mesenteric glands normal; large intestine showed a patch of congestion of the mucous membrane; liver weighed 3 lbs. 11½ oz.; its capsule was adherent to the neighbouring organs and to the parietal peritoneum; on section it showed red and white mottled patches, mostly near the surface, and it was easily broken down on pressure. Spleen weighed 3 lbs. 6 oz.; on its anterior border was a rounded swelling, which, on section, was found to be similar to the rest of the organ, which was of a dark-red colour, and broke down easily on pressure; no mark of the puncture was seen, but a little above where this must have passed was a thick band of adhesion binding the organ to the parietal peritoneum. Kidneys normal; weights 4 oz. and 3½ oz., respectively; suprarenal capsules normal; brain normal; pia-mater under medulla oblongata pigmented. Bone marrow of the shaft of the femur was of a dusky-red colour, and softer

than normal. Sections of the liver, spleen and kidneys showed well marked pigmentation under the microscope.

Remarks.

This is a typical case of the disease, and illustrates very well the dependence of the anæmia on the fever, and the absence of more than a very few anchylostoma from an early period of the illness. The fluid in the abdomen was evidently of an inflammatory nature, and the simulation of abscess of the spleen caused by an irregular swelling of the organ accompanied by pain due to perisplenitis is noteworthy. The demonstration of the malarial organism during life, and of pigmentation of the liver, etc., after death, completes the proof of the malarial nature of the disease from start to finish.

Case 2.—(Fourth from the left in the group). Nandi Keot, female, aged 22, Assamese, lives in Nowgong.

Examined on: Family history.

She has lost 11 near relatives in the last three years from *kála-ázar*, including six brothers, and has only five now living, one of whom has the disease.

History of illness.

She first got fever in November 1894; it began with shivering, and came on daily for twelve days. She came to the hospital, and stopped one month. She has had fever on and off since that time: some three or four attacks in all. She was free of fever for a month up to shortly before her re-admission on July 21st, 1896, with a relapse.

Condition on re-admission.

Her arms and face are rather thin; no dropsy; conjunctiva very pale and yellow; palate shows well marked pigmentation. Vision good; retinal arteries rather small, and retinæ pale, but no hæmorrhages in them. Temperature 102·4°F.; pulse of low tension. Heart normal, and no hæmic murmur in the neck. Appetite fair when she has no fever; bowels rather constipated; liver not enlarged. Spleen extends to the level of the navel and to the middle line, not very hard. Lungs normal.

Blood examination.

Hæmoglobin 24 per cent., red corpuscles 2,390,000, and white corpuscles 1,500 per cubic millimetre, or 1 white to 1,593 red; hæmoglobin value ·50.

Progress of the case.

The fever continued to be of an irregularly intermittent type, and at one time was tertian in its character for eight days, until the 25th of August, when there was a remission for a fortnight, which was accompanied by a temporary improvement in the condition of the blood, but this was followed by another relapse, which passed into a remittent form of fever, ending fatally on September 18th. On August 7th, the liver was found to extend to three fingers' breadth below the ribs, and on September 15th, the patient was found to weigh only 70 lbs.

During the last month she suffered on and off from diarrhœa, sometimes of a dysenteric nature, which, as I have noticed in some other cases, was worst when the fever stopped for a few days.

Treatment.

She was given arsenic and iron tonics and quinine usually in five-grain doses twice a day, and latterly various drugs were tried for the diarrhœa with very little success.

Post-mortem.

Body emaciated. Heart weighed 6 oz.; structure normal. Lungs congested at the bases. Stomach contained three round worms, otherwise normal. Small intestine contained one anchylostomum in the jejunum. The cæcum contained numerous trichocephali, and there were 18 round worms in the colon. Liver weighed 4 lbs. 2 oz., was firm, and on section was of red colour mottled with yellow. Spleen weighed 3 lbs.; on section was hard. Bone marrow of shaft of femur darkish-red in colour.

Remarks.

Here, again, the dependence of the disease on the fever is very evident, but probably the first attack noted in November 1894 was one of ordinary malarial fever, and the fatal disease began later. While in hospital, with the exception of a fortnight's

remission, the fever was constant, and the small doses of quinine which were given had very little, if any, effect on it. The attack of diarrhœa doubtless hastened her death.

Case. 3—(The small child in the middle of the photograph). Senalie Kachari, female, aged 5.

Family history. Father, mother, and a brother died of *kála-ázar*; and as she has no relatives to look after her, she came into hospital.

History of illness. She has been suffering from fever with enlargement of the spleen for six months.

Present condition. Is much emaciated; appetite fair; bowels inclined to be loose; spleen reaches three fingers' breadth below the ribs and nearly to the middle line.

Blood examination. Hæmoglobin 24 per cent., red corpuscles 2,060,000, and white 2,250, per cubic millimetre, or 1 to 916 red; hæmoglobin value ·58.

Progress of the case. She continued to get fever of an irregularly remittent type, usually rising in the evening to between 100° and 102°F., but occasionally running up to nearly 104°F., which continued up to the time of her death on September 20th.

Treatment. Treatment given was a mixture of arsenic, nux vomica, and quassia, and during a part of the time quinine in two-grain doses twice a day.

Post-mortem. Body much emaciated, and the feet œdematous. Pericardium contained 6 drms. of clear fluid; heart and lungs normal. Peritoneum contained 6 oz. of clear fluid. Stomach normal. Small intestine contained 15 anchylostoma and 20 round worms. (Santonin had been given on admission, but not thymol, owing to her bad condition.) In the large intestines, there were several ulcerations. Liver weighed 12½ oz. The spleen weighed 4 oz., and was hard and dark red. There was some fluid in the ventricles of the brain, and the bone marrow was dark red.

Remarks.

This child was in an almost hopeless condition on admission, and it is extraordinary that she lived as long as she did. Fever of a persistent type was again the marked feature of the case and the cause of death. Thymol would only have removed the small number of worms present in this case, at the cost of setting up a rapidly fatal diarrhœa.

The three patients on the right of the photograph form a characteristic family group, the man and the woman being the father and mother, respectively, of the baby, who is the youngest case of the disease that I have met with, being only one year old, and was still at the breast, although other food was given to him. They are also of interest, as showing how patients, who appear to be doing fairly well, often get fatal lung affections at the onset of the cold weather. Brief notes only need be given of them.

Case 4.—(The man on the right of the group). Patua, male, aged 42, Kachari, admitted to hospital 4th August.

Family history.

His brother and one daughter have died of *kála-ázar*; no near relative now living is free from the disease.

History of illness.

Two years ago he had fever for five months, but has had very little for the last year; he still gets it every now-and-then, but has none at present. He is slightly anæmic only, and is fairly well nourished. He has no dropsy, but had suffered from both it and epistaxis formerly, and is now in every way much better than he has been. Heart normal. Spleen extends to one finger above the navel, and three to the right of the middle line. Liver reaches to one finger's breadth above the navel, and is hard. Bowels irregular and inclined to be loose. He took opium for one month after he got ill in the hope of its stopping the fever, but without any result. Hæmoglobin 38 per cent., and red corpuscles 2,820,000, white corpuscles 4,000, or 1 to 705 red, sp. gr. of the blood 1·057; hæmoglobin value ·67. Thymol was given, and several round

worms were passed, but no anchylostoma could be found after washing the stool, etc. He remained free from fever, and seemed on the high road to recovery, until on the 1st of October, he got an attack of pneumonia, and died in three days.

Post-mortem. Body stout; no œdema. Heart normal, but right side distended with blood. The upper half of the left lung was consolidated, and sank in water; the pleura over it was adherent to the chest wall. Stomach and duodenum normal; jejunum and ilium were pigmented, no worms of any sort present. Liver weighed 6 lbs. 15 oz., and the spleen 2 lbs. 13 oz.; both organs harder than normal. Bone marrow of shaft of femur was of a yellowish-red colour.

Remarks. This man I regarded as nearly recovered when he came to hospital; and if he had escaped the lung complications of the early cold weather, would have probably got quite well, so that his case illustrates the necessity of caution in saying that a given case is cured, and it is, for this reason, that the tea garden series are of such value in proving the very high death-rate of the disease, for they were under continuous observation for years.

Case 5.—Kundari, female, aged 23, wife of Case 4. (Second from the right in the photo.) Admitted to hospital on August 4th, 1896.

Family history. Same as Case 4.

History of illness. She began to get fever nine months ago, which continued for three months; but after one month her spleen was enlarged. After this she had a remission for a time, but it came on again in the rainy season, and has continued on and off ever since. Bowels somewhat loose. She is anæmic; her spleen extends to the level of the anterior superior spine of the ilium, and two fingers' breadth to the right of the middle line, and the liver from the level of the fifth rib to four fingers below the costal margin,

both organs being hard. There is no dropsy, and she has never had any; she suffers from pain in the legs, mostly along the shafts of the tibia and femur. No enlargement of the surface veins of the abdomen.

Blood examination. Hæmoglobin 25 per cent., red corpuscles 1,750,000, white 2,000, or 1 to 875 red, sp. gr. 1·051; hæmoglobin value ·71.

Progress of the case. During the whole of August and the first half of September, she had only slight fever running up to between 100° and 101°F., and was treated with tonics and quinine in from 3 to 6-grain doses. During the last part of September and the first week of October, she had high remittent fever running up to over 104°F., and accompanied by diarrhœa, which weakened her very much, and shortly after she developed pneumonia, which went on to gangrene of the lung, and terminated fatally on October 18th. During the latter period quinine in 10-grain doses was given, while various remedies were tried to control the diarrhœa with very little effect. The external application of creasote seems to have had most effect in reducing the fever in this case.

Post-mortem. Body much emaciated; no œdema present. Pericardium contained 4 oz. of clear fluid; heart normal; upper lobe of left lung gangrenous; lower lobe congested; right lung healthy; stomach normal; small intestine contained 10 anchylostoma and 2 round worms; its mucous membrane was thin, and in places congested, but not pigmented; large intestine thickened and congested, but without any ulceration; liver weighed $3\frac{1}{2}$ lbs., and was hard on section; spleen weighed 1 lb., dark red, hard on section; bone marrow reddish-yellow in colour.

Remarks. This woman, in spite of the great size of her liver and spleen, did not appear to be by any means a hopeless case when she came into hospital, and did well for the first six weeks until the fever relapsed; and in the course of a month's high fever and

diarrhœa, she rapidly became very emaciated, and died from lung complications. The comparatively small weight of the spleen and liver found on *post-mortem*, was due to a rapid reduction in their size, which took place during the time she suffered from diarrhœa, as I have also seen happen in other cases.

Case 6.—Aghonu, aged 1 year, son of cases 4 and 5. He began to get fever three months ago, and has had it on and off ever since up to the time of his admission. Rather thin, and anæmic. Spleen extends to the level of the navel and to the middle line, and the liver nearly to the level of the navel. Bowels loose; temperature 102·4°F. Santonin was given, and seven round worms, but no anchylostoma, were passed. The malarial organisms were found in his blood when he had fever.

Progress of the case.

During most of August, low fever continued on and off; but in September, except for one week, when it was of a remittent type, it was absent; and during October there was no fever, but a slightly subnormal temperature was recorded, the child being much better. This improvement followed the use of quinine in three-grain doses three times a day, which is a large dose for an infant. Late in October, when the child had much improved, he was attacked by cholera, with a fatal result on October 29th. No *post-mortem* was done.

This is the youngest case of the disease that I have met with, although it is not at all uncommon in children under 10 years old. The use of relatively large doses of quinine was very beneficial in this case, and, but for the cholera, might have saved the child's life.

Case 7.—(Second man from the left in the group). Badal, male, aged 25, lives at Jaumpur, and has only been in Assam four months, three of which he spent in Gauhati and one in Nowgong. He has no relations in Assam. He had an attack of fever on the way up to Assam; and soon after he reached Gauhati, he began to get fever every other day,

which went on for two months, by which time his spleen was enlarged. He was admitted on May 29th, at which time he was getting fever daily.

Condition on admission.

He is thin and wasted, and says he is darker than he used to be; conjunctiva yellow, but not pale; buccal mucous membrane also of a good colour; no œdema; had epistaxis about two months ago; has had pain over his spleen for the last five days; heart sounds feeble. Spleen extends to level of the navel, and is hard; liver edge can be felt one finger's breadth below the right costal margin. Retina normal. No anchylostoma ova could be found in his fæces. Over the base of the left lung there was dulness, and crepitations could be heard. This was the beginning of an attack of pneumonia, which began on June 3rd, but he got over it, and from this time he lost his fever, and during the three months he subsequently stayed in hospital, he steadily improved, and his blood, which showed 50 per cent. of hæmoglobin and 57 per cent. of red corpuscles, also improved with his general condition, while both his spleen and liver became reduced in size, and he left the hospital fat, and well in the middle of September. I have also seen another case, who lost all fever and steadily improved in the same way, after getting over an attack of pneumonia. The malarial organisms were found in his blood during fever.

Case 8.—(Third from the left in the group). Khalai, male, aged 22. His father, mother, and an uncle died of *kála-ázar*, and he has no near relatives left alive. Has had fever for five months, and is thin and anæmic, but has never had any dropsy; bowels regular; spleen extends to the level of navel and one finger beyond the middle line, and the liver can just be felt below the ribs; heart sounds feeble; lungs normal; has pain in the legs.

Blood examination.

Hæmoglobin 40 per cent., red corpuscles 2,900,000, sp. gr. 1·045; hæmoglobin value ·69.

Thymol was given, and nine anchylostoma were passed.

Progress of the case.

He had a short attack of remittent fever just after his admission, that is, in the middle of June, and another with high fever, accompanied by looseness of the bowels, a month later. During the first half of August, he again had high and nearly constant fever running up to 100° and 105°F. Up to this time he had been taking an arsenic mixture, with quinine in two to four-grain doses, with an occasional ten-grain dose. He was now put on larger doses, namely, 10 to 15 grains, two or three times a day, and subsequently he only had slight fever up to 100° or 101°F., with intervals of a week or two free from fever. He improved a good deal during this time, and left the hospital on October 10th, much better, but still getting fever every now-and-then. He cannot be considered cured, but if he escapes lung complications, he will probably recover his health during the cold weather.

The two girls to the right of the central child in the group, are of great interest from the point of view of treatment by bone marrow tabloids, for, if the result obtained in one of them is confirmed by subsequent experience, this will be of the greatest value in saving chronic cases, who have to a great extent lost their fever, but remain in a wasted condition in spite of nourishing diet until they succumb to lung and bowel complications.

Case 9.—(Third adult from the right in the group). Maharsani, female, aged 15, admitted July 28th.

Her father, mother, and two brothers died of *kála-ázar*, and she has only one brother and two sisters living.

Family history.

History of illness.

She has had fever on and off for one year.

Present condition.

She is weak and very emaciated ; no dropsy ; the spleen extends to one finger's breadth below the navel, and nearly to the middle line, and the liver to four fingers below the ribs, and its edge is

hard. Conjunctiva pale and slightly yellow. There is a pulmonary systolic murmur to be heard over the third right costal cartilage, but no venous hum in the neck. Bowels regular.

Blood examination.

On August 13th, hæmoglobin 37 per cent., red corpuscles 3,010,000, white 2,000, or 1 to 1,505 red, sp. gr. 10·52; hæmoglobin value ·61. Santonin was given, and some round worms only were passed.

The progress of the case.

From the beginning of August to the 11th of September, there was almost daily intermittent fever rising to 100° or 102°F. From this date she nad a remission of three weeks, during which bone marrow tabloids, one twice a day, were given. Her weight was 68½ lbs. at the beginning of this treatment, and the same after three weeks, so that no change had resulted. A relapse of the fever now occurred, and the tabloids were left off. She had been taking arsenic and small doses of quinine when not taking the tabloids. As I was away on tour at this time, I was not able to record the effect on the blood of the treatment. Early in October, she had a short attack of remittent fever, and quinine was now given in 10-grain does three times a day, and in two weeks she was free of fever, but was now very emaciated and reduced to 60 lbs. in weight. She was now given tabloids again for a short time, and began to improve, and remaining free of fever, became fat and well nourished. In February 1897, however, she got an attack of pneumonia and died rather suddenly with cyanosis. Here again, but for the lung complication, I feel sure she would have recovered, and that she would have owed this to the larger doses of quinine, and I think, also in some degree, to the bone marrow treatment, although its results were not so striking in this case as in the next one.

Post-mortem.

Body very well nourished, with a good layer of subcutaneous fat. The heart weighed 7 oz., and the right side of it was distended with blood. The base of the right lung was in a state

of red hepatisation, and was loosely adherent to the pleura. The base of the left lung was congested. The spleen weighed 1 lb. 6 oz., and was rather hard on section. The liver weighed 6 lbs. 2 oz., and was of a yellow colour, showing marked fatty changes. The mucous membrane of the stomach was thick and corrugated, but the small intestine was normal, and contained only two round worms, and no anchylostoma. The bone marrow was of a light-red colour, lighter than I have usually found it in cases dying in the stage of cachexia.

Remarks.

This case well shows from what a very bad condition these cases may sometimes greatly improve if the fever only ceases, and yet eventually succumb to complications.

Case 10.—(The girl on the right of the central child). Alagae, female, aged 17, admitted May 14th.

Family history.

Father, mother, one sister, and two young brothers died in one house of *kála-ázar*; one sister only living.

History of illness.

She has been ill for one year, and was in hospital in March and April 1896. Illness began with fever of an irregular type, which has continued with longer or shorter remissions ever since. She has never had any œdema of the feet or face, but her menses have stopped since she had fever.

Condition on re-admission.

She is very thin, especially in the arms and face, and her face is very dark. Her conjunctivæ and buccal mucous membrane are pale, and the former also yellow. Vision good, but the arteries of the retinæ are very small, but no hæmorrhages could be seen. Heart and lungs normal. Spleen extends to the level of the anterior superior spine of the ilium, and is rather tender, and the liver extends from the level of the fifth rib to two fingers' breadth below the costal margin, and is rather hard. Urine light straw colour, sp. gr. 1,010; no albumen or phosphates. No ova of any intestinal

worms could be found in the fæces, but particles of undigested starch were seen.

Blood examination.

Hæmoglobin 28 per cent., red corpuscles 2,180,000, white 2,330, or 1 to 948 red; hæmoglobin value ·64.

Progress of the case.

On May 17th, her temperature rose to 104·6,° but from this date she was free from fever until the beginning of July, and the condition of her blood steadily improved, the percentage of hæmoglobin rising to 33 on June 9th, and 36 on July 1st, and the size of the spleen and liver had also become somewhat reduced. During the whole of the first week in July, she had high fever of an irregularly intermittent type, running up to 104° and 105°F., and accompanied by slight looseness of the bowels, after which only slight fever was present, but on July 17th, her hæmoglobin was found to have become reduced to 26 per cent. From this time on, she only had an occasional rise of temperature to 100° or 102°F. On July 20th, her blood was as follows: Hæmoglobin 27 per cent., red corpuscles 2,370,000, white 2,500, or 1 to 1,260 red; hæmoglobin value ·57. On July 18th, she was put on two bone marrow tabloids every day, and this was continued with only three days' intermission until October 22nd, except that during three weeks of the time she only took one tabloid a day. The photo in which she is shown was taken soon after this treatment was commenced, and she was then extremely weak and emaciated, so much so that I looked on the treatment rather in the nature of a forlorn hope, especially as she had already been in hospital for over two months without any benefit, but rather the reverse. Three weeks later on my return from a tour, I was surprised to find her much better. She was distinctly less emaciated, and, on examining her blood on August 11th, it was as follows: Hæmoglobin 31 per cent., red corpuscles 2,330,000, white 3,750, or 1 to 620 red, sp. gr. 1·048; hæmoglobin value ·67. Now I have seen quite as great an improvement in the blood in as short a time from treatment with arsenic, etc., and, although I had given the

bone marrow in the hope of producing a more rapid improvement in the blood than by other means, it was the general improvement in the state of the patient that was its most marked effect, together with an increased appetite and power of digesting and absorbing food. On September 7th she was fat compared to what she had been, while her spleen now only reached to just below the navel, and her liver could not be felt. The blood on this date was hæmoglobin 38 per cent., red corpuscles 2,900,000, white 5,500, or 1 to 527 red; hæmoglobin value ·66. On September 14th, she weighed 77½ lbs., but a month later she had improved up to 90lbs. She continued to gain weight after the tabloids were left off, but somewhat more slowly, and was discharged cured on November 11th, and is now quite well, and has married.

Remarks.

This is by far the worst case in which I have seen recovery take place, and the rapidity with which marked improvement in nutrition occurred was in striking contrast to anything I have witnessed in other cases, for, as I have before remarked, a characteristic feature of the disease is the way in which the patients remain in an emaciated state long after the fever has left them, and the frequency with which such cases fall a victim to lung and bowel complications. Unfortunately I could not continue my trials of this treatment owing to all the tabloids I could get in India having been exhausted; but Dr. Dodds Price and Dr. Lavertine are now using it in the treatment of *kála-ázar* cases, and are so far well satisfied with its effects, and we may hope to hear shortly if my results are confirmed or not. The only explanation that I can think of to account for the improvement of nutrition from the use of bone marrow tabloids, is one which assumes that the yellow marrow in the shafts of long bones has the function of secreting some substance, which affects the digestive powers and nutrition in a somewhat similar way to the action of the internal secretions of the thyroid or suprarenal capsules, and that the changes found in the bone marrow in

kála-ázar cause a loss of this secretion, the place of which is, however, supplied by the bone marrow tabloids. The relation of the deterioration and improvement in the blood to the presence or absence of fever, is again well illustrated in this case, while the family history is characteristic.

The following case, with the accompanying temperature chart, illustrates very well the extreme irregularity of the type of fever in *kála-ázar*:

Case 11.—Kamar Lalong, Kachari, aged 22, was a cultivator who lived 14 miles from Nowgong.

Family history.

He has lost his father, mother, and brother of the disease, and has no near relatives living. He came into jail on October 8th, 1895, but had suffered from fever before that time. While in jail he was in hospital for one week at the end of January with fever, and on April 16th, was re-admitted for the same disease, and has had fever on and off since that time up to the 25th of May, when I examined him, and he continued to suffer from fever up to the day of his death on July 14th, as shown in the chart. This attack, while he was in jail, was most likely a relapse of the fever from which he had suffered before admission, but it must be mentioned that there were two other cases of *kála-ázar* in the jail hospital at the same time that he was there.

His condition on May 25th was as follows:—He is somewhat wasted in appearance and dark in colour. He says he is darker than he used to be. Heart dulness is diminished; arteries small and pulse feeble. No dropsy. Spleen extends to just above the navel, but the liver dulness is normal. Appetite fair, and bowels regular.

Blood examination.

Hæmoglobin 43 per cent., red corpuscles 2,700,000. Malarial organisms were found in his blood this day. An ovum of the anchylostomum was found in his fæces after a long search, but on giving thymol, only two of the worms were passed. He had never had thymol before.

Progress of the case.

Quinine and arsenic in small doses, with occasional single doses of from ten to twenty grains of quinine, or of five grains of antifebrin, were given, but the fever continued; his hæmoglobin ran down to 26 per cent., and his white corpuscles to 1 to 1,750 red; the liver became enlarged until it reached to two fingers' breadth below the ribs; the malarial organisms were again found in his blood on June 8th, and also when his temperature was high on the day before his death, which was due solely to the fever, and *post-mortem* pigmentation of the liver, spleen, and kidneys was detected under the microscope, so that it is clear that the case was one of malarial fever from first to last.

One more case will be given, which came under my treatment in the earliest possible stage, and which illustrates in a nutshell the pathology of the disease and the proper line of treatment:

Case 12.—Kondor, male, aged 11, but looking only 8 or 9, was brought to me by his father, a Babu holding a high position in the Education Department, who said that he had lost 39 relatives of *kála-ázar* within the last six years, including five this year. This son had only suffered from fever for the last four days, and showed none of the signs of malarial cachexia. His spleen was normal, and his liver was just felt below the edge of the ribs, and was tender; temperature 103°F., pulse 118, and respirations 30 a minute. Lungs normal. His blood was examined, and he was found to have 61 per cent. of hæmoglobin, and 4,030,000 red corpuscles, and 23,000 white corpuscles per cubic millimetre, or 1 white to 174 red. Hæmoglobin value ·76. From this it is evident that his blood was just about the standard, which I have found in healthy natives of Assam in the rainy season, while the great excess of white corpuscles is evidence of very active leucocytosis, such as is always met with in early acute cases of malarial fever, but never in the chronic stage of *kála-ázar*. It is evident then that his father's statement, that he had only suffered from fever for a few days, was confirmed by the state

of the blood. The malarial organisms were also found in a specimen taken on this date.

Progress of the case.

I ordered quinine to be given in five-grain doses three times a day, and I saw the boy again on July 15th. Meanwhile a thermometer had been obtained, and his temperature recorded by his father every few hours. On working out a chart from this, it appeared that the fever was of an extremely irregular remittent type, such as is so very common in the earlier stages of *kála-ázar*. Moreover, the temperature had only once risen to 103°F., and on this day the father said that he had omitted the quinine for the first time. It was then evident that the drug had served to keep the temperature lower than it was when it was not being taken, yet had not sufficed to stop the fever. It was, therefore, increased to 20 grains in the day—a considerable dose for so small a boy—and three days later the fever stopped. I saw the boy for the third time on July 25th, and he had now been free from fever for a week after having suffered from it for just over two weeks. To all appearances, he was perfectly well, and his father regarded him as being so. He had no enlargement of the spleen or liver, nor did he show the smallest trace of anæmia in his mucous membranes. To look at the boy, one would suppose he was none the worse for the fortnight's fever from which he had suffered, yet on re-examining his blood on this date, it was found that his hæmoglobin had become reduced to 46 per cent.—a loss of just one quarter in three weeks. It was then obvious that if this boy left off all treatment, and in the course of a few weeks got a relapse of the fever before his blood had again reached the normal, he would rapidly lose another quarter of his hæmoglobin, would develope an enlarged spleen and liver and anæmia, and would become a typical case of *kála-ázar*. On the other hand, I argued, if only the fever could be kept off until his blood had been fully repaired, a second attack treated in the same way should leave him no worse than he was after the first. I therefore got him to take a mixture containing

small doses of quinine, combined with arsenic, nux vomica, and quassia, and he continued this for six weeks, by which time I found his hæmoglobin had improved up to 59 per cent. He had no fever in the meantime, and he has been under my observation now and then up to the end of February 1897, and was then still quite well.

Remarks.

Now it is unfortunately impossible to prove that this was really an early case of *kála-ázar*, but from the strong family history, the irregularly remittent character of the fever, its resistance to ordinary doses of quinine, and the rapidity with which the blood was destroyed by it, I feel morally certain that it was so, and that the boy was saved from a lingering and almost certainly fatal illness by the prompt, vigorous and continuous line of treatment adopted.

THE TREATMENT OF KÁLA-ÁZAR.

It has already been mentioned that I have had very little opportunity for systematic treatment of *kála-ázar* cases, especially after I had obtained definite proof of its malarial nature, so that much of what follows is rather of the nature of suggestions for treatment than dogmatic rules regarding it. It is also evident from the fact that the disease is entirely malarial in its nature, that no new cure for it could be expected to be discovered, but the recognition of this fact, and more still of its extreme intensity and resistance to ordinary doses of anti-malarious remedies, is of the first importance in deciding on the therapeutical measures, which are likely to afford the best results. Some information of interest and practical importance has, however, been obtained from the experience of those who have recently treated many cases throughout their long illnesses on some of the tea gardens of Nowgong, and from my own more limited observations.

It will be not only convenient, but necessary, to divide the disease up into early and late stages in discussing the treatment ; the latter including all cases which have developed

such symptoms of malarial cachexia as greatly enlarged spleens and livers, and together with emaciation, loss of digestive power, and irregularity of the bowels; while the former includes cases of early fever before they have reached this stage. It may be at once objected that the early class of cases are not distinguishable from ordinary malarial fever, and, individually considered, this is quite true. It has been shown earlier in the present section that, wherever *kála-ázar* is prevalent, there will be an excess of cases of apparently ordinary fever, some of which will really be early cases of true *kála-ázar*. It is just in this stage that efficient treatment may have a chance of curing at least some of those which, if neglected, would soon run on into the intractable chronic form. They may be caught in the early stage in one of two ways, either by their occurring in families in which well-marked cases of *kála-ázar* have been, or are, present, or by all cases of fever in any place which is affected by *kála-ázar*, which do not readily yield to ordinary treatment, being looked on as suspicious and treated in the more radical ways which are about to be described.

Treatment in the early stages.

The principles for the treatment in the early stages are very simple, but require to be fully carried out if success is to be obtained. The last case given illustrates them very well. They are (1) to get sufficient quinine into the system to check the fever which is present; and (2) to prevent any return of the fever, while the injury which has been done by it to the blood is being repaired, the latter being hastened by all available means. If this can be done before fever recurs, it is obvious that the system will be in as good a position to meet a relapse of the fever as it was at the first onset, and if a relapse should occur, the same treatment must be carried out again.

In carrying out the first of these indications, quinine will be our sheet anchor. Now, it must be admitted that this drug has fallen somewhat into disrepute in the treatment of

kála-ázar, but the reason is not far to seek. It has been repeatedly pointed out that this form of fever is of such unusual intensity that it produces a degree of malarial cachexia in about as many months as the malarial fever of the Sylhet valley will take years to bring about, although the latter is practically part of the huge swamp of Lower Bengal. It is then at once obvious, that the doses of quinine that suffice for the treatment of the ordinary malarial fevers of Assam, cannot be expected to have any controlling influence on that of the epidemic fever, which is now travelling up the Assam valley. Yet I found that in several of the dispensaries of the Nowgong district, quinine was being given in two and three-grain doses twice or thrice a day, and that it was rarely that more than enough for one or two days was given at a time. Now, although there are a large number of dispensaries in this district, yet 11 in 3,257 square miles still leaves the great proportion of the inhabitants of the district at least several miles from any of them, and it is obviously impossible for people, sick of this terrible disease, to walk or send several miles to a dispensary every day, or even every other day, for medicine. Even if they did, they would very soon find that such doses did them little or no good, and would leave off attending. If it is also remembered that a native has a great tendency to wait until he has reached advanced stage of the disease before coming to hospital, is it at all surprising that such doses of quinine should have been found to have little apparent beneficial effect in cases of an intense epidemic malarial fever like *kála-ázar?* That it is of some use, even in small doses, is evident from the fact that it is universally employed in the treatment of the disease, and from the observation that I have found it lessen the height of the temperature, and also the destruction of the blood corpuscles in some cases in which it did not entirely stop the fever, while I have already given instances in the foregoing illustrative cases of the action of larger doses in controlling, or even stopping, the fever altogether. I have seen several cases in which an increase of the

dose of quinine from two or three to ten or more grains three times a day has been followed by a rapid cessation of the fever, at least for a time. On the other hand, it is equally certain that such large doses, as 10 or even 20 grains three times a day, may fail to stop the fever in some of the worst cases, and I find that in these the proportion of the white corpuscles is usually very much reduced, and often also the power of digestion and absorption by the intestinal tract, including possible even such drugs as quinine, is very much diminished. That the latter is not, however, the whole explanation, is proved by the fact that I recently gave 15 grains of quinine daily, by the hypodermic method, to a boy of ten years old in the chronic stage of the disease, without stopping the fever; and what was even more remarkable was the fact that he never suffered from the least buzzing in the ears or from headache, or was in any way inconvenienced by the treatment, with the exception of some pain over the seat of the injections. This alone affords strong confirmation of the malarious nature of the disease, as anyone who has treated many cases of non-malarial remittent or continued fevers, knows that much distress may follow the use of even ordinary doses of quinine in them. To sum up, it may be said that in the earlier stages large doses of quinine may be given with the greatest advantage; but in the later ones it may fail to check the fever even when given in heroic quantities.

Having thus cleared the ground, it may be stated that the first indication is to give quinine in full doses, and if it does not control the fever rapidly, push it until it does, or until symptoms of cinchonism appear, which I have never yet seen occur in *kála-ázar*. If very full doses still fail to stop the fever, then I hold that enough of the malarial antidote cannot be introduced into the system through the digestive tract, and hence the drug must be given hypodermically; just as in some of the most acute cases of the Italian, and also of the Indian jungle, fevers, if the drug is not thus given, the patient will be lost before enough can be introduced by the mouth to inhibit or destroy

the malarial organisms before they kill the patient. The only instance that I know of in which this method of using the drug has been given any trial in *kála-ázar*, is by Dr. Dodds Price, and he did not meet with much success with it; but when it is mentioned that he only used it in two and a half-grain doses, it will be evident that there is still scope for a fuller trial of it in larger doses than this. Unfortunately, owing partly to my having been on tour nearly the whole of the cold weather, and to the impossibility I found of getting early acute cases during this season of the year, when, as has been before mentioned, fresh cases seldom occur, I have not been able to test this line of treatment, so can only commend it for trial as being that which is likely to give the best results. Either the soluble bi-sulphate of quinine may be used, or the ordinary drug dissolved in sufficient dilute hydrochloric acid, and then diluted to a strength of one in four may be injected into the back or loins.

(2) When once the fever has been stopped, it must not be lost sight of that the patient will most likely have had 25 per cent. or more of his blood destroyed by it, although this may not appear in the state of his mucous membranes, and can only be revealed by the hæmoglobinometer. He must then remain under treatment until this has been repaired, and now quinine, in small doses to ward off a recurrence of the fever, combined with arsenic and, if desired, some iron (although the former is of much greater use in the anæmia due to malaria than the latter, for reasons which will be pointed out in the next section), together with some nux vomica and some bitter tonic to strengthen the heart and digestion (both of which also will have suffered in the previous fever), will be indicated, and this tonic should be continued for at least a month if the fever has been at all severe. By this time the patient should be quite restored to health; and if he be warned to come again directly fever recommences, it should be possible to prevent him from ever reaching that chronic condition which is so very difficult to rescue him from. If only cases

could be got at in the early stage and treatment systematically carried out on these lines, I feel sure the disease would have a very much lower mortality than it has at present.

Treatment in the later stages.

Unfortunately, cases are very rarely seen in the early stage in dispensaries, and only too often on tea gardens, they do not attend sufficiently regularly, or are treated as ordinary fever cases until they pass insensibly into the chronic stage. It is in these latter places, however, that the line of treatment above laid down could best be given a fair trial ; but even then some would necessarily be seen in the later stages of the disease. Here again, the principles of treatment are the same as before, but they hold out much less hope, and require some modification. Quinine should be given in large doses, and its effect carefully watched. It will nearly always be found to reduce the height of the fever, even when it does not stop it altogether, and this is a considerable gain; for it has been found that, if the temperature can be kept down to 100° or 102° F., there is a much less rapid deterioration of the blood than there is if it rises to 103° or 104°F. If, however, large doses for ten days or a fortnight fail to materially check the fever, it will be better to give smaller doses combined with arsenic and strychnine ; and in this chronic stage, the best results have been obtained by pushing the arsenic until thirty minims are taken in the day. In doing so, looseness of the bowels must be carefully watched for, as this symptom will be a contra-indication to this line of treatment. If looseness of the bowels has already started, the case will be a well nigh hopeless one, but full doses of bismuth and salol have more effect than any other drugs in controlling it, while a simple and easily digested diet will be essential. In the class of cases in which, after the fever has stopped for the time being, the patient remains very wasted, and does not appear to be able to digest his food, or to gain flesh, bone marrow in

tabloids will be indicated, and will be of the greatest service in restoring the nutrition of the body if only fever remains in abeyance. It will, however, be of little use as long as there is fever of a marked degree, but a temperature running up to 101°F., or so occasionally, will not be a contra-indication to its use.

A nutritious and easily digested diet will be of the first importance, especially in the later stages of the disease. The craving for meat has already been mentioned, and should, when possible, be satisfied. All indigestible food should be prohibited, as it is very likely to start uncontrollable diarrhœa.

From what has been written, it will be evident that I look upon the disease in its earliest stages as curable by sufficiently vigorous and constant anti-malarial treatment ; but that it is rarely that cases are seen early and often enough to allow of this being carried out, even in in-door dispensaries, while it is almost impossible in the case of out-door ones, although much relief is doubtless given by them ; while no known treatment will save more than a small proportion of the chronic cases. The disease is then essentially one in which prevention is better than cure, and in which any measures which would limit in the smallest degree the prevalence and spread of the disease, will save infinitely more lives than all the dispensaries in the province put together.

SECTION IV.

THE BLOOD CHANGES IN *KÂLA-ÂZAR* AND ANCHYLOSTOMIASIS.

Although anæmia is the essential symptom of anchylostomiasis, and one of the main symptoms of *kála-ázar*, yet the changes in the blood might be expected to be different in the two cases, because the anæmia is produced in entirely different ways in the two diseases. Thus, in the case of the former disease, the anæmia is brought about by the sucking of the blood out of the mucous membrane of the small intestine by a large number, usually from 500 to 1,000, or even 3,000, of very small worms, which gain access to the intestinal tract in the water and food, but cannot increase in number while in the body. In the case of *kála-ázar*, on the other hand, as has been shown in Section III, page 45, the anæmia is in direct proportion to the fever, and is doubtless due to the blood being destroyed inside the body by the malarial organisms, which cause the fever. In the former case, all the constituents of the blood are equally lost to the economy, although they cannot all be replaced with the same rapidity. In the latter case, the corpuscular elements of the blood only are destroyed, while the albuminous fluid in which they float is not directly affected, and what is of even more importance, the hæmoglobin, or coloring element, is not lost to the system, but, as will be proved in Section V, is stored up in the liver, spleen, etc., and is available for stocking fresh red corpuscles, as these are renewed by the recuperative powers of the tissues.

I could, however, find very little in the way of examinations of the blood in such of the literature of anchylostomiasis and malarial fevers as was available to me, so I determined to make as complete an examination of it as possible in these two diseases, and it was hoped that in this way it might be determined in what respects the blood changes differed in the two diseases, and if the differences were sufficiently marked

and characteristic to be of diagnostic value. For this purpose the hæmoglobin was estimated by Dr. Gower's instrument, and the red and white corpuscles counted by the hæmocytometer of the same scientist, while in the later cases the specific gravity of the blood was determined by the accurate instrument of Dr. Lloyd Jones, as soon as this was obtained from England. The coagulability of the blood was also determined in a few cases by means of the instrument of Professor Wright, of Netley. The results exceeded my most sanguine expectations, and proved conclusively that the anæmia of anchylostomiasis differs from that of *kála-ázar* in every way it well could, while, on the other hand, that of *kála-ázar* agrees in every detail with that of ordinary chronic malaria, as met with in districts which were unaffected by *kála-ázar*, for, as has been repeatedly said, this latter disease cannot be distinguished from ordinary chronic malaria in the affected districts. The evidence on which these statements are based must be given in this section, and, although the subject is a very complicated and technical one, an endeavour will be made to discuss it as lucidly as possible. The blood of healthy natives of Assam must first be described, as it has been found to differ from the European standard, especially in the case of the amount of hæmoglobin, which is much lower in them than the standard of Dr. Gower's instrument. Then the changes in the case of *kála-ázar*, ordinary chronic malaria, and anchylostomiasis, respectively, will be given, and lastly the explanation of the differences will be pointed out.

The blood of healthy Assamese.

In estimating the standard of the blood in the healthy inhabitants of Assam, it was necessary to exclude both those who had any number of anchylostoma in their intestines, and also those who had had any material amount of fever during the last two years, for, as will be shown presently, both of these classes, the latter to a much greater degree than the former, had somewhat inferior blood. Now it must be constantly borne in mind that the presence of anchylostoma in the intestines of

of a man does not prove that he is suffering from anchylostomiasis, for they must have done some definite injury before the man can be said to be suffering from disease caused by the presence of the worms, which is what is meant by the term. It requires the presence of at least 500 anchylostoma for a space of from six months to one year in a healthy person to produce marked anæmia, but a smaller number than this would doubtless suffice to bring about alterations in the standard of the blood sufficiently great to be indicated by the delicate instruments which have been used in this research. It was then of great importance, especially in view of the frequent presence of the worms in cases of *kála-ázar*, to determine what number of worms could be present without causing any blood changes, for it is obvious that the system which can withstand for even a few months the attack of two or even three thousand of these worms, must be able to so completely repair the loss caused by only a small number of them, that no changes will appear in the blood. To test these points, thymol was given to a series of 50 healthy men in the Nowgong jail, and the blood of many of them was also minutely examined. The results were as follows: taking the hæmoglobin to represent the standard of the blood, for this is its most essential element, and also that which is least easily replaced, and consequently soonest becomes reduced when there is any drain upon it, it was found that the average amount of this constituent, according to the scale of Dr. Gower's instrument, of seven men, who had no anchylostoma, was 57·28 per cent., while that of 17 cases, who had anchylostoma in numbers varying from 1 to 20, was 57·7, so that it is obvious that these small numbers had no effect on the blood whatever. Again, two men, one of whom had one anchylostomum and 42 flukes, and the other 110 anchylostoma and 104 flukes (amphistomum hominis), each had 57 per cent. of hæmoglobin, so that even considerable numbers may be present, and yet, at least for a time, the drain of blood produced by them may be completely repaired. On the other

hand, one man, out of whom 293 anchylostoma were obtained, had only 42 per cent., although he did not appear clinically to be at all anæmic. Again, the average amount of hæmoglobin in 16 men, who had not suffered from more than one week's fever in the last two years, was 60·8 per cent., while that of 8 cases, who had had more than a week's fever in the same time, was only 52·7, so that it is evident that comparatively slight malarial fever, especially if it recurs at intervals, is a much commoner and more important factor in causing anæmia than is the presence of small numbers of anchylostoma. If then only cases who have not had more than a very few days' fever for two years, and who have not more than 20 anchylostoma, be taken as healthy men, we shall be on the safe side. The average amount of hæmoglobin in 14 such natives in the Nowgong jail in the rainy season was 62 per cent., while in 12 of them the average number of red corpuscles in a cubic millimetre was 4,734,000, which is very little below the European standard of five millions. It will be noticed, however, that the hæmoglobin falls very much below the standard of Dr. Gower's instrument, which is, I think, rather a high one, for I well remember testing the blood of myself and eight healthy students, when House Physician to St. Mary's Hospital in London, and finding the average to be only about 85 per cent., and that of seven healthy Europeans in Assam during the rainy season I found to be but 71 per cent. The higher rate of Europeans over natives is, I believe, due to the larger amount of animal food consumed by the former, which contains more iron than vegetable food, and that in a more assimilable form, for Ralph Stockman has shown that the diet of Europeans contains only from $\frac{1}{11}$ to $\frac{1}{8}$ of a grain of iron a day, and the deterioration of the blood is much greater in tropical than in temperate climates, and so the need of iron is also greater.

Quite recently, however, I found that several of these Europeans had from 8 to 10 per cent. more hæmoglobin towards the end of the cold weather than they had during

the previous rainy season, and probably the natives vary to some extent in the same way. This would partly explain the low average obtained; but as all the observations on which the figures given are based were made during the rainy season, their relative value is not altered by this fact.

It follows from the greater deficiency of hæmoglobin in proportion to the number of red corpuscles in the blood of natives, that the amount of hæmoglobin in each corpuscle must also be less than the normal standard, which may be represented by unity, and its amount may be estimated by simple rule of three. The figure for a healthy native of Assam thus obtained, which may best be termed the hæmoglobin value, works out to be ·65. The importance of this figure will be immediately seen. Again, the number of white corpuscles and their ratio to the red varies considerably in health, but we may take the European standard to be 6,000 to 7,000 per cubic millimetre, or 1 white to 600 to 700 red, in accordance with the authority of Hamilton. In eight healthy natives the same figures averaged 7,225, and 1 to 684, respectively, which agree with the European standard. Once more, the specific gravity of the blood of six healthy natives varied from 1,052 to 1,058, the average being 1,054·33. These figures may be taken then to represent the composition of the blood of healthy natives of Assam during the rainy season, and will serve as a standard of comparison with those met with in anchylostomiasis, chronic malaria, and *kála-ázar*.

The blood changes in *kála-ázar*.

It has already been mentioned that in *kála-ázar* anæmia is a constant and usually well-marked feature. It varies widely in its degree from quite a slight one, which is not evident in an ordinary clinical examination but is revealed by the hæmoglobinometer, registering only from 35 per cent. to 50 per cent., down to extreme degrees in which only from 12 per cent. to 20 per cent. is found. On the average, however, it is not so low as in anchylostomiasis, the average of 107 observations of the former disease (*kála-ázar*) being 33·45 per cent., while

that of 12 cases of the latter was 15·16 per cent., while Sandwith, of Cairo, found the average of 173 cases to be 26 per cent. The most important point, however, regarding the anæmia of *kála-ázar* is that in this disease the hæmoglobin and the number of the red corpuscles are equally, or nearly equally, reduced, so that the hæmoglobin value does not differ, as a rule, very much from the normal. Thus, the number of red corpuscles in 50 observations averaged 2,462,000 per cubic millimetre, or 49·24 per cent., and the hæmoglobin value works out to be ·65, which, it will be seen, exactly corresponds with what was found to be the normal figure for healthy natives of Assam.

The number of white corpuscles differs very widely in *kála-ázar* from the normal, but it is complicated by the occurrence of leucocytosis, or an excess of white corpuscles, actual or relative, during the presence of fever. In the very early stages well-marked leucocytosis occurs, just as in an attack of ordinary acute malarial fever, and I have seen the white corpuscles number 23,000 per c.m., or 1 to 174 red, in a case which I have very good reason for believing to be one of a very early *kála-ázar* (see page 74). In all well marked cases, on the other hand, during the absence of fever, the white corpuscles are very much reduced, there not unfrequently being only about 2,000, and, in spite of the red corpuscles also being greatly reduced, there may be only 1 white to over 2,000 red. The average number in 36 observations made during the absence of fever was 2,600 per c.m., or 1 to 1,170 red. Repeated estimations made in the same cases show, however, that during the occurrence of fever, especially if the temperature is over 102°F., the white corpuscles will be present in considerably larger numbers than they are in the same case in the absence of fever. For instance, in one case, on a day when the temperature was normal, 1 white to 1,225 white corpuscles were found; but on another occasion, when the temperature was 103·2, they were present in the proportion of 1 to 672, and many other similar instances might be given;

but the reduction of the number of white corpuscles, except when fever is present, is the most important and constant change in them in *kála-ázar*.

The specific gravity of the blood is always reduced in anæmia, chiefly, according to Lloyd Jones, in relation to the diminution in the amount of hæmoglobin. In *kála-ázar* the specific gravity of the blood is reduced, but only to a comparatively slight extent, and it never reaches the extreme degrees to which it is nearly always found to be reduced in anchylostomiasis, and not unfrequently it is scarcely removed from the normal limits, which are said by Lloyd Jones to vary considerably. Thus, out of 34 observations, the specific gravity of the blood was between 1,050 and 1,060 in 14 cases, while in 19 cases it was between 1,040 and 1,049, while in only one, and that a case in the very last dropsical stage, did it reach 1,039. The average was 1,048. Again, it has been found that the specific gravity falls as the red corpuscles and hæmoglobin become reduced, and rises as they increase in cases of recovery; but there was no constant relation between the number of the red corpuscles and hæmoglobin and the specific gravity of the blood, so that the alterations in the composition of the plasma must influence its density.

The coagulability of the blood in *kála-ázar* was found to be within the normal limits in a few cases in which it was estimated by means of Professor Wright's instrument.

The blood changes in ordinary chronic malaria.

The average composition of the blood in five cases of chronic malaria in Sylhet (including three of the cases shown in the photograph opposite page 29), which district has not been affected by *kála-ázar*, was as follows: hæmoglobin 31·6 per cent., red corpuscles per cubic millimetre 2,000,000, the number of white corpuscles per cubic millimetre 1,600, ratio of white to red corpuscles 1 to 1,400, sp. gr. 1·042, and the hæmoglobin value ·73.

These figures resemble those of *kála-ázar* both in the great reduction of the white corpuscles as compared to the red

in the high hæmoglobin value, and in the comparatively slight reduction of the specific gravity. Moreover, in the first two respects they also agree very closely with some examinations of the blood in cases of chronic malaria recorded in a paper in the *Indian Medical Gazette* of 1883 by Dr. Waddell, which are the only figures on the subject that I could find in the limited literature which I have been able to consult.

It is then evident that the type of anæmia in *kála-ázar* is precisely similar in all its details to that met with in ordinary chronic malaria. Let us now see if it resembles that found in anchylostomiasis.

The blood changes in anchylostomiasis.

In anchylostomiasis, which is the disease produced by anchylostoma sucking small quantities of blood out of the mucous membrane of the intestine (and not simply the presence of anchylostoma, which may not have been present in sufficient numbers, or long enough, to produce any disease whatever), the essential symptom is anæmia. We have just seen that in *kála-ázar* also, anæmia is a constant and marked symptom, and it will be obvious that if the two diseases are essentially the same, except that the latter is more frequently complicated by malaria, as was maintained by Dr. Giles, then the blood changes must be also similar, or nearly so ; while if the disease is a mixture of malaria and anchylostomiasis, then the blood changes should present a gradual series intermediate between the two primary conditions. Let us see if this is the case or not. Taking the different observations in the same order as before, it has been found that the hæmoglobin in twelve cases averaged 15·16 per cent., showing that the anæmia was more marked than it was in the *kála-ázar* cases. The number of red corpuscles in eleven cases averaged 1,145,000, or 42·9 per cent., and the hæmoglobin value in nine cases averaged only ·31, showing that the hæmoglobin was reduced more than twice as much as the number of the red corpuscles, and this was a constant feature in the disease, for the highest figure met with was ·39, which is much lower than

the lowest figure obtained in any uncomplicated case of *kála-ázar*, so that this alone serves to absolutely differentiate the two forms of anæmia, and consequently also the two diseases from one another. Again, the number of white corpuscles in ten cases averaged 5,338, and their ratio to the number of red was as 1 is to 524, showing that, although they are actually reduced in number, as compared with the standard of healthy Assamese people, yet relatively to the red corpuscles, they are increased in number, or, in other words, they are not reduced in as great proportion as are the latter. This again is in marked contrast to the state of affairs in *kála-ázar*, in which in the intervals between the fever, when the temperature is not raised, they are much reduced, both actually and relatively, to the red corpuscles.

Once more, the specific gravity of the blood is always very much reduced in anchylostomiasis, varying from 1,038 down to as low as 1,030, and the average of eight cases was only 1,034. This is partly due to the greater degree of anæmia in the series of cases of anchylostomiasis as compared with those of *kála-ázar*; but if cases of the two diseases presenting the same degree of anæmia be taken, the specific gravity of the blood is still found to be much lower in the anchylostomiasis ones than it is in the *kála-ázar* cases. Here, again, this point is of the greatest importance, as it serves to differentiate the two diseases, and as the estimation can be made in two minutes with very little practice, it is likely to prove of practical importance, for the treatment of the two different types of anæmia should be quite different.

The type of the anæmia produced by anchylostomiasis then differs so materially from that found in *kála-ázar*, as to absolutely differentiate the two diseases from one another.

Mixed cases.

It has already been mentioned that a small percentage of *kála-ázar* cases were found to be complicated by a sufficiently large number of anchylostoma to constitute them a factor in the production of the anæmia met with in these cases, and it may be said at once

that they presented blood changes which were exactly intermediate between those met with in the two contributing diseases, thus showing that the worms had in these instances been present in sufficient numbers and for a long enough period to bring about characteristic alterations in the blood. Thus, in five such cases, which were met with out of a total of 77 examined, or 6·49 per cent., the hæmoglobin value averaged ·43, which is just intermediate between the same figures for the anchylostomiasis and *kála-ázar*, respectively, as were also the other figures which will be found in the table on page 95, and thus served as a test as to whether any number of the worms, which might be met with in a given case, had been a factor in the production of the anæmia or not, for, if they had materially assisted in the production of the anæmia, then the type of the blood changes found would show the characteristic variation from that which is typical of *kála-ázar*. Further, I have even been able to correctly suspect the presence of anchylostoma in active numbers in an early case of *kála-ázar* before anæmia was a marked symptom, and when there was clinically no suspicion of them. I have, moreover, been able, by an examination of the blood, to diagnose from each other with certainty cases of both *kála-ázar* and anchylostomiasis, which I had previously either been unable to differentiate without it, or in which I had even made a wrong diagnosis by the ordinary methods in spite of considerable experience, for cases do not very infrequently occur in which *kála-ázar* very closely simulates anchylostomiasis, and this doubtless accounts for the former confusion between the two diseases. Thus, on one of the tea gardens, when working with Dr. Price, we picked out three cases, one typical of *kála-ázar*, the second of anchylostomiasis, whilst the third man in some respects resembled the latter disease, but he had an enlarged spleen, and dropsy of the legs and abdomen, with only slight fullness of the face, and, on the whole, we thought was probably suffering from *kála-ázar*. The following table gives the blood changes which were found in

them, from which it will be seen that the third and doubtful case resembles the second in type, although not in degree, or anchylostomiasis one, so closely as to remove at once all doubt as to the diagnosis, and the subsequent course of the case, with a re-examination of the blood after an interval of two months, proved it to be undoubtedly anchylostomiasis :

—	Hæmo-globin Per cent.	Red corpuscles. Per c.m.	White corpuscles Per c.m.	Ratio of red to white.	Hæmo-globin value.
Kála-ázar case	28	1 690,000	1,800	1:945	·84
Anchylostomiasis case	15	2,560,000	7,500	1:330	·28
Doubtful case	33	5,230,000	9,250	1:561	·31

Again, not long after, on returning to Nowgong I found in the dispensary, what I had no hesitation in saying was a typical case of anchylostomiasis in a girl of 17, who had marked anæmia, a "pearly white" conjunctiva, and no enlargement of the spleen or liver. As up to this time (and the same remained true to the end of my investigation) I had failed to find a single case of pure anchylostomiasis among the indigenous Assamese people, except on tea gardens, or in those who had worked on tea gardens, or on the Assam-Bengal Railway, in spite of visits paid to several of the dispensaries and villages of the Nowgong district, after due notice, for the purpose of seeing cases both of this disease and of *kála-ázar*, I was therefore much interested in this case, and as thymol had been given the day before, I immediately examined the stools passed during the preceding 24 hours before enquiring into the history of the case, and was surprised to find no worms. The blood was therefore examined, and the hæmoglobin value proved to be ·57, and the ratio of white to red corpuscles 1 to 1,403, the red corpuscles numbered 3,650,000, and the hæmoglobin was 42 per cent., all these figures being typical of *kála-ázar* as opposed to anchylostomiasis. The history showed that she had lost four relatives of *kála-ázar*, and had only one left alive, and that she had

suffered from repeated attacks of fever during the last two years, including four attacks during the rains of 1895, and for six weeks of this year up to a short time before admission. When seen, however, she had no fever, and only suffered from anæmia, being in a fair way to recover, which she subsequently did. Here, then the blood examination rectified an actual mistake in diagnosis, for the case was one of nearly recovered *kála-ázar*.

It only remains to consider the constancy of the typical blood changes in *kála-ázar*, and any circumstances which may modify them. It may be said that the hæmoglobin value of uncomplicated cases of *kála-ázar* never falls below ·5, as compared with that of ·65 for a healthy native of Assam, although it may rise considerably above the latter figure. I have only met with four exceptions to this rule, which, however, puzzled me a good deal, until one day, when going over my notes to see how often hæmorrhage from the nose, which is so often met with in *kála-ázar*, was of sufficient extent to be likely to affect the blood changes, and to make them approximate towards the anchylostomiasis type, I found only three such cases, and on looking up their blood analyses, I found all three of them among the four exceptions above mentioned out of over 50 estimations. This is a good example of the value of minute notes of a series of cases combined with special observations in elucidating a complicated problem, for three out of the four exceptions were at once explained, and the correctness of the observations themselves was confirmed. The fourth was on a tea garden, where I have found other cases complicated by anchylostomiasis, and, although no worms were expelled by thymol, this is not surprising, as he had very likely had the drug on a previous occasion, and I have found that it takes some months after the parasites have been expelled before the blood reaches the normal type again, so it is very probable that he had suffered from the disease at some time previously to his being attacked by *kála-ázar*.

The following table shows at a glance the difference in the average blood composition in healthy natives of Assam, *kála-ázar*, ordinary chronic malaria, anchylostomiasis, and mixed cases of *kála-ázar* and anchylostomiasis :

—	Percentage of haemoglobin.	Red corpuscles per cubic millimetre.	White corpuscles per cubic millimetre.	Ratio of white to red corpuscles.	Specific gravity.	Haemoglobin value.
Healthy natives of Assam ...	62	4,734,000	7,385	1:684	1·054	·65
Kála-ázar cases	33·45	2,402,000	2,600	1:1,170	1·048	·65
Ordinary chronic malaria ...	31·6	2,000,000	1,600	1:1,400	1·042	·73
Anchylostomiasis	15·2	1,145,000	5,338	1:534	1·034	·31
Kála-ázar and Anchylostomiasis	27·4	3,120,000	3,200	1:975	1·039	·43

The explanation and significance of these various changes met with in the blood must now be given, as they may not at first sight be evident. It will also be necessary to see if there is any confirmation of my results to be found in such literature as is at hand.

It will be evident from the facts given in the clinical section of this report, that in *kála-ázar* the anæmia is produced by the destruction of the blood corpuscles by the fever, which, as will be proved later, is purely malarial, and is caused by the plasmodium malariæ living on the red corpuscles, which they destroy, while the white corpuscles become gradually reduced, doubtless owing to the losses sustained by them in their unremitting fight with these organisms, as is shown by the constant occurrence of actual or relative leucocytosis during fever. The other elements of the blood are not directly affected, but in the latter stages they suffer some deterioration owing to the loss of digestive power, preventing their daily expenditure being replaced, as it is in health. On the other hand, the primary cause of the symptoms in anchylostomiasis is the daily loss of the small quantities of

blood which are sucked out by the worms; while, again in the latter stages this may be supplemented by some loss of digestive power owing to the injury to the intestine which may be produced by the worms. It is evident that in anchylostomiasis all the elements of the blood will equally be lost to the economy, including the albuminous parts, which are not primarily affected in the case of *kála-ázar*. The different constituents are not, however, equally readily renewed by the system. On the contrary, the white corpuscles are more easily replaced than the red so that in anchylostomiasis the former, although actually reduced in number, might be expected to become increased relatively to the red corpuscles; and this is just what we have found to be the case. Much more important, however, is the hæmoglobin, and this element of the blood, containing, as it does, iron in an organic combination, is much less easily replaced. It has been shown by the analyses of the late Dr. Bevan Rake, that after death from anchylostomiasis the amount of iron in the liver is often much reduced. This is due to the reserve iron in the liver being used up to stock the newly-formed red corpuscles, as these are turned out by the bone marrow. As soon as this reserve is exhausted, the renewal of the hæmoglobin will be entirely dependent on the amount of iron absorbed from the food through the digestive canal. Now this has been shown by Dr. Ralph Stockman to only amount to one-eighth of a grain daily in the case of a diet containing animal food, and will be even less in a vegetable one, such as the vast majority of the natives of India subsist on. Consequently, there is soon a great difficulty in the formation of enough hæmoglobin to put into the red corpuscles, which are being formed, and so a deficiency of hæmoglobin relatively to the number of the red corpuscles gradually arises, and the hæmoglobin value sinks to half, or less than half, that met with in health. Thus, the change in the composition of the blood, which we have seen actually occurs in anchylostomiasis, is only what might have been expected to result from a slight, but steady, daily loss of blood, and my

observations confirm the opinion that this loss of blood is the essential factor in the production of the anæmia. Again, the much lower specific gravity of the blood, even when the more intense anæmia is allowed for, which I have found in anchylostomiasis as compared with *kála-ázar*, is easily explained by the loss of the albuminous elements in the former disease being partly replaced by water, and hence the specific gravity will fall, especially when the changes in the digestive system prevent the absorption of a full amount of nourishment from the elementary canal ; while in *kála-ázar* this loss does not occur, and the density of the blood remains nearer to the normal being only affected by the loss of the red corpuscles and hæmoglobin.

It remains to be explained how it is that the hæmoglobin value of the blood in *kála-ázar* maintains its standard, as compared with anchylostomiasis. It has already been mentioned that the red corpuscles are replaced by the action of the bone marrow. In health it is only the marrow in the ends of the long bones and in the cancellous tissue of the other bones, which has the physiological function of forming new red corpuscles to replace such as become worn out day by day. It has been calculated that if the red marrow of all the bones of the body could be massed together, it would exceed in bulk the spleen itself, so that it can easily be seen how important are its functions during health. In the case, however, of such diseases as *kála-ázar* and anchylostomiasis, in which the blood is daily being impaired, the work of renewing the loss of the red corpuscles becomes doubly difficult. It has, moreover, recently been shown by Robert Muir and others that in the disease known as pernicious or essential anæmia (under which term some authors would include all extreme forms of anæmia due to whatever cause, and therefore those now under consideration), the marrow of the shafts of the long bones, which in health consists only of fat, becomes converted into red marrow, this being a compensatory change, or an effort on the part of the system to cope with the excessive

demand for red corpuscles. This change I have found to be a characteristic and constant one in all cases of *kála-ázar*, and they are also met with, although in my experience not quite to so marked a degree, in cases of anchylostomiasis, being doubtless purely of a compensatory nature in each case. It is in this way then that new red corpuscles are produced in both diseases, but in the case of anchylostomiasis, there soon becomes a dearth of hæmoglobin with which to stock them. In *kála-ázar*, on the other hand, the hæmoglobin is not lost to the economy, as it is in the former disease, for, when the plasmodia destroy the red corpuscles, the colouring matter becomes converted into pigment, and on the division of the parasite into numerous small ones, and the breaking up of the enclosing red corpuscle, such as occurs in the case of quotidian fever every day at the time of the onset of the fever, this pigment is set free in the blood, and gets filtered out by the spleen, liver, and other organs, where it forms the pigmentary deposits, which are of such a characteristic feature of chronic malaria, and which will be shown in the next section, to be also a marked and constant feature of *kála-ázar*. Thus, when in this disease fresh red corpuscles are formed by the bone marrow, there is already a stock of iron in these organs in organic combination, waiting to be used up to re-stock the red corpuscles, and this may even proceed until they contain more than the normal amount, as shown by the frequent abnormally high hæmoglobin value in *kála-ázar*. The ease with which the hæmoglobin may be replaced constitutes the essential cause of the difference of the type of the anæmia of *kála-ázar* from that of anchylostomiasis, and thus we see every change which I have found in the blood in either disease, is completely accounted for by what we know of the causation and pathology of them, and the complete differentiation of them as separate and distinct diseases, which, however, sometimes complicate each other, is placed on a firm basis.

It is also evident from the above that while iron will be essential in the treatment of the anæmia of anchylostomiasis,

it is much less important in that of the anæmia produced by malarial fevers, for, in the latter case, there is plenty of iron in an organic combination in the liver, etc., ready for use, but here arsenic will be indicated to increase the output of red corpuscles by the bone marrow, which explains the value of this drug in malarial and so-called pernicious or essential anæmia.

The question remains—can any confirmation of my results be found in the scanty literature within my reach ? I made a search for such in Calcutta before beginning work, but only found those by Dr. Waddell, which have been already mentioned on page 90, and, with regard to anchylostomiasis, I find, in Dr. Sandwith's valuable paper on this disease, in the *Indian Medical Gazette* of July 1894, details as to the estimation of the hæmoglobin and the number of the red corpuscles in 173 cases ; and working out the hæmoglobin value from these figures, it agreed so closely with what I had found it to be in my smaller number of cases, allowing for the low blood standard of natives in Assam, that I considered it to be quite unnecessary to spend more time in examining a larger number. The differences in the type of anæmia in chronic malaria and anchylostomiasis above described are then confirmed by the results obtained by independent observers, and the proof that the anæmia of *kála-ázar* differs essentially from that met with in anchylostomiasis, while it conforms to that caused by malarial fever, is placed on a sound basis.

SECTION V.

THE PATHOLOGY AND NATURE OF KÁLA-ÁZAR.

In this section the pathological anatomy, both macroscopic, as seen in the *post-mortem* room, and microscopic, as far as this has been worked out by means of cutting sections of the diseased organs, will first be given, and then the true nature of the disease will be discussed in the light of all the facts which have been adduced in the first five sections of this report.

With regard to the pathology of the disease, again, but little help is to be obtained in the literature of the subject, and the value of that little is greatly diminished by the former confusion between *kála-ázar* and anchylostomiasis. I much regret that I have not been able to devote as much time to the microscopical part of the work as I should like to have done, owing to the pressing claims of the more immediately practical work of studying the mode of spread and present limits of the epidemic which has necessitated prolonged tours; while at the time of writing I am anxious to submit my report without delay, so as to allow of a full consideration of my recommendation before the next cold weather arrives, which is the only time for action being taken on them. As I still have the material by me, I shall hope to go into this—the more scientific side of the question—as soon as I shall have any leisure time, and only the more important facts will be given here.

The following description of the changes found *post-mortem* is based on 25 autopsies on cases of *kála-ázar*, which have been made by myself, with the exception of a few which were kindly performed for me, in accordance with a scheme I drew up, by Mr. McNaught and Dr. Dodds Price. Four others on cases of anchylostomiasis, and seven medico-legal ones were performed for the sake of control observations.

These have been described in the clinical part of this

report, namely, great wasting of the extremities, and upper parts of the body contrasting markedly with the enlargement of the abdomen, due to the greatly increased size of the liver and spleen, and in some cases dropsy of the feet, legs, and rarely of the abdomen. Anæmia may also be noted in the mucous membranes. The comparatively dark skin is often a noteworthy feature, and on cutting into the body, the marked absence of subcutaneous fat, in which respect the disease differs from that of anchylostomiasis, concerning which Lutz writes—"In most cases subcutaneous fat is well developed, sometimes remarkably so," as is also the case in the chlorotic anæmia so often seen in England.

External appearances of the body.

The pericardium contained from 1 to 5 ounces of fluid in one-fourth of the cases, while I once found 12 oz. present. Signs of recent inflammation of the serous membrane were also occasionally met with.

Cardio vascular system.

The heart was nearly always much smaller than normal, the average being only 6 oz., as against a little over 8 oz., in healthy people who had died from accidental causes. This point struck me very much at first, and is again in marked contrast, as has already been mentioned in the clinical section, with what is met with in cases of anchylostomiasis, in which the heart is usually dilated and often also hypertrophied to such an extent as to cause very evident palpitation. The difference is due to the more or less constant presence of fever in *kála-ázar* cases, which, besides causing granular degeneration of the heart muscle, followed by fatty changes, also allows of no rest during which hypertrophy may take place. This is borne out by the fact that in one or two very chronic cases of the disease with prolonged intervals between the fever, slight enlargement was met with. In several cases I found distinct fatty degeneration of the cardiac muscle, but in some cases this was absent. The heart muscle was usually of nearly normal colour and consistency, and no signs of endocarditis were seen.

The arteries of the brain were in several cases found to be in a state of advanced fatty degeneration, which probably also affected the other arteries of the body, and accounted for the frequency of the slight epistaxis, which has been previously noted.

The respiratory system.

One or both pleural cavities often contained a small quantity of clear fluid, and in one man who died of pleurisy, 6lb were found in his chest.

The bases of the lungs were almost invariably found to be congested, especially in the cases who died during the rainy season of actual fever. During the onset of the cold weather, deaths were frequent from lung complications, which comprised pneumonia, pleurisy, and also in one case gangrene of the lung. Œdema of the lungs also caused the death of one of the dropsical type of cases.

Digestive system:

The stomach was in two or three cases very small, and its mucous membrane thick and rough with projecting ridges. This condition I have also met with after death by accident in healthy persons, so its occurrence in *kála-ázar* is probably accidental, especially as symptoms of inflammation of the organ, such as vomitting, are very rare in this disease, while chronic dyspepsia is common enough among natives. In one case ulceration was found, but, as a rule, the mucous membrane appeared to the naked eye to be normal. In one case also, marked pigmentation of the spaces between the rugæ of the stomach and on the valvulæ conniventes only of the duodenum was found—a distribution which suggested a deposition of the pigment from the blood stream, rather than its formation *in situ*, as a result of chronic inflammation. The case was a very long standing one.

The small intestine was often thinner than normal, especially in the more chronic cases which had died of asthenia accompanied by diarrhœa, and in these instances it was often also pigmented, more especially in the upper part, but extending down to the ileo-cæcal valve, and usually less marked on

the peyers patches than elsewhere, causing them to stand out in contrast to their surroundings. These changes were certainly the result of chronic inflammation of the small intestines, which, doubtless, plays an important part in the pathology of the disease. The mesenteric glands have also been found in one or two cases to be pigmented.

Now, in the few cases of anchylostomiasis on which I have made *post-mortems*, I have found the intestine to be sometimes slightly thicker than normal, but not thinner, and I have not seen any pigmentation of the mucous membrane. As the point seemed to be an important one, and my experience was too limited to allow of any safe generalizations being made from it, I wrote to Dr. Sandwith on the subject, and am much indebted to him for his kind reply to this and other questions. He writes —

"The stomach contains no pigment or thickening, but I have once or twice found hæmorrhage in the stomach as described by Giles. I cannot explain this. The intestines and mesenteric glands are neither of them pigmented."

In his paper on anchylostomiasis in Egypt, he also writes:

"I have not observed the constant changes in the mucous membrane of the stomach described by Giles."

The pigmentation, which I have met with in true cases of *kála-ázar*, is moreover of such a distribution and so closely set, that it could not possibly be due to the remains of the small hæmorrhages caused by the bites of the anchylostoma worms; although it might possibly arise from inflammation of the intestines set up by these parasites, this must be rarely the case, or it would have been seen by Dr. Sandwith in Cairo. Most, then, of the cases in which Dr. Giles saw these changes during his investigation of *kála-ázar* in Assam, must have been true cases of the disease, and not, as he thought, due to anchylostomiasis. His description of the exact changes which he found, however, is not sufficiently minute or systematic to allow of accurate comparison with those which I met with.

The large intestines showed ulcerations in a very few

cases, and congested patches and thickening or even œdema of the mucous membrane rather more frequently, and in one or two cases some pigmentation.

The anchylostoma were very carefully counted in each case, and the amount of damage done by them minutely scrutinised by the help of a magnifying glass, which was very often necessary in order to enable it to be distinguished. In 4 out of the 25 autopsies, no anchylostoma were found; in 7 more, less than 10 were present, in 5 there were from 11 to 20, in 5 from 21 to 30, and in the other 4 cases, 48, 50, 80, and 103, respectively, the average of the whole being 21·48. In 7 healthy persons, who had died from accidental causes, on the other hand, the numbers present were 11, 10, 11, 13, 24, 34, 61, and 72, respectively, with an average of 32. The slightly smaller numbers in the former series is doubtless due to the fact that some of the *kála-ázar* cases, but by no means all, had been given thymol during life. In four cases, on the other hand, in which the anæmia was really due to anchylostomiasis, as proved by the character of the blood changes, the numbers found were 161, 350, 40, and 249, giving an average of 200. Dr. Sandwith, in *post-mortems* on 18 cases, which had not had the benefit of the thymol treatment, found in six of them less than 10 worms, in three others 20, 40, and 50 worms, but in the other nine numbers varying from 170 to 381, terminating with the maximum record of 863. Thus, it is evident that, although in some cases very few worms may be found after death, yet in a series of cases a very different record is obtained from that met with in *kála-ázar*, and the evidence here given, together with that detailed in the clinical section, and the figures obtained by Dr. Dobson, prove incontestably, apart altogether from the absolute scientific proof afforded by the differences in the blood changes given in Section IV, that the anchylostoma worms are not present in the majority of *kála-ázar* cases in larger numbers than they are in the healthy people of Assam, much less are they present in sufficiently larger numbers in this disease, than they are in

healthy people, to constitute them an important factor in the production of the affection.

As to the lesions caused by the bites of the anchylostoma, I was fortunate enough to get several autopsies within a very few hours after death, when the large majority of the worms were still adherent to the mucous membrane of the small intestine. I was surprised to find that, although under many of them a small hæmorrhage was easily seen, yet on close examination it was found that at the point of attachment of the majority of them, no hæmorrhage was visible, even with a magnifying glass, but only a very small white dot or circle. Doubtless, whenever they bite a new place, one of these small hæmorrhages must be formed, which will certainly take at least a week to be absorbed at the very lowest estimation, so that it is evident that the worms usually remain attached to the same place for at least that time, which is only what might naturally be expected. Again, I found fresh blood in only a minority of the worms which I examined under the microscope, although it does not clot in the bodies of the parasites, and would keep its natural appearance for some time, which seems to show that the worms are not constantly sucking blood out of the intestine, but only take sufficient to nourish them, and to allow of the rapid formation of ova; and as they are quite small and their food is of the most nutritious kind, there is no necessity for their taking more than a drop or two of blood a day, or for their changing their feeding ground more than once a week, perhaps, when the minute scar, which they make, begins to contract around them. Now Dr. Braddon, of the Malay Peninsula, in a criticism of Dr. Giles' report, estimates that, even if it be allowed that a hundred parasites are present and inflict several fresh bites daily, it would take a year to produce such considerable erosion as would destroy even one-twentieth of the digestive area of the small intestine alone. Yet Dr. Giles writes that—

"The mere loss of the nutritive matter required for the support

of the parasites, is a quite small and quite unimportant factor in the causation of the fatal symptoms produced by them . . . . The real damage is mainly caused by the destruction of the digestive powers."

The last sentence is a very true statement as applied to the later stages of the more chronic cases of *kála-ázar* (only the loss of the digestive powers is one of the effects of the fever, and is not due at all to the very small number of anchylostoma which are present in ordinary cases of the disease), but is very wide of the mark as applied to anchylostomiasis. Thus, Lutz writes of the latter—

"It may be said that adults, in the absence of any complication, and in whom the disease runs a tolerably quick and uniform course, do not begin to show symptoms until the number of anchylostoma passes into the hundreds, so that when pronounced, general symptoms are present, three to five hundred parasites may be set down as present in the duodenum. In severe cases I have found over a thousand present, but in the Gotthardt epidemic, two or even three thousand were found."

And he attributes the main symptoms to the loss of blood brought about by the parasites, and traces the three stages into which he divides the disease, namely, (1) stage of purely local symptoms; (2) stage of simple anæmia, and (3) dropsical stage, to the gradually produced anæmic condition of the blood. With regard to the dyspeptic symptoms, he writes—

"The gastric catarrh, occurring constantly in the advanced stage of the disease, and leading in its train loss of appetite, diarrhœa, and imperfect absorption, helps to bring about emaciation."

But that this even in the advanced stages of the disease is not so fatal as Dr. Giles would have us believe, is quite certain from the very low mortality of the affection when properly treated, as has been shown in Section III. Some other observations in connection with the subject of anchylostomiasis, which do not come into my general argument on the nature of *kála-ázar*, will be given in an appendix to this report.

The liver.

This organ is much enlarged in *kála-ázar*, and averaged in 21 adults 3lbs. 13oz.; and this in spite of the emaciation of the great majority of the patients. In four adult healthy people, who died from accidental causes, it averaged 2lbs. 5oz., but in four cases of anchylostomiasis it only averaged 1lb. 14oz., which confirms my clinical experience, and the statements of Lutz and Sandwith, that the organ is either normal or smaller than normal in this disease. The average size of the organ in *kála-ázar* cases given above, conveys but a feeble notion of the size to which it may attain in this affection, for in the more chronic cases I have found it to weigh over 5lbs. in two cases, over 6lbs. in one, while the maximum recorded was 7lbs. 2½oz. In three cases only was it under 2½lbs., and they had suffered much from diarrhœa during life. Its appearance on section varied a good deal, as it was usually of a mottled yellow and red colour, which was distributed in a manner precisely similar to that figured in Dr. Drury's paper in the transactions of the first Indian Medical Congress on "a peculiar form of fatty degeneration in malarious poisoning." In addition to this there were often darker patches, most frequently seen just under the capsule of the organ, which were due to the deposition of pigment, and in other cases this was indicated by a general darkening of the whole cut section of the organ, giving it a dirty brownish appearance. In one or two very chronic cases it was of quite a dark hue, while in one case, which ran rather a rapid course and finished with a temperature of 105°F., it only presented an appearance of great congestion. In one case, well marked cirhosis of the liver was present, but I was unable to absolutely exclude alcohol as a factor in its production, and although Kelsch and Keiner describe a form of the disease as occurring in chronic malaria, yet most cases of *kála-ázar* certainly run too rapid a course to allow of its production in a marked degree. The organ is, however, frequently harder than normal, and some excess of interlobular tissue may often be seen under the microscope.

The gall bladder was normal, and contained bile, which was often of a rather dark colour.

The spleen.

The spleen is always enlarged, and usually markedly so. It averaged 2lbs. 5oz. in 21 cases in adults. The lowest weight was 14oz. in an acute case; it was between one and two pounds in five cases; between two and three in seven cases, and over three in seven others, the maximum being 3lbs. 14oz. If the great wasting of the body be taken into account, the relative enlargement of the organ would be much greater than these figures indicate. Except in the most acute cases, the organ is harder than normal, especially in its peripheral portions, and under the microscope an excess of fibrous tissue is found. The organ is also usually of a dark-red colour, which, in the peripheral parts, often gives it an almost black appearance, which is again due to the deposition of pigment in the organ. In four healthy adults, who died from accidental causes, the organ averaged 1lb., varying from 8oz. to 1lb. 8oz., and in four anchylostomiasis cases, it only averaged 10½oz., being under one pound in each case, in which respect, once more, my observations are in accord with the much more extensive experience of both Lutz and Sandwith, in showing that in this disease, except when it is complicated by malaria, the spleen is either of normal size or it is diminished in bulk.

The kidneys presented no pathological changes to the naked eye, except for the occasional occurrence of small cysts, such as will be met with in any series of *post-mortems*, but their colour was often rather darker than normal, instead of being lighter, as might have been expected in a disease in which anæmia is such a constant feature; but this was subsequently found to be due to the constant presence of pigmentation of the organs. Their weight was also less than normal, but this again was only in proportion to the general wasting of the body, and was in marked contrast to the enlargement of the liver and spleen. The suprarenal bodies were also carefully examined, but nothing abnormal was found in them.

The thoracic duct was also dissected out in all the earlier cases, but was always found to be patent, so that the emaciation was not due to any obstruction of this channel of absorption.

The cerebro-spinal system.

A small quantity of fluid was often found beneath the arachnoid, or in the ventricles of the brain, which probably filled the vacancy formed by slight atrophy of the organ. On section the brain and spinal cord were normal. Pigmentation was always found to be well marked on the under-surface of the medulla oblongata, which in a few cases extending over the pons as far even as the optic commissure, and over the upper surface of the cerebellum. At first this was looked on as constant and important sign of the disease, but it was subsequently found that it was also to be met with under the medulla in healthy persons, and even in anchylostomiasis, so that it must be a normal condition in the natives of this country, although I have not seen it mentioned in any book on pathology. It extends down over the anterior surface of the cervical portion of spinal cord. In one case hæmorrhage beneath the arachnoid was the immediate cause of death; but this was in a mixed case, and was, I think, due rather to the blood changes brought about by the anchylostomiasis factor than dependent on the fever of *kála-ázar*, as I met with a similar hæmorrhage between the anterior surface of the bladder and the peritoneum in a case of pure anchylostomiasis.

The bone marrow.

It has recently been found in some cases of pernicious anæmia observed in England, that the marrow of the shafts of the long bones, which in health consists of yellow fat, is converted into red marrow, such as is normally met with in the loose cancellous tissue of the end of the long bones, and which forms the bulk of that of the short bones. This red marrow has the function of forming red corpuscles of the blood. An examination was therefore made of the marrow in the shaft of the femur or humerus

in all my *post-mortems*, and it was found that in cases of *kála-ázar*, the yellow marrow was always converted into the red variety, often being of a dusky-red hue, and that this change was in proportion to the length and chronicity of the case. It was also much softer than usual, and nearly, or quite, free from the small spiculæ of bone which are met with in it normally. The cancellous tissue of the ends of the bones was also sometimes looser than normal, showing that a certain amount of the bone salts had been absorbed. Pigment is also to be found in the marrow under the microscope. These changes are doubtless of a compensatory nature, due to the efforts on the part of the system, to repair the unusually rapid deterioration of the blood, and hence their occurrence in this disease is only further evidence of the destructive effect of the fever on the blood. As the changes have been fully described in the Journal of Pathology and Bacteriology, Vol. II, No. 3, by Dr. Robert Muir, they need not be further discussed here. Out of four healthy persons, who died from accidental causes, on the other hand, in two the bone marrow was quite yellow, and in the other two only faintly tinged with red. In three cases of anchylostomiasis it was reddened, but not to the same degree as in *kála-ázar*, and Dr. Sandwith, who examined it in a recent case at my suggestion, found it to be of the colour of "strawberries and cream." I expect that slight changes of this nature will be found to be very frequent in such countries as India, where malaria and other diseases which destroy the blood are so common, and where even in health the demands on the system for the repair of the blood are much greater than in temperate climates, so that its occurrence in *kála-ázar* is probable of more pathological interest than of diagnostic importance. Its discovery, however, suggested to me the line of treatment by bone marrow tabloids, which promises to be of considerable value. These changes account for the pains in the limbs, which have been noted to be such a common feature of the disease.

The chief point to which attention was directed when

sections of the different organs were being prepared for examination under the microscope, in accordance with my plan of work, as set forth in Section II, was to determine if pigment was present in the liver, spleen, and kidneys, and if so, whether it was as marked and constant feature as Kelsch and Keiner have shown it to be in the cases of chronic malaria, which they examined in Algeria. It may at once be said that it was constantly met with in the series of cases examined, and that it was roughly in proportion to the chronicity of the case, being easily seen in properly prepared specimens in the more acute cases, while in the more chronic ones it was so marked as to be visible to the naked eye in thin sections floated out in a white vessel, especially in those cases which showed a dark colour of the surface of a section of the organs in the *post-mortem* room. The distribution was also similar to that figured by the French authors in their work, so that it will not be necessary to describe it in detail here. Its presence affords complete proof of the malarial nature of the disease, and its importance is enhanced by the complimentary results of the analysis of the amount of iron in the liver to be immediately mentioned. In one case, in which *kála-ázar* was complicated by anchylostomiasis, the changes were less marked than usual, probably owing to a portion of the deposited pigment having been used up to re-stock the red corpuscles, the hæmoglobin values of which were low in this instance, being only ·4. No other changes, besides those already mentioned, were met with, which are regarded as being of sufficient importance to require description here: suffice it to say that no changes were seen, which were inconsistent with the malarial nature of the disease, and it is also worthy of note that all the pathological changes recorded in this section, as well as all the clinical signs detailed in an earlier one, were found by Kelsch and Keiner in cases of chronic malaria in Algeria, as recorded in their *Traite des maladies des pays chauds.*

The microscopical anatomy.

The foregoing description of the pathological changes met

with in the disease is so typical of that which is seen in cases of malarial cachexia all over the world, that there are very few points to which special attention need be directed. The changes in the liver and spleen are characteristic of the disease. The granular and fatty degeneration of the heart, together with its atrophied condition, are of considerable importance in determining the great fatality of the inflammatory complications of the lungs, as the right ventricle becomes acutely dilated when any extra work is thrown on it, as was seen in the two *post-mortems* after death from pneumonia given in illustrative cases in Section III. There was no change in the suprarenal capsules such as could account for the darkening of the skin so frequently met with in the disease. It is due partly to the natural pigment of the skin being more apparent owing to the anæmic condition of the patient, but more still to a deposit of fresh pigment in the integument, which is derived from the hæmoglobin of the broken-down red corpuscles, for it may be very noticeable in chronic cases in which the anæmia is slight.

The atrophy and pigmentation of the mucous membrane of the small intestine are changes which are of great significance in relation to the high mortality of the disease, as they are the result of an inflammation of the mucous membrane, which shows itself clinically in the diarrhœa which so often begins during an acute attack of the fever, and goes on, after the temperature has fallen, in a most obstinate manner. Not unfrequently, as was very well illustrated, in some cases of which the notes and temperature charts were kindly recorded for me by Dr. Dodds Price, a rapidly fatal form of diarrhœa sets in directly the temperature falls after a severe and prolonged attack of remittent fever. In these cases it must be set up by an enteritis, which occurs coincidently with the high temperature, and it is well known that this is a symptom of almost absolutely fatal significance. It is in marked contrast, as already pointed out in Section III, to the constipation, which is the general rule in anchylostomiasis. These intestinal changes are the cause of the

loss of digestive powers, which so greatly retards recovery of strength and weight after the fever has become less, or has ceased, and to them must be attributed much of the fatality of the disease.

Analyses of the iron in the liver after death.

Portions of the livers of five cases were sent to Calcutta for analysis of the amount of iron in them, which was kindly carried out for me by Dr. Waddell, Chemical Examiner to Government, at the Medical College. The object was to determine if there was an excess of iron such as is present in chronic malaria, or a diminished quantity, as was found in some cases of anchylostomiasis by the late Dr. Bevan Rake. Unfortunately the specimens which were obtained at the time they were required, were a singularly unfavourable series for the purpose, as the case of anchylostomiasis was complicated by malaria, while three out of the four *kála-ázar* cases were complicated by anchylostoma in considerable numbers, considering that two of them were children.

An examination of the amount of iron found in the liver by different observers in health, vary so much that it must depend greatly on the exact method adopted in the analysis. Thus, while William Hunter gives eight analyses of the liver in pernicious anæmia by different observers to average ·713 per cent., and that in 14 cases of other disease to average ·203, Ralph Stockman found an average of ·185 in two cases of pernicious anæmia, ·07 in five normal livers, and ·03 in four cases of anchylostomiasis. My figures are lower still, in spite of the fact that an excess of iron was found in some of these livers by microscopical examination, and only average ·023. It is then obvious that the methods of analysis were not sufficiently similar to allow of accurate comparisons between the figures obtained by different observers, and those of any series will only be comparable with one another.

The figures given in the following table will therefore be of only relative value:

—	Disease.	Age.	Anchylostoma found, p. m.	+ percentage of iron in liver.	Total iron in liver.
(1)	*Kála-ázar* ...	25	21	·0316	·50 gramme.
(2)	Ditto ...	7	50	·0237	·162 ,,
(3)	Ditto ...	8	80	·0233	·195 ,,
(4)	Ditto ...	27	103	·0134	·123 ,,
(5)	Anchylostomiasis *plus* malaria.	20	161	·0242	·203 ,,

From this table it will be seen that No. (1), which was the only uncomplicated case of *kála-ázar* in an adult of the series, had a much higher percentage of iron than any of the others, while the total amount in the liver in this case was more than double that of any of the rest. In the other three *kála-ázar* cases, the percentage of iron decreased in proportion to the increase in the number of anchylostoma present, just as might have been expected.

In the last case, although the patient died of anchylostomiasis, it was complicated by chronic malaria, as was shown by the spleen being large and very hard, and consequently the amount of iron is comparatively high, although not nearly so high as in the uncomplicated case of *kála-ázar*. It must also be borne in mind that owing to the intense and comparatively rapid course of *kála-ázar*, it could not be expected that as much iron pigment would have time to collect in the liver in these cases, as in ordinary chronic malaria, which so often last for years.

It is evident then that in uncomplicated *kála-ázar*, there is an excess of iron in the liver after death, as has also been proved to be the case by means of cutting sections of the organ, as shown at page 115, but that there is not sufficient difference between the results obtained in *kála-ázar*

complicated by anchylostomiasis, and in anchylostomiasis complicated by malaria, as to be of any practical use in differentiating the two conditions.

It is also worthy of note that the hæmoglobin value was not much reduced in some of the cases of *kála-ázar* in which the iron analysis was comparatively low, which supports the conclusion that the reserve iron in the liver must be considerably diminished before the hæmoglobin value falls materially, a point of considerable interest.

Bacteriology.

A series of *agar agar* tubes were inoculated with the blood obtained by pricking the fingers of *kála-ázar* patients with due precautions. Some were also inoculated with blood withdrawn directly from the spleen of a case in which abscess of that organ was suspected, but not found. Negative results were obtained, in which respect my observations are in accordance with those of Dr. Giles, so it may safely be concluded that there are no organisms in the blood of *kála-ázar* patients, which will grow on *agar agar*.

Although the plasmodium malaria is not, strictly speaking, a bacterium, its presence in *kála-ázar* may be conveniently considered under this heading. A great deal of time has been expended in an examination of blood of *kálá-ázar* cases for these organisms, as the probable malarial nature of the disease being soon evident, it was hoped, and even expected, that the plasmodia in this virulent form of fever would differ from those met with in ordinary cases of malarial fever in Assam, in such a degree as to allow of the differentiation of the two types of fever from one another. In the later stages of the disease, when anæmia has become a distinct feature, great changes in the sizes and shapes of the corpuscles, together with neucleated ones and heamatocytes, etc., take place to such a degree as to render a minute study of any malarial organisms that may be present doubly difficult. In order to overcome this difficulty, a series of early cases of fever on affected tea gardens were examined, which could not be

definitely diagnosed as *kála-ázar*, but some of which occurred in families or houses in which true cases of the disease were present, and so were likely to develope into the typical condition. After an interval of six or eight weeks, they were all seen again, and the notes and sketches of the organisms which were found in the blood in those cases which had developed into typical *kála-ázar*, were compared with those which had turned out to be apparently ordinary malarial fever. No differences, however, were found in the two classes, except that in the *kála-ázar* cases the organisms seemed to be less frequently pigmented, but the difference was not sufficiently constant and striking to allow of their being differentiated by it. The forms found include most of those figured by Italian authors as typical of quotidian fever, only I never came across any cresent forms, which at first surprised me, as they are usually found in relapsing kinds of malarial fever, such as *kála-ázar* is; but, on the other hand, cases in which they have been met with by Italian authors, are always of a very acute nature, often ending with coma or other complications, in which respect they differ greatly from the type in *kála-ázar*, in which such acute symptoms are never seen. My former work on the malarial organisms (*Indian Medical Gazette*, February 1896), moreover, together with that of other observers, seems to show that the cresent bodies are only met with in certain less common types of fever in India, and not in the great majority of them. In addition to the series above noted, the organisms were also found in nearly all the more advanced typical cases during the presence of fever, in all stages of the disease up to the day before death, and in numbers roughly proportionate to the height of the fever.

It may therefore be concluded that the malarial organism is a constant accompaniment of the fever of *kála-ázar*, and that the organisms found do not differ from those seen in the ordinary malarial fevers of Assam and other parts of India, to a sufficient degree to differentiate the type of the disease from them, and this observation is in accordance with the absence

of any definite type of fever in the affection, and with its being indistinguishable in individual cases from ordinary malarial fever.

THE NATURE OF KÁLA-ÁZAR.

The nature of *kála-ázar.*

We are now in a position to discuss the true nature of the pathological processes, which underlie the disease, in the light of the facts set forth in the last three sections. It will be convenient to do so under the headings given in Section II, as being possible views of the nature of the affection, although the order there given need not necessarily be adopted.

This is essentially the view taken by Dr. Giles, who

Is the disease anchylostomiasis?

writes:

"All I wish to convey is that the increased mortality is due to anchylostomiasis, and to no other cause."

From an article in the *Indian Medical Gazette* of July last, it appears that he is now more inclined to attribute a share in the production of the disease to malaria, for he is quoted as writing:

"The increased mortality is due to anchylostomiasis acting in people already worn down by malaria . . . . Anchylostomiasis is, of course, quite capable of acting alone, but it is only occasionally that it gets the chance of showing what it can do without its ally, malaria."

If by this Dr. Giles means that nearly every native suffers from malarial fever at some period or other of his life, this is quite true of Assam, and indeed of most parts of India; but if, on the other hand, he means that nearly all the natives of Assam show definite physical signs, such as enlargement of the spleen, of the action of malarial disease, such as would predispose them to other diseases, then this does not apply to the case of the Nowgong district, for, as I have shown in Section III, only one-quarter of the ordinary inhabitants of this district show any such signs, even during the height of the fever season. Moreover, as late as 1892, in a letter to the

Chief Commissioner of Assam, in reply to the criticisms of Mr. Melitus, the Secretary to the Chief Commissioner, Dr. Giles writes:

"I will now take up the consideration of the objections advanced by Mr. Melitus to the identity of *kála-ázar* and *beri-beri*."

(Anchylostomiasis), and again:

"What then is *kála-ázar*? *Kála-ázar* is anchylostomiasis."

This is certainly what he is generally understood to have meant by all in Assam who have read his report, and the quotations given in the first section of this report bear this out.

Unfortunately for Dr. Giles, it was not known at the time of his investigation that over 80 per cent. of healthy natives have the anchylostoma worms in them, for the most part in quite small numbers, so that he regarded the mere presence of these parasites, or even the finding of the ova in the fæces, which I have shown, can be done when less than 20 of the worms are present, as evidence of anchylostomiasis, that is, disease produced by these parasites, and unfortunately he did not make any control examinations in healthy persons, or he would have found out his mistake. But having found "a number of anchylostomes" in one autopsy, and "enormous numbers of the ova of the parasite" in the dejecta of five or six other cases, he writes:

"Other *post-mortems* followed, and proved incontestibly that, whatever *kála-ázar* might be elsewhere, the disease so called in Gauhati was undoubtedly anchylostomiasis."

He does not give the number of the worms which he found except, in two cases the full notes of which he records, one at least of which was certainly a pure case of anchylostomiasis, and not *kála-ázar* at all, "about a thousand anchylostoma" being found, while the spleen and liver were smaller than normal. Dr. Giles' further statement, after visiting Upper Assam to study anchylostomiasis, that—

"nothing here struck me so strongly as the absolute identity of the clinical pictures presented by these cases of acknowledged anchylostomiasis with those I had just been seeing so much of under the name of *kála-ázar*,"

can only be reconciled with the unanimous opinion of the meeting of planters' doctors at Kokilamukh in October 1894, that—

"as the disease observed in the patients (*kála-ázar* cases) brought before the meeting by Drs. Dodds Price and Lavertine, and from the history of similar cases, appears to be entirely unknown in Upper Assam above Nowgong, and differs in every particular from anchylostomiasis, with which it has been confounded; and as the disease is prevalent in certain districts, and is slowly, but most certainly, advancing up the valley, an expert is necessary,"

on the supposition that, considering as he did that, enlargement of the spleen and liver and fever were only accidental complications, and not ordinary symptoms, of the disease, he compared such cases of simple anchylostomiasis as he saw in Gauhati with similar ones in Upper Assam. Dr. Giles' opinion that fever was not a common or 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 and other work in Shillong, 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. Moreover, as he admits in his preliminary report, the longest time that he had any one case under observation was one month, while the majority of them he could have seen but once or twice in the villages which he visited. Now I have seen fever remain absent from typical cases for more than a month, even in the rainy season, and yet a fatal relapse occurs, while at the time of writing (February) out of seven cases in the Nowgong dispensary, just about the number that Dr. Giles commenced work on in Gauhati, only one has regular fever, and then only a temperature of 101° or 102°F., and this has been the kind of thing ever since the end of November, so much so that I have found great difficulty in getting

cases on which to try the effect of various modes of treatments in checking the fever. In fact, if I had begun work at this time of the year, I should probably have fallen into the same error that Dr. Giles did in this respect. Once more, malarial complications are more frequently present in the apparently healthy people of the Kamrup district than in Nowgong, and anchylostomiasis is also more common in the former, than I have found it to be in the latter district; and here again I have had a great advantage over Dr. Giles. Indeed, the facts just mentioned afford the true explanation of the very different clinical results arrived at by Dr. Giles and myself, and it is only fair to point out the great advantages which I have had over him, both in my happening to begin my clinical studies in the fever season, and still more in having the help of all the work that has been done by the Civil Surgeons and private practitioners of Assam during the last six years, and especially of the knowledge of the frequent presence of the anchylostoma worms in healthy people of both Assam and other parts of India.

To return to the question—Is *kála-ázar* anchylostomiasis? In reply, it may be said that I have shown these parasites to be present in sufficient numbers to complicate the disease, as proved by their effect on the type of the anæmia in under 7 per cent. of *kála-ázar* cases, while in 83 per cent. of a series of cases taken in as early a stage as possible, and which had not previously been treated by thymol, there were less than 20 of the worms present—a number which I have proved to have no effect whatever on the blood of healthy persons in the Nowgong jail, while Dr. Thornhill, of Ceylon, admits that 50 or less anchylostoma form a number "altogether too small to have any deleterious effect." It is also worthy of note that Dr. Dobson found less than 20 anchylostoma to be present in 82 per cent. of 547 healthy coolies, which he examined in Dhubri, which agrees with my results in *kála-ázar* cases. Again, I find an average of fewer anchylostoma on the average in a series

of 25 *post-mortems* on *kála-ázar* cases, than I do in healthy men who have died from accidental causes, and only about a tenth of the number that I found to be the average of my cases of pure anchylostomiasis. Once more, in the series of 200 cases of *kála-ázar* on a tea garden, 96 per cent. died, although the factor of anchylostomiasis was excluded from them by the use of thymol in an early stage of the disease. Finally, and most conclusive of all, I find the blood changes as illustrated by the type of anæmia present to be quite different in the two diseases, and the clinical details in this report are based on a series from which all cases of anchylostomiasis were excluded by means of this check. These facts prove incontestably that whatever *kála-ázar* may be, it is not anchylostomiasis, 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 six or seven 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 part of India.

Is the disease a combination of both anchylostomiasis and malaria?

This is the view most widely held outside Assam? Those who hold this view, including at the present time apparently Dr. Giles himself, look upon the anchylostomiasis as accounting for the spread of the disease, while the malarial complication explains the much greater mortality of *kála-ázar* over that of simple anchylostomiasis. The facts adduced in answer to the first question, are sufficient to negative the present one as well; but one or two further points may profitably be discussed in this place. Firstly, if anchylostomiasis is to account for the spread of the disease, it must be a constant and marked feature, such as Dr. Giles considered it to be, while the malarial complication might be sometimes absent. The facts are quite the reverse. Again, has anchylostomiasis ever been known to cause a fatal epidemic which has spread from one district to another, dying out as it goes along (for this will be shown to have been the case with regard to *kála-ázar* in a later section), and lasting without

sign of cessation of its spread over a series of twenty years, as this epidemic has, and absolutely depopulating certain tracts of country? If anchylostomiasis could do this, then the St. Gotthardt tunnel outbreak should have caused such an epidemic when the infected workers dispersed to their homes on the completion of the work, for it is recorded that hundreds and thousands of them suffered. Was this the case? Lutz writes:

"As for the Gotthardt tunnel epidemic, it seems to have disappeared *in loco* after the completion of the work. The infected labourers were scattered elsewhere, and afforded opportunities of observing the disease in still wider circles; yet these do not seem to have given rise to any fresh epidemic outside Italy."

Again, both the observations of Dr. Dobson and myself show that the resident natives of Assam less frequently harbour the anchylostomia worms than do the coolies imported from Bengal and other parts of India. Why then should Assam only suffer from *kála-ázar?* It cannot be due to any climatic reasons, for there is no essential difference in this respect between Assam and Lower Bengal.

Once more, if *kála-ázar* is due to a combination of these two diseases, then it would follow that the severity of the epidemic would be in proportion to the intensity of the two factors in any given place or district. Let us see if this is the case. Now it is generally recognised, and this was also Dr. Giles' own experience, that there is plenty of anchylostomiasis in Upper Assam, at least on the tea gardens, while I find that the recent fever mortality of the Sibsagar and Dibrugarh districts, which have not yet been affected by the epidemic, has been higher during the last four years than it was in the Nowgong district before the appearance of *kála-ázar* there. Allowing then for the slight improvement of registration during recent years, it is evident that malarial fevers are at least nearly as prevalent in these upper districts as in Nowgong, apart from *kála-ázar*, while the fever-rate of Darrang was higher than that of

Nowgong in the years before either of them were attacked by *kála-ázar*. Now I have adduced evidence in Section III to show that anchylostomiasis is so rare among the inhabitants of the Nowgong district, apart from the coolies working on the tea gardens and railway, that I have not been able to find a single case which would stand the test of the blood examination, and the administration of thymol and counting the worms passed, so that it may at least be said that this parasite is certainly not more common in the Nowgong district, if it is as common as it is in Tezpur, Sibsagar, and Dibrugarh, while the fever prevalence differs very slightly, apart from *kála-ázar* in the two cases. Yet we have Nowgong literally decimated by the disease, while the medical practitioners of Upper Assam (Sibsagar and Dibrugarh), some of whom had been over twenty years in the country, unanimously testify, after being shown cases of *kála-ázar*, that "the disease appears to be entirely unknown in Upper Assam above Nowgong, and differs in every particular from anchylostomiasis," while Tezpur (excluding the Mangaldai subdivision) was until quite recently free from the epidemic, although I know from personal observation that there is plenty of anchylostomiasis on some of the gardens there, and it used to be more malarious than Nowgong. Thus we find that one district in which anchylostomiasis is very rare among the indigenous inhabitants, who, be it remarked, suffer most severely from *kála-ázar*, and in which, previous to the advent of the epidemic, suffered little, if at all more, from malarious fevers than those of Upper Assam, is more than decimated by the disease, while the latter in which both these diseases are present in much the same, if not in greater, degree than they are in Nowgong, have so far entirely escaped it.

The same thing is true of individual places in the affected area. For example, a certain out-garden in the Nowgong district is in daily communication with the head garden, distanced five miles, and both have suffered from *kála-ázar* for several years past. Yet another independent garden, only

three-quarters of a mile from the affected out-garden, but with which it is not in direct communication, had remained quite free from the disease up to the time of my visit. Yet on this very garden, which escaped the disease, I found more anchylostomiasis (verified by thymol and blood examinations) than on any other garden of the district that I visited, and there was plenty of malaria on both the attacked and escaping gardens, for they are both situated just under low hills. The two supposed contributing diseases were present in this case in a most marked degree, yet not a single case of *kála-ázar* had arisen.

One other conclusive point may be mentioned here. Dr. Sandwith has shown that anchylostomiasis is as common in Egypt to-day as it was in the time of Griesinger (1851), and gives historical evidence to prove that "there is no reason for thinking they were not equally common in the days of the eighteenth dynasty (over three thousand years ago). Dr. Thornhill, of Ceylon, whose writings on the subject of anchylostomiasis are well known (but who labours under the disadvantage, when writing on *kála-ázar*, of never having seen a case of this latter disease), in an answer to some questions that I put to him, for which I am much indebted, says that, in the absence of improved sanitation or migration of the inhabitants, anchylostomiasis would never die out of a place into which it had once been introduced, but would always be increasing in the numbers it attacked, and be slowly spreading to other places; and that if *kála-ázar* dies out of the places it has previously affected in the absence of such conditions as he mentions, then it cannot be due to anchylostomiasis. In this opinion I quite agree with him. I have carefully enquired into the facts; and, as will be shown in Section VI, it is quite certain that the disease does die out nearly, if not quite completely, in the absence of either improved sanitation or migration of the inhabitants. One quotation on the point only need be given here. Mr. Heath, formerly Deputy Commissioner of the Garo Hills, after touring through the country

which suffered, perhaps, more severely than any other part of Assam, namely, the foot of the Garo Hills, writes in 1887 :

"Between Parakhasma and Singimari"(some 70 miles)"it would be hard to find one single case of the real illness."

And again :

"Elsewhere along the border it has cleared away the weak and sickly, leaving the strong, but having done this, has cleared itself away."

Here it is evident that there has been no migration, and I can vouch for the fact of there having been no appreciable improvement of sanitation. But enough has been written on the negative side of the question, which but for the unfortunate errors of the earlier investigation need scarcely have been touched upon, while the facts already given in this report speak more eloquently in disproof of Dr. Giles' conclusions than anything I can write on the subject.

Is the disease only an intense form of malarial fever ?

The way in which the affection has spread steadily up the valley, without there being any evident physical or climatic changes in the districts attacked, and the opinion, which is nearly universal in Assam, that it is in some way or other a communicable disease, have always been the great difficulties in accepting this, the original, and always the most commonly-held, view of the disease. However, in accordance with my plan of work, I have looked to clinical and pathological evidence to prove whether malaria is only one factor in the production of the disease, as it is now universally believed to be, or whether it forms the entire pathological entity, and have regarded the question of its communicability or otherwise as an entirely distinct problem, which requires to be studied in a different way altogether. Leaving then out of the question for the present, whether it is possible to explain the origin and spread of the disease on the hypothesis of its being simply malarial, let us see what light the clinical and pathological facts already given throw on the problem. In passing, I would point out that much of

the confusion which has surrounded this subject, has been due to those who held that the disease was entirely malarial, saying that, therefore, it could not be infectious, while those who held it was infectious, said it could not therefore be entirely malarial, while, as is usually the case, both sides were partly right and partly wrong.

As the clinical and pathological sections of this report consist almost entirely of a description of malaria, it will only be necessary here to enumerate the main points which have been established in them. Firstly, fever accompanied by enlargement of the liver and spleen is the one marked and constant feature of the disease. The type of the fever is indistinguishable from ordinary malarial fever to such a degree, that in the early stages, it is quite impossible by the most minute work to tell whether one is dealing with a case of ordinary malarial fever or the commencement of *kála-ázar*, and it is only when signs of malarial cachexia begin to develope, and it is found that ordinary doses of quinine will not suffice to control the fever, that a positive diagnosis is possible. In the later stages, the disease presents only the usual symptoms of malarial cachexia, and even then, individually considered, it is impossible to distinguish them from chronic malaria, as can be seen at a glance by a comparison of the photographs of *kála-ázar* cases opposite pages 33 and 57, with those of ordinary malarial cachexia as seen in Sylhet, where there is no *kála-ázar*, opposite page 29. How then can *kála-ázar* be diagnosed. Only by the extreme rapidity, a very few months, in which marked cachexia is produced, and by the occurrence of several cases of the disease in the same village or household, where previously such a type of fever was unknown, for it is a remarkable fact, which is well illustrated by the result of the Kokilamukh meeting of planters' doctors already quoted, that, except perhaps under the hills in the southern portions of Goalpara and Kamrup, this type of relapsing malarial fever running into cachexia, is very rare in the Assam Valley. This point will be further illustrated in Section VII, when it will

be shown that the villagers of Assam readily recognise the fever of *kála-ázar* immediately it occurs among them as something quite different from anything they have previously suffered from. The other great distinguishing feature, besides the intensity and frequency of relapse, is the extraordinary tendency it has to run in families, which at once suggests the possibility of the fever being communicable from one person to another, and this feature and its intensity are the only points which I have met with in which *kála-ázar* differs from ordinary malarial fever.

To continue, the anæmia of *kála-ázar* has been shown to be of precisely the same type as has been met with in malarial fever in other parts of India, and is totally different from that of anchylostomiasis, and it is also proportionate to the fever, and lessens at once if this is absent for two or three weeks, which could not be the case if it were produced by anchylostoma. More conclusive still is the fact that in all early cases that were examined, and which subsequently developed into typical cases of the disease, the malarial organisms were easily demonstrated by an examination of the fixed specimens of the blood under an oil-emersion lines of a microscope, and these did not differ from those found by me in simple cases of malarial fever, both in Assam and in Ranchi in Bengal, while similar organisms were also found in all stages of the disease. Further, a series of sections of the liver, spleen, and kidneys showed the constant presence of marked pigmentation, such as has always been considered an absolutely constant change in chronic malaria in all parts of the world, and which to many people is better evidence of the malarial nature of a disease even than the discovery of the plasmodium malariæ itself, while the excess of iron in the liver, as shown by the analysis done in Calcutta, confirmed this point. Once more, the constant and marked enlargement of the spleen, and almost as frequently of the liver also, has never been found in any chronic fever besides malaria, and all the other symptoms and pathological changes met with in

the disease are also found in chronic malaria in other parts of India and elsewhere. Lastly, the seasonal distribution of the disease, as will be shown more fully in the next section, and as can be very well seen in the charts there given, is precisely that of the ordinary malarial fever of the province, with the exception that it is somewhat more extended one in accordance with the greater intensity of the fever. The disease then is a very intense form of malarial fever.

Is *kála-ázar* a new and hitherto undescribed disease?

After what has been written above, it is scarcely necessary to discuss this view. It is, however, open for any one to say that there is something in the disease in addition to malaria, which I have not discovered, but which accounts for the spread of the affection. Whether any will continue to hold this view will depend on the success or otherwise of the evidence and argument given in the latter part of this report, to prove that the origin and spread of the epidemic can be satisfactorily explained on the hypothesis that the disease is entirely malarial. It may, however, be remarked that, apart from the inherent improbability of the supposition that some other disease is so engrafted on the malarial element as to give it the power of infection, for which purpose it must necessarily be a constant feature, and yet does not reveal its presence by producing one single symptom in addition to those caused by malaria, I know of no such combination of diseases in any analogous epidemic affection.

SECTION VI.

THE GENERAL COURSE AND DISTRIBUTION OF THE EPIDEMIC.

In the history of *kála-ázar*, which comprises the first section of this report, a general outline of the course of the epidemic has been given. This it will be necessary to amplify in some particulars in the present one, in which will be discussed the ravages which the disease has caused, its general course in a district, its present distribution, and the directions of its recent spread.

The ravages of the diseases.

Much could be written on this heading, and many quotations could be given, showing how the epidemic has depopulated whole tracts of the terai portions of Goalpara, Kamrup, and Nowgong districts, and has so greatly reduced the population of other parts as to make it impossible for some of the tea gardens of Kamrup to get more than a very small part of the labour they want from local sources, although, before the epidemic affected the district, they had more applicants than they could find employment for; how certain tracts of the Nowgong district, which a few years ago were covered by fields of mustard and populous villages, now only support a few Nepalese people who graze their cattle on the once-cultivated land; how the very name *kála-ázar* strikes such terror into the heart of natives of the affected parts, that whole villages have deserted their homes in order to escape from the disease, and have even migrated to a different district, and wretched sufferers from the fever have been turned out of the villages in which they lived and bandied about from one to another, no one being willing to admit them for fear of catching the fever themselves, while parts of villages have cut off all communication with their relations in a neighbouring portion of the same village for fear of infection; how the Garos were commonly credited in former days with taking affected persons

out into the jungle, and after making them unconscious with drink, setting fire to the temporary huts in which they were placed, and so burning them to death, while, even in the present day, I have myself seen a patient placed in a grass hut in the middle of the dry-up rice-fields, a little way outside the village, as the people would not allow her to come inside it, even to the house of her own father; but still more eloquent in testifying to the fearful results of the epidemic on any district over which it passes like a fell scourge, are the following figures showing the actual decrease in the inhabitants of the affected parts between one census and another, and the area of land that has fallen out of cultivation directly owing to this decrease of the population.

When the figures of the census of 1881 are compared with those of 1891, it appears that in the Goalpara district, which felt the full force of the epidemic within the decade between the two censuses—

"The indigenous population shows a decrease of 20,449, or 4·8 per cent., of which 8,344, or 1·9 per cent., is attributable to an increase in the number of emigrants to other districts, and the remainder to the very heavy mortality due to the spread of *kála-ázar*. The actual loss is much greater, as will appear if we consider the changes in the population of each subdivision separately. In the sadr division (on the north side of the river), where the disease has not spread to any great extent, and there has, moreover, been a considerable immigration from the neighbouring Bengal districts, there has been an increase of 35,771 persons, or 19·36 per cent. In the Goalpara subdivision (on the south side of the river), on the other hand, the ravages of *kála-ázar* are chiefly responsible for a reduction of 29,699 persons, or 18·08 per cent."

Again, with regard to Kamrup it is recorded that, allowing for the increase in the number of emigrants—

"The falling off in the indigenous population is reduced to 10,245, or 1·6 per cent., which is wholly owing to the mortality from *kála-ázar*. Between the years 1872 and 1881, the number of the inhabitants rose from 561, 681 to 644, 960—an advance of 14·83 per cent.—and, although this was in part due to the more thorough counting of the people which was effected in 1881, there is no doubt

that there was a substantial increase of population during these nine years. The only cause which has prevented further growth is the mortality from this disease, but for which it is not unreasonable to suppose that a further advance of at least 10 per cent. would have been recorded. On this assumption the population now recorded is less by 75,000 persons than it would have been had there been no deaths from *kála-ázar.*"

Here again, the figures for the north and south banks must be taken separately in order to see the full effect of the disease, thus—

"The north bank has been comparatively free from *kála-ázar*, and its population has been slowly growing. (Increase in ten years 2·04 per cent.). The south bank, on other hand, which contains the affected tracts, shows a very heavy decrease. Its population in 1891 was 172,125, and this has now fallen to 151,802, or by nearly 12 per cent. If figures were available to compare the district born in this track in 1881 with those now returned, the decrease would be greater still, as this part of the district contains the town of Gauhati, and almost all the tea gardens, so that its immigrant population is larger than in 1881, and its district born population less by the same amount."

It must be remembered, too, that this district was still suffering to some extent from the epidemic at the time of the 1891 census, so that the full loss of population is not shown in these figures. In Nowgong, on the other hand, which had not been appreciably affected up to the time of the 1891 census, there was, allowing for emigration from the district, a natural increase of 32,518, or 10·4 per cent. The next census will tell a very different tale with regard to this district, and it will not be until its results are known that any correct estimation of the degree to which it has suffered from the epidemic during the last seven years, will be possible. The same remark applies to the Mangaldai subdivision of Darrang.

There was no regular census in the whole of the Garo Hills district in 1881, but a census was taken of the plains portion, and in the hills an estimate was formed by applying to the ascertained number of houses the average population

per house found in certain test villages, and it is recorded that—

"Including persons born in the Garo Hills, but censused elsewhere, the natural growth of the population amounts to 12·21 per cent. in the ten years, or 1·14 per cent. per annum. As the district has in parts suffered very severely from *kála-ázar*, it is improbable that this increase is altogether real. It seems likely that that part at least is due to the population having been slightly under-estimated in 1881."

Allowing for this under-estimation, it is still evident that there must have been some increase in the population during this decade, which seems to show that some of the loss that must have occurred during the prevalence of the epidemic, had been made up during the later eighties, when the epidemic had largely, if not entirely, died out, and this affords hope of a similar phenomena being seen in the next census returns for the Goalpara and Kamrup districts.

For the following figures, showing the extent to which land has fallen out of cultivation in the affected districts, I have to thank Mr. Gait, the Secretary to the Chief Commissioner of Assam, to whom I am indebted for much information with regard to this and other particulars.

In the Gauhati subdivision, the number of *bighas* of land under cultivation in 1887-88 was 379,377, but three years later, in 1890-91, this number had fallen by 10,946, but in 1894-95, about half of this loss had been regained. It must be remembered that all of the country comprised in this subdivision did not suffer from the disease, and in particular mauzas the loss was proportionately much greater. During the same years there had been a smaller decrease in the Barpeta subdivision, which was much less affected by the epidemic. In the affected parts of Mangaldai, namely, the Pathorighat and Mangaldai tahsils, there has been a steady decrease year by year from 208,096 *bighas* under cultivation in 1891-92 to 182,149 in 1896-97, or a loss of 25,952 *bighas*, which is just about one-quarter of the total, although there is still a great demand for land in this densely-populated portion

of the district. In Nowgong, the whole of which has been affected by the epidemic, although it is not over there yet, there has been a decrease of 29,477 *bighas* of land under cultivation since 1891-92, in which year there were 150,465 *bighas* rented, while the average of the years 1890—92 were 23,024 more than that the average of the years 1894-96—a most notable decrease which corresponds with a loss of revenue of about one-fifth of the total. These figures speak for themselves, and when it is remembered that, unless there are an unusually large number of deaths in a village, the survivors take up the land of those who have died, then some faint idea can be formed as to the amount of mortality and sickness such a falling-off in land cultivation and of revenue must indicate.

From the figures given above, it will be evident that some very marked effect on the death-rate of the affected districts ought to be noticeable during the epidemic years in any district. Such is indeed found to be the case when the number of deaths returned under the heading of "Fever" (including those returned as *kála-ázar* during the last five years since this has been made into a separate heading), is examined.

The chart opposite this page shows the monthly death-rate from fever and *kála ázar* combined during the years it was prevalent in the Goalpara, Kamrup, and Nowgong districts, one under the other in the order named from above downwards. It was only in September 1891 that the deaths from *kála-ázar* were separately returned, and since that date in the Nowgong district there has been a steady decline in the number of deaths returned under the head of Fever, as those returned as *kála-ázar*, has increased. It is evident then that many deaths from ordinary fevers have been returned under the head of *Kála-ázar*, so that it would be incorrect to take the latter figures as showing the increased death-rate from the epidemic disease. By combining both together and comparing the total with the former death-rate from ordinary

fevers alone in the period before *kála-ázar* appeared in the district, an approximately correct estimate may be formed of the mortality which is caused by the epidemic; but it must be borne in mind that the vital statistics of the two last affected districts more especially, are very far from being accurate, and the figures obtained are only of relative value for use in comparing one year with another, but cannot be taken as giving nearly the real total of the deaths from fevers in these districts. The chart, however, shows very well the wave of increased fever death-rate which overtook, one after the other, the districts named, and also the monthly maxima and minima of fever death-rates. I worked out separately the monthly distribution of the deaths returned under the heading of *Kála-ázar* in the Nowgong district, and this showed the same seasonal distribution as that of the ordinary fevers, except that it was a somewhat more extended one, a variation from the normal that has been observed in other epidemics of malarial fever, such as the "Bardwan fever."

A study of the chart shows that the upper tracing, that of the Goalpara district (that of the Garo Hills cannot be given, as there was no registration of deaths there at the time of the outbreak) began to rise markedly in 1883, which is just the time that this part began to suffer severely from the epidemic, and the next three years the death-rate was at its height, declined slightly in the two following years, and in 1889 it reached much the same height as it was at in 1882. But for the year 1887 being a very unhealthy one in many parts of the province, the decline would probably have been seen in that year. It will be noticed that in the subsequent seven years the fever-rate has never reached to nearly the height that it attained to the epidemic *kála-ázar* years, in spite of the fact that only the southern portion of the district suffered severely from the epidemic, with the exception of 1892, in which the death-rate under the heading of Fevers was greatly increased by a severe outbreak of influenza. When it is also mentioned that some allowance must be made for

mprovement of registration during these latter years, it will be evident that the death-rate from fevers was very much increased during the prevalence of *kála-ázar* in the Goalpara district. It is necessary to insist on this point, because Dr. Giles, judging by the absence of any marked increase in the general death-rate, including that most variable factor, cholera, of Kamrup since it had been attacked by *kála-ázar*, came to the conclusion that the disease "enhances the sick-rate more than the mortality"—a statement that is directly contrary to the very high percentage of deaths among those suffering from this disease. He, however, admits that "in the registers all cases of *kála-ázar* are of course included under this term," namely, "fever," and he goes on to point out, what is very evident, that deaths from many different diseases, of which fever is the main symptom, will also be returned under this heading. It is then evident that the increased death-rate from fevers during these epidemic years must have been due to *kála-ázar*. The same sequence of events is seen in the second curve, but here the rise is not very evident until the upper one has begun to fall, namely, in the year 1888, and remains very high in 1889 and 1890, after which it also begins to decline, with the exception again of the influenza year 1892, and reaches its normal in 1895, while 1894 was also a very unhealthy year, not only in Assam, but in many other parts of India; but even then the fever death-rate did not reach the height that it did in the *kála-ázar* years, and the peak of the curve is not so broad as it was in those years, showing that the excess of sickness was of a shorter duration. Coming to the third curve, the rise in the Nowgong district is seen to begin in 1893, although part of the rise of the influenza year 1892 might have been due to *kála-ázar*, especially the increased height towards the end of the year; 1894-1895 were very bad years, but there was a slight decline in 1896, which is pretty sure, judging from what I have seen in the district, to be more marked next year.

Another point which can be seen from the chart, is that during the worst years in any district, the death-rate rises earlier than it does in ordinary years. Thus, in 1889 in Kamrup, and in 1895 in Nowgong, there is a well marked rise as early as the month of April, and, moreover, the decline is often later than in the non-epidemic years, both facts indicating a longer seasonal distribution of the disease.

It will have been noticed that during the height of the epidemic in any one district, there are three or four years in succession during which the mortality from fevers is not only much higher than the average of the preceding or following years, but in each of which it is actually higher than any one exceptionally unhealthy year at other times. Again, the increased mortality from fevers does not become marked until two or three years after the district has been invaded by the disease, that is, until it has had time to diffuse itself over a considerable portion of its area, so that the returns from whole districts are of no use in helping to detect the presence of the disease at an early period of its being attacked. For this purpose, the figures of individual mauzas must be examined, but still better the tahsildars and mauzadars themselves can be relied on to furnish very early information as to which villages in their respective portions of the district are affected by the epidemic fever, while the revenue returns are influenced as soon as there has been any great death-rate in any group of them.

The seasonal distribution of the disease.

This, as has already been observed, is very well shown in the chart opposite page 133, but the point is one of such extreme importance, that it must be amplified somewhat here. In Section III it was mentioned that the seasonal distribution of the disease is exactly that of ordinary malarial fever, except that it is rather a more extended one, and it was mentioned that the beginning and end of the rains were the most fatal seasons of the year. Extended inquiries in the affected villages and tea gardens confirm the impression that nearly all new cases of the disease occur

during the rainy season, beginning with the fall of the first few inches of rain, which usually takes place in April or some time in March. Many chronic cases died off early in the cold weather from lung complications; but from November to March most of the cases lose their fever, only too often to relapse at the onset of the next rainy season. This great decrease of the disease in the cold weather is a most remarkable fact, and one which most strongly supports the view of the malarial nature of the affection. It is also of the greatest practical importance, as will be seen in Section X, and it is for this reason that it is again insisted on in this place.

Relation of the distribution of the disease to the ground water-level.

Particular attention was paid to this point in order to determine if any of the peculiarities of the distribution of the disease could be explained in by it, for I have shown elsewhere (*Indian Medical Gazette*, 1896, page 49) that the prevalence of ordinary malarial fever varies with the rise and fall of the ground water in some places at least, and I have confirmed these observations in the Nowgong district. It may be said at once that no relation whatever has been made out between the two phenomena in the case of *kála-ázar* except that in places situated just under the hills, that is, in terai lands, the disease is more fatal, and has a greater tendency to completely wipe out the inhabitants. I have not been able to make out any constant relationship between water-logging of the land and the intensity of the epidemic, except that the disease is somewhat worse in low lying parts. It can, moreover, no longer be said that the disease is only found in terai lands, as might have been considered to be the case as long as the affection was limited to the strip of country between the Garo and Khasia Hills and the Brahmaputra river, for Nowgong cannot be placed in this class, yet it has suffered most severely. Again, it must be recognised that there has been absolutely no change whatever in the soil or ground water-level of this district to account for its

invasion by an epidemic of malarial fever. No part of the district has escaped, but it has been most severe, and has spread most rapidly in those parts which are most thickly peopled, namely, all along the banks of the Kulung river.

Relation to sanitary conditions.

Here, again, there has been absolutely no change to account for the origin or spread of the disease in any of the affected districts, and what little change there has been is for the better, yet this frightful epidemic of fever has spread itself steadily over the country in a manner that has scarcely a parallel. It cannot be due to the water-supply, for this again has improved rather than deteriorated, and I may say that in the course of my travels I saw nothing that would lend any support to the hypothesis that the disease is spread by means of water, but, on the contrary, I found some villages, or even parts of villages, that had so far escaped the disease, although other parts of the same village, or neighbouring ones with the same, or a similar water-supply, had suffered for several years from the pestilence. It must also be borne in mind that the disease dies out of villages and districts in the absence of any change in the local conditions. I had expected to have found some local difference between affected and unaffected villages; but in this I was in error, for no such differences are to be detected, and, as will be shown in the next section, the mode in which the disease really spreads is quite independent of any such changes or differences.

The distribution in the villages.

This rather belongs to the next section, but it may be stated here that I have been able to confirm the facts pointed out by Dr. Giles, namely, that one part, or certain houses, of a village may suffer severely from the disease, while other houses in the same village may entirely escape it for some considerable time, and that although all are living under precisely the same conditions.

THE GENERAL DISTRIBUTION OF THE DISEASE IN RELATION TO ITS MODE OF SPREAD.

The reports of the Civil Surgeons of the affected districts have been examined as far back as they are available, namely, to 1885, and all other possible material has been consulted in order to try and trace the exact spread of the disease year by year, but the results obtained have been somewhat disappointing. In order to avoid a wearisome repetition of dates and names of places, the facts, as far as they could be obtained, have been embodied in the accompanying map of the Assam Valley, which will be of use in connection with the first and eighth sections, as well as this one, and only the recent spread of the disease, as far as it bears on the way in which it travels, will be given here. The figures in the map represent the years in which it invaded different parts, and thus enable the spread of the disease year by year to be followed. Every one of them is based either on some actual record, on my own inquiries, or on the figures of the fever mortality in the different mauzas, which have been examined as far back as they were obtainable.

We have seen in Section I how the epidemic spread from the foot of the Garo Hills to the neighbouring portion of Goalpara, and from this district up the grand trunk road into Kamrup, and thence to Gauhati, overwhelming almost every village in its course; but, except in the case of the low Garo Hills, never extending far from level of the Brahmaputra Valley. The north bank, on the other hand, suffered comparatively little, owing to the population being much more sparsely and irregularly distributed, for in that part long stretches of jungle intervene between one group of villages and another, such as has always, throughout the twenty years that the fever wave has been spreading up the valley, formed a great obstacle to its passage, and hence the disease has been obliged to cross the river again and again in order to attack the scattered groups of villages on its north bank.

The recent spread of the epidemic.

When the disease once got a firm hold on Gauhati, it began to spread in various directions. In the first place, it attacked some villages just opposite the town on the north bank of the river severely. Mangaldai was invaded both from the last-named villages and in those near the Rangamati-ghát, which is the main line of communication between this subdivision and Gauhati town, and one along which there is daily intercourse by means of some three hours' run by the mail steamers. The disease was even introduced directly as far as Mangaldai town itself in this way, as will be shown in the next section; and between the years 1890 and 1894, it diffused itself over the whole of the more densely-populated south-western portions, and has recently broken out in one or two places in the northern and eastern parts of this subdivision, having been carried north by some Kachari labourers, and has now affected a tea garden near the hills. This invasion of Mangaldai is of great interest, as it appears to have been more extensive and fatal here than in any other portion of the north bank of the river, although Barpeta has also suffered severely, these two divisions being mainly low-lying rice-land and thickly populated. Between Mangaldai and Tezpur, there is a stretch of some 30 miles of jungle with very few inhabitants, and this has served to check the spread of the disease to a very great extent in that direction, so that Tezpur has not been reached in that way.

At the same time that the disease was passing into North Gauhati and Mangaldai, it was also continuing its general easterly direction along the south bank of the river into the Nowgong district, and here it at first followed its usual course and crept up along the foot of the hills, but later on it branched out in a north-easterly direction towards the Brahmaputra river along the course of grand trunk road and the thickly-populated banks of the Kulung river. In this district its spread is of great interest, as hitherto it had been more or less confined to a narrow strip of country

between the Garo and Khasi Hills and the Brahmaputra river, but now it was able to wander over a very much wider area. It is all the more unfortunate that there are so few details on record as to the order in which the different parts of the district were invaded year by year; but from what I have been able to find out in the course of my inquiries, there is no doubt that the following is broadly the way in which it has spread. The disease first appeared in some villages of the Jagi outpost, near the Kamrup boundary, in 1889. In 1890, it had spread somewhat further westward, and at the end of this year it also appeared in Nowgong town. In 1891, it had reached Nokhola and Roha, and was more prevalent in Nowgong town. In 1892, Nowgong town suffered severely, and the villages and mauzas all around it were affected; and by 1893, it had spread widely over the district, although it is still noted to be attacking new villages. It was not until 1894 that it reached the Silghat, or north-eastern end of the district. The general distribution of the disease was such that the larger places were first attacked, that is, before many of the smaller villages between them suffered, and these were subsequently filled in as it were. This seems to always be the way in which the affection spreads, but no general move takes place from one part of the district to another until the great majority of the villages of the first part are badly affected by the epidemic, and then it will begin to creep insensibly into the next part of the district, the same order of proceedings being repeated.

In the case of the villages along the Kulung river, which form, for all practical purposes, one continuous straggling community extending throughout the whole length of the district, and which must accommodate a very considerable proportion of its inhabitants, the disease seems to have spread along them in a steady stream, dying out in the first-affected portions as it passed on, and its march being occasionally rendered somewhat irregular by large places on the road or bank of the river, such as Nokhola, Roha, Nowgong itself, and Pura-

nigodam, 8 miles to the west of the former, being comparatively early attacked. It has only reached the extreme east end of this series of villages during the last two or three years, and in the last portion between the place where the grand trunk road crosses the river, 4 miles from Silghat, and a place called Joklabanda, some 4 miles to the east of Silghat; but on the Kulung river, the disease is still on the increase. Beyond the last-named place there stretches a long tract of jungle extending for some 40 miles with scarcely a village in it, and here the spread of the disease has ceased for the time being at any rate.

Along the base of the hills to the south-west of the district, on the other hand, the disease early spread as far as Karakhana and Lunka (on the Assam-Bengal Railway), and it has since got at least as far as Lumding on the western border of the Nambar forest—a dense, uninhabited wood, which stretches between the Mikir and Naga Hills for many miles, reaching up to the Golaghat district. Across this forest there is no traffic, but the railway is being pushed through it, but will not be open for some years yet, by which time all danger of the disease spreading up that way should be at an end by reason of its dying out of the Nowgong district, which it is now taking place, especially in the southern portion of the district. The Mikir Hills stand then as a bulwark between the attacked portions of district of Nowgong and the subdivision of Golaghat, and indeed the whole of the Sibsagar and Dibrugarh districts, for I visited Golaghat and travelled back to Nowgong along the narrow tract of country between the Mikir Hills and the Brahmaputra river, but found no cases of *kála-ázar* there. The grand trunk road runs through this part, but there is little traffic on it, and for some years past a careful watch has been kept for any persons coming up the road suffering from the disease, and in one instance a whole village, which had migrated from Nowgong, was found to be suffering from it, and was sent back, and this was the very village that had

infected two tea gardens in the Nowgong district. But for this constant vigilance and action, it is exceedingly probable that the epidemic would have got a hold on Golaghat by this time; but as it is, although there were more deaths than usual returned from fever in the year 1895, the figures for 1896 show a decline to the normal level, and this is true of each mauza considered separately, and the decline is most marked in just those parts which would be the first to be invaded by the disease, so that it is certain that this subdivision is not yet affected by the epidemic to any degree, although it is not impossible that it may have begun in a few isolated villages. There is also great danger of the disease gaining admission by the river, just as it has done in the case of Mangaldai, Tezpur, and Bishnath, etc., and it is certain to surmount the obstacles in one way or other before very long, if no steps are taken to prevent its doing so. With regard to the south bank of the river then, it may be said that the present condition of things is more favourable to the success of vigorous measures directed against the spread of the disease, than they have never been since the epidemic first attracted attention in the early eighties; and it is not too much to say that if the present opportunity is neglected, the disease will certainly spread up to the end of the valley beyond Dibrugarh, and from what I have seen of the character of the country in the Sibsagar district, from the alluvial nature of the whole of the soil of the valley, and from the recorded death-rate from fever in these districts, I see no reason to believe that the epidemic is likely to occasion any less loss of life and revenue in these more easterly districts than it has already done in Nowgong, while the vastly greater importance of the tea industry in the upper part of the valley will double the calamity produced by such a spread of the affection.

The recent extension of the disease in the Darrang district.

The introduction and early spread of the disease in the Mangaldai subdivision has already been described, but it has been mentioned that it has recently appeared among the

Kacharis of the north of the district, and has broken out among some of the labourers of this tribe on a tea garden near the hills.

In Tezpur the disease has been introduced within the last three years, and information has been obtained with regard to many of the first cases, which is of great interest and importance. The town itself, as might have been expected, was the first to suffer; but fortunately, although several imported cases have died there, it does not yet seem to have spread extensively among the regular inhabitants, although some, in whose houses imported cases have lived, have suffered and died of the disease. With regard to its introduction into Tezpur town, it will suffice to record that the Civil Surgeon, Dr. Macnamara, writes in his report for 1896 as follows:

"As far as I can ascertain, there are six or eight cases in the town, all of which are traceable to Nowgong. They form two groups, one on the river bank above the town, and the other in the town on the north side of the dispensary."

I can confirm this observation from personal observation, for Dr. Macnamara was kind enough to take me to see them, and it is worthy of note that the first-mentioned group is in a Dom village above the town, at the very spot where a ferry from the Nowgong side lands its passengers, and the villagers have asked to have this ferry moved from near them, on the ground that it has been the occasion of the disease being introduced amongst them.

In the country around Tezpur, I found the disease had attacked nearly all the villages to the north-west of the town, extending out for at least 5 miles, and there are other affected villages, considerably further north of the town among the tea gardens. The number of cases that I saw in any one village was small, but there can be no doubt that the epidemic has got a hold on the district, although it may be hoped it will follow the general rule, to which Mangaldai was an exception in not being so severe as it is on the south bank.

Bishnath, which is to the east of Tezpur, is also beginning

to be affected by the disease, a village about a mile from the ghát having been attacked during the last rainy season (1896), while I have also been informed of an outbreak of the disease in some villages on the Bishnath side of Tezpur between the two places. I know of no outbreak of the disease to the east of Bishnath.

Summary of the broad facts relating to the spread of the disease.

The facts given in this section may be summed up in the following propositions:

(1) The epidemic of the malarial fever, which is known in Assam under the name of *kála-ázar*, shows itself in a district as a wave of increased mortality returned under the head of "Fever," which takes two or three years to reach its height, which is then maintained for another three years, after which it declines, leaving behind it a decrease both in the population, the amount of land under cultivation, and the land revenue, together with tracts of country from which all the inhabitants have been cleared off. The seasonal distribution of the disease is that of ordinary malarial fever, only slightly more extended.

(2) There has been no alteration in either the climatic or physical condition, or in the sanitary state of the affected districts to account for the origin or spread of the disease.

(3) The disease spread steadily up the south bank of the Brahmaputra river, along the grand trunk road, until it reached Nowgong, where it spread both along the foot of the hills directly east and also north-east along the grand trunk road and the thickly-populated banks of the Kulung and Kopeli rivers, but has been checked by the uninhabited tracts of jungle stretching east of Silghat and Joklabanda.

(4) The districts on the north bank of the Brahmaputra river have suffered less severely than those on the south bank, owing to their being more sparsely populated; but wherever free inter-communication has existed between groups of villages on the north bank and the affected tracts on the south side, the

disease has spread to the north bank, as has been seen in the case of parts of Goalpara, Barpeta, Mangaldai, Tezpur, and Bishnath.

(5) Throughout its whole course the extension of the epidemic has presented a persistent relationship to lines of communication, both on land and in its frequent crossing of the river, which can only be explained on the supposition that it is spread by means of human intercourse, and hence is a communicable disease.

The precise way in which it is carried must be left for consideration in the next section.

SECTION VII.

ON THE COMMUNICABILITY OF KÁLA-ÁZAR.

We have seen in the last section that the general distribution of *kála-ázar* is that of a communicable disease, and that it always spreads along the lines of traffic or human intercourse, while it is checked by any tract of uninhabited country through which there is no road; that it has crossed the Brahmaputra river on several occasions, always in the lines of the greatest traffic, and that the way it has gradually crept up the Assam Valley, cannot be explained on any local change of the soil or sanitary conditions of the affected districts, while the natives themselves, and also many of the Europeans who have come into intimate contact with the disease, agree in believing that the disease is in some way communicable from man to man, while this opinion was confirmed by Dr. Giles as a result of his investigation in 1889-90. It is then of the first importance to try and trace in what way the disease is transmitted from place to place. In order to ascertain in what way, if any, its progress can be checked, for it is evident that its spread cannot be explained on the ground of its being anchylostomiasis, as this is not the cause of the affection.

That this has not yet been done, and that there are some who are still sceptical as to the disease being communicable at all, is due to the great difficulties in tracing the spread of an affection, which must be present in any individual case for a long period, always several weeks, and often two or three months before it is recognisable from such a universally present disease as ordinary malarial fever, and from which it may, individually considered, be absolutely indistinguishable throughout its whole course, and sometimes whose very presence in a village, or even on a tea garden, has not been recognised until it had produced a great increase in the death-rate, which may take it at least a year to bring about. When

it is remembered that outbreaks of such diseases as cholera and plague, which have an incubation of period of only a few days, and which are so rapidly fatal as to immediately attract attention, are yet not unfrequently impossible to trace to their original source, it is not surprising that it has been found difficult hitherto to conclusively prove the communicability of *kála-ázar*, although I have met very few in Assam who were not perfectly convinced that it is so.

In the course of my earlier travels in Assam, I every now-and-then met with instances in which the history of infection seemed to be very clear, and which encouraged me to hope that a systematic inquiry into this part of the subject would produce interesting and very valuable results. A tour was accordingly undertaken through certain parts of the Nowgong, Mangaldai, and Tezpur districts, that is, those which had most recently been attacked by the epidemic, in order to test this point, and it may be said at once that I was much astonished with the frequency with which a clear history of the introduction of the disease by means of human intercourse could be obtained, and still more by the remarkable way in which the villagers very often recognised the first case, which occurred amongst them, as something quite different from anything they had previously experienced within the memory of the oldest. In the great majority of villages, in which either the headman or a relation of the first case of the disease could be questioned, a clear history was obtained. This will be best illustrated by the narration of a morning's work, and then instances which illustrate the various ways in which the disease is spread, and other points of importance, will be given.

A morning's inquiries in some affected villages.

In the course of a five hours' tramp around six villages near Lunka, in the south-east corner of the Nowgong district, the following results were obtained. As far as possible, leading questions were avoided, and it was generally only necessary, after

ascertaining how long they had suffered from the disease, to ask how it began, and immediately would come the answer, that the first case was such and such a man, who had returned from such and such a village, which was at the time affected with *kála-ázar*, suffering from fever, and that the next case or two were members of his household, after which it had spread to the others in the village. Notes in short-hand were taken on the spot in each case.

Village L.

The headman of the village informed me that four years ago, in March, his son-in-law came to live in this village from another, four miles away, which was affected with *kála-ázar* at that time, and he had also been in Nowgong town a short time before, where the disease was also very prevalent. At the date of his return he was suffering from fever, which had begun when he was in the neighbouring village. He was the first to die of *kála-azár* in this village, and the next case was that of a man who lived close to him, and who used frequently to come and sit with him when he was ill. After that it spread to the others in the village, but the infection of individual cases was not known. Two-thirds of the people of this village are said by the headman, who is responsible for the registration of the deaths, to have died during the epidemic, but at the present time (December 1896) it is much less, although I saw several well-marked cases. They were inclined to very seriously consider my suggestion to remove their houses to a new site during the cold weather, as they were convinced that the soil and houses were infected. In one house in this village seven of the members of the family had died of the disease, and the other two now have it. They were also certain that any one who lived in a house in which other cases of the disease resided, was very likely to get it, and that it was often spread by orphans, who had lost their parents of the disease, being taken into other houses, which had been previously free from it. In this village, out of five men who took opium regularly before it was attacked by the

disease, four had escaped, and the fifth only got it after seven of his household had suffered from it. Many of them now took it, as they were convinced that those who did so, were both less likely to suffer from it, and also had a better chance of recovering; and they showed me a boy, who had been given three grains of the drug a day while he was suffering from *kála-azár*, and who was now quite well. It is worthy of note that this history of an increased consumption of opium since the appearance of the epidemic, and as a direct result of it, was obtained in all the villages around here, which are inhabited by the Hosais—a people who are addicted to the habit; and on inquiry at the local opium shop, this increased consumption was confirmed.

Village S. A man lived for twelve days in a village on the Jumuna river (only a few miles from here), in which there was *kála-azár* at the time, and got fever whilst there, and returned with it to this village. He and his brother, who lived in the same house with him, were the first to die of the disease in this place, and it afterwards spread through the village. There have been very few fresh cases this year, 1895 having been the worst year.

Village H-a. This village was found to be deserted, although a month previously there were two families here who had three bad cases of the disease among them. I subsequently learnt that two of these had died, and the people had then gone to live in another village not far off, which had, however, previously suffered from the epidemic. It is easy to see how disease might be spread in such a way as this, as, indeed, it often is.

Village H. Had suffered from the epidemic for the last five years; 1895 was the worst year. The first case was that of a man of this village (whose name as usual was given), who was a boatman. He went to Roha (some 30 miles off) and came back with fever, of which he died in this village. The second case was that of a man who used to go and eat in

the house of the first case, and the third and fourth cases were the wife and son, respectively, of the first case. The whole of the family of the second sufferer died of the disease. The first cases occurred during the rainy season. When nearly all the first infected family had died of the disease, another brother came to live with one of them, who had the fever, and this brother also got fever and died of *kála-ázar*.

Kála-ázar has been present here for the last five years.

Village D-n.

The headman of this village first got fever in another village, one mile off, where it was prevalent at that time. This was in the month of January, and on his return to this village, some of his family next got the fever, but it was not until the ensuing rainy season that it spread to others in the village. About 80 persons have died of the disease here.

Village G-a.

This village has suffered for three years from *kála-ázar*, and about half the people have died of the disease. The first case was that of a man who came from Doboka (a large village, some 15 miles from this place) with fever, and died in this village of *kála-ázar*. There was *kála-ázar* in Doboka at the time he got fever there. Two others afterwards died in his house. In the same year, a woman married a man of this village, and came to live here from a neighbouring one. Soon after she came, it was noticed that she had fever, and six months later she died of *kála-ázar*, and her husband left the village. It was subsequently ascertained by the villagers that this woman had lost a husband of the disease before marrying this man. Here then we have two separate infected people coming to live in this village in one year, although it was apparently only through the first that the disease was spread here. In this village again, out of ten or eleven, who took opium before the epidemic fever broke out, none died of the disease, and many more now take it.

This group of villages is situated in a very out-of-the way part of the district, and it will be noticed that in some of them the disease was brought from a distance by persons coming to live in them after having contracted the fever in one or other of the larger and earlier infected places, while others are infected from the neighbouring villages into which the disease was first introduced from some little distance. This is the usual way in which the disease travels, and the very irregular distribution of the affected villages in any part of a district which is just being invaded by the epidemic, is thus accounted for. Subsequently, when once the disease has broken out in a few villages in any place, the frequent inter-communication between the affected and unaffected villages ensures all of them being attacked sooner or later, except in the rare cases where the villagers recognise in time the infectiousness of the disease, and cut off all communication with those who have got it, and then a part of a village may escape, although it is present in the rest of the same village from which it is only separated by two or three hundred yards of rice-fields. Another common mode of conveyance of the disease is through the custom, amounting almost to a religious duty, of the people visiting sick relatives in the neighbouring villages, which very often means eating and sleeping with them. Again, I have met with instances in which the disease was spread by persons who are actually suffering from it, and who have lost all near relatives on whom they were dependent, being taken into the houses of other relations in different and previously unaffected villages. In these and many other ways the disease, when once it has been introduced into a district, spreads slowly and surely, but in a very irregular manner until almost every village suffers.

The day's experience which I have detailed above, taken with facts that had previously come to my knowledge, quite convinced me of the communicability of the disease; but as this is a most important and crucial point, both from the theoretical and practical points of view, I subsequently

tramped over 150 miles from village to village along roads and across country, chiefly in the Mangaldai district (as, owing to the comparatively slow spread of the disease there, it offered the most favourable field for my inquiries), in order to collect convincing evidence on this point. The results confirmed and extended those previously obtained, and left no possibility of doubt in my mind that the popular, and nearly universally-held, view of the communicability of the disease was based on absolutely uncontrovertible facts. A few of the more typical and practically important instances only need be given here, taken from notes made in the villages in course of this part of my inquiries.

The following history of a *basti* (collection of houses) in Mangaldai town is of great interest from several points of view. The first case was that of a boy who was brought by his mother when suffering from the disease to stay with a relative in this village. They brought him in the hope that he might get rid of his fever here. He only stayed two weeks in the village, and then was taken back to Gauhati, from which place he had come, and he died there. At about the same time, another woman brought her son, aged 17, who also had *kála-azár*, and they stayed one month, when they also returned to Gauhati. These events occurred in the cold weather, and up to this time none of the permanent inhabitants of the village had suffered from the disease. A month or two after these two boys had left, two men of the village were attacked by *kála-ázar*. One of these lived in the house in which the first boy had resided, while he was in the village, and a year later his brother also died of the disease. The other man lived in the house in which the young man from Gauhati with *kála-ázar* had resided, but his wife, who was the only person who lived with him, escaped. These two early cases occurred at the beginning of the rainy season, and during the rains it spread to others in the village, and eventually two-thirds of the people died. A year ago, that is, at the beginning of 1896, they decided to move the site

of their village, as the disease was still very fatal among them. They moved to a different piece of land which is between one and two hundred yards from the old site. The deserted site still remains unoccupied, for, when it was proposed to build police lines on it, all the men of the force so strongly objected on the ground of the fear of infection, that the proposal was abandoned. It took these people, who had been reduced from about 600 to only 200, one month to effect the move. They had at the time of my visit been a year in the new place. During the year before they moved, they had 20 deaths from *kála-ázar*, and at the time of their moving, they had two cases, which they took with them. One of these died soon after, but the other recovered. Two new cases only appeared in the last year, one of whom was noticed to have fever very soon after they had moved, the other began early in the rains. Of these last two, the former recovered and the latter died. The two cases who recovered, or were at least alive and much better in January 1897, are shown on the right of the photograph cases at the Mangaldai dispensary, which was taken in August 1896, opposite page 33. I have met with other instances in which whole villages have been moved to new sites during the cold weather, purely in order to get rid of the disease, and it was very commonly done by the Garos years ago for the same reason.

No more conclusive evidence of the communicability of the disease could be wished for than that furnished by this instance; but what is of the greatest interest is the proof which it furnishes of the infection being of an indirect nature in some instances, for the two sufferers from *kála-ázar*, who undoubtedly infected this village, had both left it a month or two before the persons who resided in the very two houses in which they had lived, began to get fever. It is of course possible that the disease may have a long incubation period, but this is very unlikely, for the fact that I have met with an instance in which a man got the fever within three days of arriving in Gauhati, although he only stayed there one week, and then returned to

his own village, which previously had not had a case, he died of the disease and infected the place, which shows that the incubation period may be very short, and other instances point in the same way. The houses must in this case have been infected during the residence of these cases, and when the rains came on, the infection became active, which is in accordance with the much greater prevalence of the disease during this season of the year. The good effect of moving the site of the village during the cold weather, as is shown by their much greater freedom from the disease in the ensuing rainy season, is of the utmost importance, and will be referred to again.

The peculiar distribution of the affected villages was well seen around Mangaldai. Thus, although many of the villages had been affected from four to six years, yet others, a few miles off, or even in the middle of those affected, had so far entirely escaped. Thus, in walking 11 miles due north from Mangaldai town, along a main road nearly all the villages met with in the first 4 miles, had suffered very badly from the epidemic for several years past; in the next 3 miles, some of them only had suffered, and these less severely, while in the last 4 miles I could not find a single case. The middle zone was the most interesting and instructive, as the following case will show:—A village, 4 miles from Mangaldai, had been attacked six years ago, and had lost 100 out of 600 inhabitants. Three miles further north was a village which had only had one case, in the person of a girl, who had married a man in the village nine months before having come from a village, 4 miles further south, which had suffered very severely from the disease. It was found, very soon after her arrival, that she had had fever in her own village, and a few months later she became very bad with it, so the people of the village turned her out, as they said she had *kála-ázar*. She went back to her old village, but here they also would not admit her. She then went to her father, who lived in a village just across the road from that in which her husband

lived; but as this village was also free from the disease, the people objected to having her, but her father built a temporary hut for her outside the village, in the middle of the rice-fields, which were now dried up. I saw all the people in connection with this case, and also the patient herself, who was in a terribly wasted condition, and had a very large spleen and liver, and dropsy of the feet, and I tried to get her sent to the hospital, but this the father said would be against his religion as long as he was able to support her. I mention this case to show the terror the people have of the disease, and the extremes they will go to in order to prevent it being introduced into their villages, because I am convinced that in the Mangaldai district, the spread of the epidemic has been partially checked by means of this kind of action on the part of the people. A similar condition of things was found along another road, which runs parallel to the one that I have just described, but separated from it by a river and about three miles of country. This is the main road north from the Rangamati-ghát, and about 11 miles up it there is a village, two parts of which have had the disease for four or five years, while the third part, which is separated from the rest by about two hundred yards of rice-land, has entirely escaped. On inquiry here the headman told me that ever since the disease had been prevalent in this part of the district, he would not let any one from his part of the village hold any communication with the infected villages, and he carried this to such a degree, that when any of his people had relatives ill with the fever, he would not allow them to be visited, even if they lived in the other parts of their own village; and if they died, their funeral was not attended. This is of course against all their customs and religion, and it must have required a very strong belief in the communicability of the disease for such a thing to be possible. The good effect was, however, undoubted, as every other village, but this one in the immediate vicinity had suffered severely from the epidemics. This man had an idea that if any one bought any article of value which had belonged to a

man who had died of *kála-ázar*, he would be liable to get the disease. This was also so strongly the belief in the Kamrup district a few years back, that the property and even the cattle of persons who had died of *kála-ázar*, were left untouched by not only the relatives of the deceased, but by everyone else for fear of infection. That this is one of the ways in which the disease may be spread, is very likely, when we consider that the bargaining over such sales may last two or three days, and will be carried on in the affected village, and usually in the infected house, and I found an instance in which the infection had probably taken place in this way in another part of this very village. A few miles further north along this same road was a small village of Brahmins, who had no relations or people of their caste in the neighbourhood, and who were free of the disease. Quite close to them, but the other side of the road, was a collection of houses in which some fifty people remained, while some forty had died of *kála-ázar*. The history of the introduction here was very instructive. At first they said they did not know how it came, although they gave me the name of the first case. On enquiry I found the wife of this man was still alive, and on questioning her I ascertained that her deceased husband had a brother suffering from the disease, who used to visit him, and eat and sleep in his house while he was ill with the fever, although he lived in another village, two and a half miles away. My informant's husband then got the disease from his brother, and his son was the next victim, after which it spread to other families. Here, again, another part of this village, which was situated some quarter of a mile away, cut off all communication with this affected part, and still remained free from the epidemic, although the affected part had been attacked four years ago, and was now rather better than it had been. A big village further still to the north had suffered severely for four years, but a small one of a different caste near them had so far escaped, while only some two miles further up nearly every village was free of the disease, and to the west two or three miles in from the road

the disease had not reached so far north by three or four miles as it had along the main road.

The above facts are sufficient to prove that when all communication is cut off between one village and another, either by caste restrictions or by voluntary abstention, from visiting between related villages, then the spread of the disease is greatly, or even entirely, impeded. Further, when there is any special communication between a village and an infected part of the country, which is not shared in by the surrounding villages, then the communicating village may alone be attacked, as the following instance shows. On enquiry from the tahsildar of the Kalgaon division as to what villages were affected in his part of the district (which as yet is scarcely touched by the disease), he gave me the names of four or five in which, he said, the disease was beginning. As I was anxious to test the correctness of his information, as well as to visit some of the most recently-affected places, I made a point of going to some of those he had indicated. I found his information was perfectly correct, for, owing to his being responsible for the collection of the land revenue, he is immediately made aware of any unusual mortality in the villages under his care by the consequent reduction in the amount of land cultivated. One of these places was called Berhampore, and after some difficulty I found it more than two miles from any road, and surrounded by unaffected villages; in fact, the last place which one would have expected to have been singled out for attack by the disease. When, however, I ascertained that it was a farm belonging to people of the Sipajhar district, which had suffered very severely from the disease for the last six or seven years, the mystery was explained, and on enquiry I found the first cases in this village were in persons who had been brought from Sipajhar in the hope of the change doing them good, and other cases had occurred in some of the houses in which they had lived.

Another very instructive case is the following. It has been mentioned in Section VI that there is a long stretch of very sparsely inhabited jungle to the west of Mangaldai town

between it and Tezpur, which the epidemic has not yet crossed. There is, however, a village in about the centre of this tract, 17 miles from Mangaldai town, which has recently become infected. The first case was a policeman who returned to his home here from Mangaldai town on sick leave in October 1896, suffering from *kála-ázar*. In January 1897, a member of his household had also got the disease. Here we see the disease carried 17 miles, but it must be noted that Mangaldai town and district had suffered from the epidemic for six years before it was carried over this stretch of jungle.

A few days later I visited the Sipajhar district, and found that the disease is now dying out of this part ; but in the course of a walk across country there, I was much struck by seeing patches of jungle grass of from one to three years' growth in the midst of stretches of rice-fields. On looking closer, the low ridges, which serve to retain the water on such ground during the rainy season, were found to also traverse these patches, showing that they had been cultivated within a short period. On enquiry it was ascertained that this jungle was growing on land which had formerly been cultivated by families, all of whose working members had died of the epidemic ; and as there were not sufficient adults left in the villages to take up this land, it had fallen out of cultivation. Further experience showed that very fairly accurate estimations of the proportion of deaths from *kála-ázar* in these villages could be made from the condition of the fields around them. The extent of this may be gathered from the fact that the amount of land under cultivation in this mauza alone has fallen from 16,569 *bighas* in 1891-92 to 13,636 in 1895-96, or a decrease of 3,333, just about one-sixth of the whole.

It is also a fact that nearly all the first cases which occurred in Tezpur town were traceable to Nowgong or Gauhati. Thus, according to a report by Dr. Macnamara, the disease was first introduced into a house opposite the club, by a man from Gauhati suffering from *kála-ázar*, of which he subsequently died, living in this house for three months, while a second

Gauhati man stayed there for two weeks and went away very ill with fever. Then a boy in this house died of *kála-ázar*, after an illness of two years, and subsequently three others died of the disease in the same house. I myself found there had been two deaths from the disease in a row of native houses in the bazar within one week, both in the persons of people who had come from Nowgong suffering from the disease. In short, all the facts with regard to the recent infection of Tezpur town go to prove that the disease has been introduced by sufferers from the disease coming to live here from the Nowgong and Gauhati districts. I have met with an instance in which the disease was apparently introduced into a village some 30 miles from Nowgong station by a school boy returning from there with the disease, and a place north of Gauhati is recorded as having become infected in a similar way.

Enough has been written to show, both from specific instances and from the peculiar distribution of the disease in neighbouring villages, that it is a communicable affection, and that the infection is often of an indirect nature. The explanation of the facts recorded in this section, and the exact manner in which the infection takes place, will be considered in Section IX after the origin of the disease has been described.

Infection in Europeans.

I have not seen any cases myself of the disease in Europeans, but have given in Section II, page 27, the information I have collected with regard to the instances which have occurred, of their suffering from the affection. In only two cases, however, was I able to get reliable information as to the habits, etc., of the sufferers. In one of these a forest officer had an orderly coming constantly into his bungalow, who had the disease at the time, and who died of it; and in the other a police officer employed as punka-coolies more than one boy, who had the disease at the time he was working in the bungalow, and who subsequently died of it. These boys used to sit on the floor of his office within a few feet of him, pulling the punka while he was doing his work. Both the

officer in question and also his young son contracted and died of the disease.

Infection on tea gardens.

The usual ways in which tea gardens became infected is of importance, as a knowledge of it will allow of proper precautions being taken to avoid it. I am indebted to Dr. Dodds Price, of Nowgong, for nearly all the information I have obtained on this part of the subject. He has been most kind in working out various figures and facts for me concerning it in the gardens under his care.

The disease nearly always appears first in coolies who have been years on the garden, and who should be acclimatised against malaria if such a thing is possible. The reason why they get it is because they have relations and connections in the surrounding villages, which recently imported coolies have not, and the old coolies therefore are more likely to visit any neighbouring infected villages. Now two of the gardens which suffered most in the Nowgong district were infected in this way from a small village which was within a mile or so of both of them, and which had the disease so badly that they nearly all died of it, and the survivors eventually deserted the site, which still remains unoccupied. On one of these gardens, moreover, two of the earliest cases were in the dâk-runners, who used to go into Nowgong station and stay there a night before returning, and that at the very time when the disease was bad in the town. The true nature of the disease was not at first recognised, but it was thought that the garden had become unhealthy for some unknown reason, as there were more fever cases than usual, and these were very resistent to treatment with ordinary doses of quinine, etc. Some of them were therefore removed to other sections of the garden, and between this and the general inter-communication between an out-garden and the headquarters, the disease broke out in the central, and also in the furthest out-garden, some 6 miles from that first affected. The death-rate in this furthest out-garden became very high, so some of the

affected coolies were moved out of it; and in order to carry on the work, 28 selected healthy coolies, who had been many years on the garden, and who were thought to be least likely to suffer from any form of malarial fever (for the disease was considered to be such from the beginning) were moved into the infected lines of this out-garden. No less than 14 of these 28 healthy selected coolies contracted the disease, and almost all of them died of it, and many of those who got it were related to each other. Vigorous measures are now being taken to eradicate the disease,—new lines have been built during the cold weather, into which all the healthy coolies have been moved, and the old ones will be burnt down, and not re-occupied for some years at least.

The beginning of the disease on another garden of this district is very instructive. The first case was diagnosed early in July by Dr. Price and myself. The patient was an old coolie, who had settled down on the garden, where he had worked for many years, and had connections in villages around. He had had constant fever for some two months, and already had a large spleen and liver, and was rather dark in the face. Within a month he had died of *kála-ázar*, and a brother of his had got the disease. The second brother died in another six weeks, and a third brother was then getting fever, and he died early in January 1897, so that all three brothers had died within six months of the disease, and there was not another case on the garden, nor is there one at the time of writing, namely, early in March. The houses occupied by these people have been destroyed by fire, and the other inhabitants of the group are being watched.

In another one garden two successive Assamese wives of a garden sirdar have died of *kála-ázar* within 18 months; but as they were *purdah* women, there was no opportunity for other coolies in the lines to come near them, and no other cases have occurred.

Once more, since the disease has been epidemic at the headquarters of the garden which has been mentioned as

having suffered most severely, as many as possible of the new coolies have been put in a new line just about 200 yards from the badly infected lines. In January 1895, a batch of 200 coolies arrived on this garden, and 150 of them were placed in the new lines, but 50 were put in the old infected lines for want of room. During the two following years, 2 of the 150 new line coolies died from dysentery, but none from *kála-ázar*; during the same period, 10 out of the 50 old line coolies died, 8 of the deaths being from *kála-ázar*. Thus 16 per cent. of the 50, who lived in the infected lines, died in two years of the epidemic fever, but none of the 150 coolies of the same batch who lived in the new line some 200 yards from the old ones. Comment seems unnecessary, as the figures speak for themselves.

The exact way in which the first cases occur on any garden is not always very easy to trace, especially if, as has usually been the case hitherto, the disease is at first thought to be ordinary malarial fever. The following case is an interesting one in this connection :—An out-garden at the end of a valley, and consequently with no traffic through it, suffered severely from the disease, and the main garden to which it belongs now also has the disease rather badly. A neighbouring garden, only one mile from the first-mentioned out-garden, but under a separate management, has remained quite free of the disease for several years, during which its near neighbour was suffering so severely. This peculiar distribution of the disease was not easy to explain until it transpired that the badly infected out-garden was almost entirely worked by old coolies, many of the relatives of whom had settled in a village, some 8 miles distant, near the out-garden, the lines of which have been already mentioned as having been moved on account of the severity of the disease in them. This village has also suffered from the disease, and through them doubtless their relatives in the out-of-the-way garden at the end of the valley became infected.

In another part of the Nowgong district, there had been

no *kála-ázar* in any of a series of three or four gardens until the last month or two. These gardens have recently been placed under a new manager, whose father also managed a garden, some 40 miles off, up to three months ago, when he left the district. A case of *kála-ázar* has recently been detected on one of the first-mentioned gardens in the person of an old servant of the last-mentioned distant garden, which had suffered very severely from the epidemic. This man had come to the garden on which he now lives, when his old master left the district. During the last few months there has been a good deal of communication between the two gardens, and the disease is now present in a village near the newly-infected garden, and in two of the garden coolies, but active measures are being taken, and the early detection of the disease should allow of a severe epidemic on this instance being avoided.

The above evidence regarding the spread of the disease on tea gardens confirms that derived from the villages of the affected districts, and the facts recorded in this section, taken with those relating to the general distribution of the disease given in Section VI, prove that the disease is a communicable one, but that it is usually necessary to visit or reside in a house or village which is infected in order to contract the disease.

The question remains to be considered—How then did a malarial fever get this power of communicability? In order to answer this, the origin of the epidemic must first be ascertained.

SECTION VIII.

THE ORIGIN OF KÁLA-ÁZAR AND ITS RESEMBLANCE TO THE "BARDWAN FEVER" EPIDEMIC.

I have not been able to find any attempt whatever in the numerous reports on the subject of *kála-ázar* to explain the origin of the epidemic. It seems to have been generally taken for granted that the disease is endemic in the Garo Hills, and has been so far a very long period, and that it spread from there in the early eighties to Goalpara, at the foot of the hills, and has been epidemic in the Assam Valley ever since. Why it should have suddenly spread in this way, has never been explained. Dr. Giles makes no attempt in his report to solve the mystery, but in a letter to the Chief Commissioner of Assam, dated Sanawar, the 27th June 1892, he writes—

"My personal opinion is that the disease has been introduced, within the last thirty years or so, and its appearance in the tea gardens as what is now called *beri-beri* and in the Garo Hills as *kála-ázar*, are alike phenomena, dependent on the improvement of communications and increase of intercourse which have resulted from the establishment of British rule, and the consequent advance in civilisation."

The question is, however, one of considerable practical importance, for if the epidemic arose from a disease which is always present in the Garo Hills, it may do so again at any time, and unless we know what causes it thus to spread, we shall not be in a better position to meet it than we were in the eighties. If, on the other hand, it is not endemic in these hills, but was introduced therefrom without, a correct knowledge of its true origin will be equally of importance Again, it has never been explained why the disease never broke out to the south of the Garo Hills, and spread to Mymensingh and Sylhet, as it might have been expected to have done at the same time that it spread up the Assam Valley, if it really arose in these hills.

In order to try and throw some light in these questions, I visited the Garo Hills, and examined all the administrative reports as far back as 1870, at which date they were commenced, with the result of obtaining much valuable information which, together with a subsequent and complimentary examination of the Bengal Sanitary Reports of the seventies, has resulted in a complete solution of the origin of the epidemic.

It was mentioned in the first section of this report that three different statements as to the probable date at which *kála-ázar* was first noticed in the Garo Hills, are to be found in the earliest description of the disease, showing that this point was not clearly made out at that time. A more definite idea can, however, be derived from the fact that the Garos themselves called the disease "Sirkari disease," or "Saheb's disease," not because the Europeans suffered from it, for up to that time they had entirely escaped, but because they said the disease was unknown among them until after the Sahebs took over their country. Now although Tura, the headquarters of the district, was occupied in 1867, the greater part of the country was not taken over until after the expedition of 1871-72, so that the epidemic was apparently not known until after this date. On the other hand, Colonel Maxwell, who was in charge of the district in 1881—83, writes to me—

"Elderly Garos have often told me their grandfathers died of the disease, which, however, was not so virulent as it has lately become."

Now it is obvious that, as the disease is only a very intense form of malaria, the Garo people for generations past, indeed, as long as the physical conformation of the country has been in its present state, must have died of chronic malarial disease indistinguishable, individually considered, from that subsequently called *kála-ázar*, but it does not follow that the disease has been present in an epidemic and communicable form all that time; in fact, Colonel Maxwell himself states that it was not formerly of so virulent a type.

That is the true explanation of the apparent discrepancies

is shown by a study of the oldest records, from which it appears that the statements that the disease has been in the Garo Hills "as far back as 1869," and that the Garos give accounts of it "at periods varying from 3 to 30 years," are based on reports of the disease in 1869 and 1873, in which from one to five cases of malarial cachexia are described as having been found in several villages spread over many miles of country, which are all noted as being at the foot of the hills in extremely malarious places, and the average duration of which cases is said to have been about two years. It is particularly noted that the disease is not met with in the higher land in the interior of the district. Now, in the accounts of the epidemic in the early eighties in the Garo Hills, it is recorded that the disease attacked the villages in the hills themselves, and, moreover, the cases in the villages were much more numerous and of much shorter duration than those above described, so that it is evident that the earlier descriptions apply to the ordinary malarial cachexia in the terai tracts of the country, and not to the epidemic malaria of later years, while it also follows that the term *kála-ázar* is the Garo term for ordinary malarial cachexia, which was very naturally also used to describe the epidemic disease, for the condition induced by the latter was identical with the former.

Fortunately, it has been possible to get clear evidence as to the exact date when it did become epidemic at the foot of the Garo Hills, for it began there, and not in the hills themselves. On looking through the administrative reports of the district, it appeared that the revenue was collected in the form of a house-tax, and that when for any reason the estimated amount was not realised, an explanation of the reason of the deficiency was recorded in the reports. Now up to the year 1875 the revenue was always collected in full, with an occasional slight remission, owing to the destruction of the crops in certain places owing to a flood or some such cause. In 1875, it is recorded that there was a deficiency of revenue from some villages in the south-west corner of the district

owing to several families having died of a disease called "*kála-házar*, which seems to show that the term was not a familiar one, as in the reports of subsequent years it is rightly called "*kála-ázar.*" From this date onwards, it is regularly referred to year by year as causing the deaths of a large proportion of the people of various villages, and thus occasioning a loss of revenue and depopulating certain portions of the country chiefly at the foot of the hills. With regard to the spread of the disease, Colonel Maxwell writes—

"The home of the disease, in my experience, is the Karaibari Mehal" (to the south-west of the district, and the part above alluded to as the first place mentioned in the administrative reports as suffering) "in the west of the hills. There the numerous villages marked on the map are entirely gone, and there only in 1881, when I went to the district, was the sickness raging; from this tract the disease travelled slowly towards Tura" (in the centre of the hills, observe that it spread up from the foot of the hills to the hills themselves, not in the reverse direction, as would have been the case if it had arisen in the hills) "and on to Damra in the plains at the foot of the hills to the south-east to Goalpara town *viá* the bridle-path to Rongrengiri and Damra. Thence it spread to the Assam plains, and it also worked its way back and attacked the Garo villages to the east of the district.

I am also indebted to Colonel Maxwell for the following most important piece of information:

"The first time *kála-ázar* was brought to notice was at a place called Bengal Kutta, at the foot of the hills south of Dhubri. I think this was after 1872, that is, after we occupied Tura, as the Civil Surgeon of the Garo Hills investigated the sickness. The people attacked all died, and the disease ceased in that locality."

This last sentence is a most significant one, as it shows that the people were quite ignorant of the virulence and fatality of the disease, which so suddenly attacked them, or they would have adopted the measures which they subsequently learnt to be the only means of escape from being annihilated, and would have deserted the affected sites of their villages, and dispersed and built new houses in separate places, as they did

later on when they became familiar with the epidemic which was exterminating them, to such an extent that the Deputy Commissioners of this district to this day complain that when they visit such and such a village, they have to go over from one to three square miles of ground in order to find all the houses.

Still more important is the fact that the place Bengal Kutta, which appears to have been attacked at just about the same time as Karaibari, is 50 miles distance from the last-named place, a distance which would have taken at best four or five years for the disease to spread over if it travelled at the same rate as it subsequently did in other districts; so that the disease must have broken out independently at these two places. Moreover, Bengal Kutta is just about opposite to the Jatrapurghát on the other side of the Brahmaputra river, which is one of the main lines of traffic between Rungpore and the foot of the Garo Hills, while the other main line is between Chilmari in Rungpore and the Karaibari or Mohendraganj district. The full significance of these facts will be seen later in this section.

It is evident then that up to 1875 there had been no loss of revenue due to the ravages of the epidemic malaria, first called *kála-ázar* in the Garo Hills, but that subsequently to that date it spread steadily through the district. Moreover, it began in two different places, 50 miles apart, and both in direct communication with the Rungpore district. This made me think that *kála-ázar* might possibly be a continuation of the epidemic of malarial fever of the sixties and seventies in Lower Bengal, which is generally called the "Bardwan fever." I found, moreover, that the first two medical officers who investigated the outbreak, namely, Mr. McNaught and Dr. Dobson, both state that the Garos always said that the disease came across from Rungpore, and not out of the hills themselves. It must be borne in mind that, except for the central range which runs up to a height of 4,000 feet, the Garo Hills are very low, and between them are patches of flat rice land, so

that the greater part of the district is, for all intents and purposes, a terai country, and consequently the very place in which an intense form of malaria would flourish when once it had gained a footing.

The question remains as to how far the disease has died out, and if it still remains in an endemic form, which may at any time break out again in the form of an epidemic. When I went to the Garo Hills, I was informed that the disease was still present, more or less, all over the district; but as this statement was at complete variance with what was stated in the administrative reports, as I shall show immediately, I asked where it could be seen, and was told of a village only 6 miles from Tura, which was said to be very bad with it. I accordingly walked out there one morning, but found the headman, who, I had been told, was dying of *kála-ázar*, was down with a slight attack of fever, which he had had for a few days only; and, although many of the apparently healthy children had enlarged spleens, showing the malarious nature of the place, I saw nothing at all characteristic of *kála-ázar* in this or in some other villages that I visited. On going over the administrative reports, I came across the following paragraph written by Mr. Heath, after he had been several years in the district. In the report for the year 1887, he writes—

"Between Parakhasma" (on the south side of the foot ofthe hills) "and Singimari" (towards the north-west and some 70 miles from the former), "it would be hard to find one single case of the real illness, *kála-ázar*. The disease travelled northwards to Salmara, and then turned eastwards along the northern boundary. Its ravages were somewhat severe; but in the stretch of country between Singimari and a point south of Goalpara, I can again say that hardly one case now exists; the disease has disappeared. It was some seven or eight years ago that it commenced its attacks on the villages of the Muzzarana Mahál. Some villages it has quite depopulated, others severely handled; but what marks the peculiarity of the course of the disease in this part of the country, is the persistence with which it clings to this mahál. Elsewhere along the border it has cleared away the weak and sickly, leaving the strong; but having done this, has cleared

itself away. In the Muzzarana Mahál it has found a congenial home This winter in my tours I found the number of sick something terrible to look at, or think of, and the number of deaths also has been appalling."

In the next year he records a similar experience, the portion of the district bordering on the Kamrup border alone being affected, and this part had suffered for four years only, so that it could not have been expected to have died out from it at that time. The completeness with which it had disappeared from the previously-affected parts is very evident, although the people were still living in the same places and villages; and, as has been shown in the previous section, this is also the tendency in all the districts which have been affected, in spite of the entire absence of any alterations in the physical or sanitary conditions. In the report for the year 1889, no mention is made of *kála-ázar*, and in the subsequent reports it is usually recorded that malarial fevers and *kála-ázar* were prevalent throughout the district as usual, and as at about this time both the administrative and medical officers of the districts were both changed, it is evident that they had very naturally confused the deaths from ordinary malarial fever with *kála-ázar*, although the epidemic form of the disease had quite died out. In fact, I am convinced, both from my own observations and from a study of the records, that there is no epidemic, or endemic, communicable, and very intense type of malarial fever, such as *kála-ázar* has been shown to be, in the Garo Hills, or in Goalpara and Kamrup at the present time.

Lastly, with regard to the escape of Mymensingh and Sylhet, it appears that the disease did actually spread along the south side of the foot of the Garo Hills, as far as a place called Parakhasma, but that between that place and the next inhabited village, namely, Dalu, there is "a stretch of 20 miles of uninhabited forest, and over this stretch it has not leaped": another excellent example of the way the spread of the epidemic is limited to lines of communications.

We have seen then that the disease invaded, decimated and died out of the Garo Hills just as it afterwards did in the southern portions of Goalpara and Kamrup, and that it is therefore not endemic there. The escape of Sylhet is also explained in accordance with the evidence as to the spread of the disease given in the last section. The true place and cause of the orgin of the disease still remains to be demonstrated, and for this purpose it will be necessary to go back a little further both as regards time and place.

When studying the literature of the subject in Calcutta before commencing work in Assam, I was struck by the general resemblance between the so-called "Bardwan fever" of the sixties and early seventies and the descriptions of *kála-ázar*, and some time afterwards it occurred to me that the latter epidemic might possibly be a continuation of the former. I therefore applied for the older Bengal sanitary reports, and found that just about the time the fever was dying out in Bardwan itself, there was a marked increase of fever in the districts to the north, extending right up to and including Dinajpore and Rungpore. Owing to the vital statistics being only started in 1870 and to the frequent changes in the manner in which they were collected in different years, rendering it difficult to compare the prevalence of fever year by year, and the further fact that no reports were received from the Civil Surgeon of one of the districts during several of the worst years in the northern part of the affected tract of country, it was not easy to trace the exact sequence of events. However, a minute study of all the material available, together with a tabulation of such mortality returns and meteorological data as were recorded, and a comparison of these with the more accurate figures of recent years, has shown that the fever epidemic in the north-western part of Bengal in the seventies was independent of the "Bardwan fever,' and was started by a succession of several years of very deficient rainfall, such as is to this day commonly followed by an unusual amount of fever in these parts. As the

"Bardwan fever" epidemic is certainly an exactly parallel occurrence to the *kála-ázar* outbreak, a very brief account of it will be first given, then the Dinajpore and Rungpore outbreak of fever will be described, and its connection with the Assam epidemic fever will be demonstrated, and lastly, the resemblances of this to the "Bardwan fever" will be illustrated.

The following extract from the Bengal Census Report of 1881 is the most concise account of the "Bardwan fever" that I know of:

"This fever, as has been said above, invaded the Bardwan division from the east. It appears to have originated in the eastern part of the Presidency division some 30 years ago. Fever was very fatal in the Jessore district in the years 1847-48, and after a temporary cessation it broke out again in 1854-56. About this time it began to spread westwards to Nuddea and the 24-Parganas, and finally culminated in the severe epidemic which devastated these districts from 1857 to 1864. No notice of this fever seems to have been taken by the authorities till the end of 1861, although it prevailed in a most virulent form in Nuddea from the end of 1856. Towards the end of 1861, however, its ravages in the Baraset division and the northern portion of the 24-Parganas attracted the attention of Government, and efforts were made to combat it. A few months later, measures of a similar kind were introduced into the Nuddea district, and in all the three districts then included in the Presidency division, relief operations were continued until the fever died out about 1864. Briefly stated, this fever was most severe in the rainy season and the winter months of the years 1860 to 1862-63. In the winter of 1863-64 there was a perceptible improvement and a radical change for the better in that of 1864-65. But while the epidemic was wearing itself out in the districts of the Presidency division, where it was first observed, it had spread slowly westward into those of the Bardwan division. The Bardwan district is separated from that of Nuddea by the Bhagiratil river, and the thanas of Burdwan which abut on the river, are three, *viz.*, Cutwa the north, Purbasthali in the centre, and Culna to the south. It was in the two riverside villages of the Culna thana that the first cases of the epidemic occurred in the year 1862. In 1863 the fever re-appeared in the Purbasthali and Culna thanas, and, attacking first the villages on the river bank, advanced slowly inland, spreading also southward

into the northern portion of Hooghly. In 1864 and 1865 the fever moved still further west both in Hooghly and Bardwan, and extended in a southerly direction to the boundaries of Howrah. The features of the epidemic in 1866-67 were the same as in 1865, except that there was no further spread to the south in Hooghly and Howrah. In 1868-69 a great advance occurred, the town of Bardwan being involved in the former year and the epidemic spreading far to the north and south, besides continuing its usual westerly progress In 1870 its western extension was not very great ; but on the north it invaded Beerbhoom, and raged along an extensive tract on the southern portion of the district, the left bank of the Adjai river. In the following year, 1871, the extent of its advance was unprecedented ; for not only did it progress many miles to the west in Bardwan, but it spread to the northand north-west in Beerbhoom, and appeared in a large tract of country in the north of Midnapore. By 1872 the utmost westerly limit that the fever reached in Bardwan was attained, for it was arrested by the high land lying in the extreme west of the district and along the Bankura border ; but the Beerbhoom district was devastated still further north than in the previous year, and in the Midnapore district a great southern extension took place, enveloping almost the whole of the north-eastern portion of the district. In 1873 the fever made no further westerly progress in Bardwan ; and, though still severe in the west, it was gradually dying out in those parts of the district where it had been first observed. In Hooghly and Beerbhoom also there was some slight abatement, but in Midnapore and Howrah the mortality was twice as great as in the preceding year. The year 1874 may be taken as the last of the epidemic in this division (the Bardwan) ; from all quarters came reports that the fever was less fatal and less prevalent than in the previous years. In 1875 the same facts were observed again, and what fever there was wanted the virulence of the epidemic, and had all the characteristics of the ordinary seasonal malarious fever of the country."

The deaths in the Bardwan district also are estimated at " not less than three-quarters of a million of persons, " and the Bardwan division was the only one showed a decrease of population between the census of 1872 and 1881.

The further history of the epidemic as recorded in the reports of Sanitary Commissioners of Bengal, was as follows :

In 1874 the fever was noted to be less prevalent in both Midnapore and Beerbhoom, it never having obtained a sure footing on the dry, porous laterite soil in the former district, and in the latter it seems to have turned towards the west, and to have been checked by the high rocky land of the Sonthal Parganahs border. Fever was, however, unusually prevalent in the district of Moorshedabad to the north of Beerbhoom in both 1873-74, and also in Malda further still to the north ; but it is recorded that this was not an extension of the Bardwan epidemic, although the Sanitary Commissioner, Dr. Jackson, who had closely investigated its prevalence in Bardwan during two years, and written a very full report on it, to which I shall have to refer presently, seems to have been somewhat doubtful if it was so or not. It was, however, coincident with a great increase of fever in the still more northern districts of Dinajpore and Rungpore, and was therefore most likely due to the seasonal causes that the fever of these last mentioned places will presently be shown to have been due to. The further fact that the fever would have had to spread many times more rapidly than it had ever done in Bardwan in order to have covered so large an area in one or two years, and that the type of the fever in these northern districts was different from that of the Bardwan epidemic, also support the view that the latter was independent of the former epidemic.

Having thus cleared the ground, we are now in a position to study the great outbreak of fever in the districts of Dinajpore and Rungpore in the years 1872—79, which also involved the continuous northern portions of Rajshahye and Bogra. It is singularly unfortunate that for some of the years when the fever mortality was highest in the Dinajpore district, no report was received from the Civil Surgeon in charge, and that the monthly rainfall in the affected districts has not been regularly recorded in the Sanitary Reports, as the absence of full data prevents me illustrating the origin of the epidemic by a chart made out from the monthly fever-rates and rainfall year by year. I will first give one or two quotations to

show to what a great extent fevers were prevalent in this part of Bengal at the time mentioned, for it does not seem to have attracted as much attention at the time as might have been expected. In the Bengal Census Report for 1881, it is recorded as follows:

"Dinajpore has made no progress at all during the last nine years, for the advance of the population has only been 0·28 per cent., which would be much more than accounted for by better enumeration. This pitiable result is due only to the ravages of malarious fever, for which this district has as evil a reputation as its neighbour, Rungpore. In 1872, the reported deaths from fever in Dinajpore were higher than in any other district in the Rajshahye division. The four following years showed little improvement, and in 1876, the fever mortality was 22·05 per mille of the whole population. In 1877, which a general consensus of opinion declares to have been the most unhealthy year in this district within living memory, this rate rose to 30·06, and 36,000 deaths were reported from this cause alone. Out of 17 adult Europeans 15 had to leave the district during the year broken by repeated attacks of fever, and official business could hardly be carried on. ..... It so happened that this district was at the time that in which death registration was best carried out in all Bengal; and it was found that the death-rate in the municipality was 42 per mille, nearly double the death-rate of London, while the police died at the rate of 46, and the prisoners in the jail at the rate of 74·6 per mille per annum."

Again, of Rungpore, it is recorded that—

"Rungpore fever has passed into a proverb in Bengal, and though it is now thanks to improved drainage, less virulent than formerly in the town of Rungpore itself, its effects on the population of district are still widespread and unmistakable. It is a slow, lingering fever, usually attended with spleen and liver complications, and leading in the winter months to dropsy, phthisis, and other pulmonary affections."

As early as 1874, the Civil Surgeon reported, after a special inquiry, that—

"Eighty per cent. of the people were anæmic or suffered from enlarged spleen, or were laid up with illness, while the 20 per cent. found healthy could not be considered so in the European sense of the word."

From the fact that rich were not of more exempt than the poor from sickness, he rightly inferred that—

"the unhealthiness of the district was not due to poverty or privation, but to the malaria in which the whole country is steeped."

And he particularly mentions—

"the damp banks and islands of the Brahmaputra to the east and north-east of the district"

as having suffered. There was a decrease of 2·58 per cent. of the population of the Rungpore district between the censuses of 1872 and 1881, which was attributed to the fever epidemic.

In 1871, there was no excess of fever in Rungpore, and the rainfall was normal amounting to 94·15 inches. In 1872, there was much fever in Dinajpore, more especially in the south and south-west, and intermittent fever was very prevalent in Rungpore, which caused much mortality, and was of a chronic type. Malarious remittent fever was also very prevalent. This was the first of the series of years in which the rainfall was unusually low, although the deficiency was not very great in this year. There were twice as many deaths returned from fevers in Dinajpore, and three times as many in Rungpore in 1872 as in 1871, although the deaths per mille in the whole of Bengal were 4·62 in the former year as against 3·32 in the latter, part of the increase having been due to better registration of deaths.

In 1873, no report was received from Dinajpore, except that—

"fevers of a malarious type prevailed during the year."

The rainfall was only 43·53 against an average of the five years previously to 1873; of 84·59, a deficiency of nearly one-half. Of Rungpore, it is recorded that—

"fever of the intermittent type was present in the district throughout the year. From May to December it continued increasing in intensity, so that the majority of the population were completely prostrated by repeated attacks of the disease. The poorly-feed and those who did not take advantage of treatment, suffered most, and were affected with spleen and liver disease, and subsequently with a fatal type of dysentery and dropsy."

It is further stated that—

"there is no material change in the country to which the prevalence of this fever could be traced; and the most probable cause to which it could be ascribed is climatic influences, *viz.*, scantiness of rain and excessive heat, which intensified and concentrated the marsh poison."

That this is the true explanation is proved by the fact that a study of the monthly rainfall and fever-rates in both Dinajpore and Rungpore from 1881 to 1894 shows a most definite relationship between a low rainfall, especially if it is also irregularly distributed so as to cause alternate flooding and drying up of the land, and an increase of the fever-rate. Moreover, in the years 1873, 1875, and 1877, in addition to the rainfall being much below the average in Dinajpore and Rungpore, in every case it was very irregularly distributed. For instance, in the year 1873, the monthly rainfall in Rungpore was as follows :—In the first three months only 1·2 inches fell, in April there were 8·24 inches, in May 1·07, in June 13·09, in July 3·93, in August 14·11, in September 2·63, and none in October or November, and but 0·38 in December, the total being 44·64 inches, or barely half the normal average, which was 88·19 for the five years previous to 1873; so that there must have been a succession of floods followed by the exposure and drying up of the saturated soil under a May, July, and September sun, than which no more favourable circumstances can be imagined for the multiplication and escape into the air of the malarial organisms by their being carried up in the evaporating moisture. When we see a series of such years one after the other, is it any cause for wonder that fever should become more and more prevalent and intense until an epidemic of malarial fever is produced? Is it surprising that in this year it is recorded of the fever in Rungpore that—

"From May to December it continued increasing in intensity, so that the majority of the population were completely prostrated by repeated attacks of the disease?"

In this connection the Civil Surgeon's description of the

physical condition of the country in the district of Rungpore, will not be inappropriate. He writes—

"The *bheels*, the ever-reeking repositories of putrefying organic matter, had not their contents diluted or overflown into rivers, as is the case with other years; but the thick slime from the heat got thicker and thicker, exhaling the offensive gases in a concentrated form" (which put into the more precise, but less picturesque, language of modern science, might be paraphrased: exhaling the malarial organisms in an intensified form). "The soil, too, by scanty rain and alternation of rain and sun, had its deposite of organic matter set into putrefaction, which, if the rains were plentiful, would have been washed off into large bodies of water before doing harm."

To continue, in 1874, the rainfall was again very difficient in the early part of the season, but a heavy fall took place in September and October, which brought the total nearer to the average than in the preceding or following years, and the mortality from fevers was slightly less in the district of Dinajpore. In Rungpore, however, the deaths from fever were much greater than in 1873, the mortality in the selected areas, which furnish the most correct figures, being 23·61 in 1874, as against 11·45 in 1873, which, allowing for improvement in registration, still leaves a large margin of actual increase. The fact that the seasonal incidence of the disease extended over a much longer period than usual, shows that it had attained to epidemic intensity (this being, as I have shown in Section VI, characteristic of the epidemic malaria of Assam), while the fact that there was a marked decrease in the fever after the heavy rain in September, illustrates still the further dependence of the increased fever-rate on the previous low rainfall. The following quotation will suffice to prove these two occurrences:

"The fever, which had commenced in May 1873, earlier than in former years, and increased in intensity to December, reducing the people to the lowest state of vitality from repeated attacks, and inducing a state of spleen and liver affections, and finally, desentery and general dropsy during the sharp cold of December and January 1874, continued to prevail up to the rainy season of the year under

report (1874). But curiously, after the heavy rains in September, and especially after the cyclone of the 16th October, the disease abated."

In 1875, there was a further increase of the fever-rate in both Dinajpore and Rungpore, the mortality in the former district having increased in the selected areas from 20·36 to 24·75, and in the latter from 23·61 to 24·56. From Dinajpore no report was received from the Civil Surgeon for the third year in succession, so details as to the type of the prevalent fever cannot be given here. In Rungpore, however, it is recorded that—

"Remittent fevers are common and fatal in this district, but the principal disease is intermittent fever, which is most prevalent during and after the rains. Chronic enlargement of the spleen and liver follows repeated attacks of the disease, and in the cold-weather months diseases of the lungs and general dropsy supervene."

This year once more the rainfall was very much below the average, being only 43·6 in Dinajpore, and in Rungpore was 60·45. It was also very unevenly distributed. It is worthy of note that the six areas, which are especially mentioned as having suffered most severely from fever during this very unhealthy year, are all situated in the east of the Rungpore district, that is, in the portion which borders on the Garo Hills terai, from which it is only separated by the Brahmaputra river, which with its numerous islands is included in Rungpore, and, as already noted, these river banks and islands shared in the outbreak. Now this year was the very one in which it was ascertained by an independent study of the Assam district reports, before I had any knowledge of the details of the Rungpore outbreak, to have been that in which the fatal depopulating epidemic malarial fever first made its appearance in the south-west corner of the Garo Hills district; and, although the fever in this terai country at the foot of the hills might have been expected to be intensified by the same seasonal influences, which have been just shown to have affected that in the Rungpore and Dinajpore districts, yet as a matter of fact the rainfall in the Garo Hills was not deficient at that

time, for the fall there for the years 1873—79 averaged 120 inches, which happens to be exactly the same figure that the average for the years 1880—87 works out to be. Moreover, the fever in the Garo Hills appears to have started in two separate places, both in the direct lines of communication from Rungpore, and not uniformly over the district, as it should have done had it originated there owing to seasonal influences, and in this respect it follows the same course that the epidemic of Lower Bengal did when it invaded Bardwan. It will also have been observed that the type of fever in Rungpore was exactly that of the Assam epidemic, while the mention of remittent fevers as having been common and fatal in the Rungpore district in 1875, is worthy of note, as indicating a still further increase of the intensity of the fever in the district during that year.

The subsequent history of the outbreak of fevers in Dinajpore and Rungpore need only briefly be given. In 1876 the rainfall was again deficient, being only 66·55 in Dinajpore and 65·69 in Rungpore. The fever in both districts showed a still further increase, the mortality in the selected areas being 32·90 and 28·15, respectively, as against 24·75 and 24·56 in 1875. In 1877, the rainfall was once more low in Dinajpore, being only 67·27 inches, but in Rungpore it was 91·03, but was extremely unevenly distributed. There was a further increase of the fever mortality in both districts, more especially in Rungpore, in spite of the heavy rainfall, which shows what an epidemic hold the fever had got on this district. In 1878 there was an unusually heavy rainfall in Rungpore, namely, 121·5 inches, and the fever both here and in Dinajpore showed a slight decline. In this year it is recorded that—

"at Rungpore the disease prevailed virulently in some places" (just as it was at this period doing in some places in the Garo Hills district under the name of *kála-ázar*), "while others enjoyed a tolerable amount of immunity; but, on the whole, a lesser area was covered by it, and the mortality had slightly declined."

In 1879, the rainfall was again above the average, and there was a marked decline in Dinajpore of the fever-rate, and a distinct improvement also in Rungpore.

We see then that in this remarkable series of years, from 1872 to 1877, there was a very deficient rainfall in five out of the six in Dinajpore, and in four out of the first five of them in Rungpore, and that the influence of this deficiency in increasing the prevalence of malarial fevers, which are always severe in these districts, was still further strengthened by the very irregular distribution of what did fall in alternate periods of heavy rain and prolonged intermissions, while that of the year in which the total fall was not much below the normal, was very deficient in the first three months of the rainy season. An examination of the monthly rainfall of these and other districts of Bengal for the years 1879 to 1894, inclusive, shows nothing approaching to such a series has occurred during this prolonged period, nor, let us hope, is likely to occur again for many years to come. That it was accompanied by, and was indeed the cause of, the steadily increasing prevalence and intensity of the fever of those years, there can be no doubt, while the absence of any such cause for the intensification of fever at the foot of the Garo Hills at the time that *kála-ázar* began in that place, together with its simultaneous outbreak at two widely separate places, both in the direct lines of communication with Rungpore, and its exact similarity with the type that was prevalent in Rungpore with epidemic intensity at the time of its commencement, makes it certain that the epidemic malarial fever of Assam arose from an intensification of the ordinary malarial fever of Rungpore by an extraordinary succession of abnormally unhealthy seasons such as can fortunately very seldom recur.

THE RESEMBLANCES BETWEEN THE ASSAM EPIDEMIC FEVER AND THE "BARDWAN FEVER".

The description already given of the spread of the epidemic malarial fever in Lower Bengal, usually known by the name of the "Bardwan fever," is sufficient to show that in its general course it is exactly similar to the epidemic fever of Assam, known by the name of *kála-ázar*. That it was also

similar in all its essential details, will now be briefly proved by extracts from the Bengal Sanitary Reports of the seventies, in which will be found the views of all the Civil Surgeons and others who studied the disease in the affected districts, especial attention being paid to the important points of its nature and mode of spread.

Firstly, as to its nature, there was a practically unanimous consensus of opinion that it was from beginning to end a malarious fever, although,—as was also the case in Assam with regard to *kála-ázar*,—suggestions have been made that there was something in it in addition to malaria, pure and simple, the chief of which is that it was a typho-malarial fever, the typhus fever factor accounting for the infectious properties of the disease.

Dr. Elliot, who was Civil Surgeon of Bardwan up to 1871, and had a great experience of the disease, considered it to be—

"an exaggerated and congestive form of malarious fever, most frequently of the intermittent, but also of the remittent, type."

Dr. French, who succeeded Dr. Elliot in Bardwan, writes in 1874:

"Ever since I came into the district, I maintained the opinion that the 'Bardwan fever' was only an exaggerated or malignant form of the ordinary endemic fever of Bengal, and similar to that observed in marshy places and other unhealthy tracts. I am further of the opinion that the events of 1874 have set the matter beyond all doubt."

The events referred to were the nearly complete absence of the bad type of cases, their place being taken by a milder type and fewer remittent cases, and its seasonal distribution had become reduced to its normal limit. Dr. Wilkie, who was specially sent to investigate the fever in 1874, agreed with Dr. French in considering it to be entirely malarial in its nature. Dr. F. Barker, who saw much of the disease in Beerbhoom, says:

"This fever is in every respect identical with malarious fevers which prevail elsewhere. It is, however, of a more tenacious and fatal character than I have seen in other parts of India."

Dr. Joubert, who was Civil Surgeon of Bardwan in 1875, writes—

"I am of the opinion that it was a purely malarious fever locally intensified into a very virulent type. This type is now rarely met with, either because the exciting cause has died out, or because the disease has exhausted itself on all such as were liable to be affected by it. All those who have had opportunities of studying the disease thoroughly, hold the same opinion that it was purely a malarial fever."

The disease also followed the seasonal mortality curve of the ordinary malarial fever in the same district. Thus writing on this point, Dr. Wilkie says—

"Now the year 1872 was a genuine *jor-bekar* year; keeping that fact in view, the similarity of the mortality curves in the three years in all respects, except that of height, seems to me a very significant fact with respect to the question of the nature of the disease."

The way in which in any place it began insidiously and became worst after a year or two, was also a characteristic feature of the epidemic in Bardwan. Thus, Dr. Jackson, the Sanitary Commissioner for Bengal, who spent part of two years in the affected districts studying the disease, and whose voluminous and invaluable report will be frequently quoted from, writes on this point—

"It is to be remembered that in all these areas successively attacked the year of invasion is never the year of intensity. The fever is generally present for a year without attracting much notice. As a rule, the year of greatest intensity in villages is the third year, in towns the second year, is not unfrequently the worst."

The tendency of the fever to relapse again and again until all the signs of extreme malarial cachexia supervened, was also frequently mentioned in the descriptions of the disease. Thus it is recorded:—

"In some severe cases fever was prolonged for ten days or a fortnight, and the prostration of strength was excessive. The patients were reduced to skeletons, recovering gradually in two or three months, subject, however, to relapses, which, as a rule, invariably followed. The fever, however, in these cases of relapses

does not last long, yet the patients become weaker and weaker, and ultimately enlargement of the spleen, liver, anæmia, and dropsy set in."

The type, however, of the Bardwan cases differs in some important points from that of the Assam epidemic, as would naturally be expected, seeing that the two epidemics had their origins in such widely separated places as Jessore and Rungpore. Thus, the Bardwan fever cases seem to have been of a more açute nature, and to have been frequently rapidly fatal with cerebral symptoms. Again, a larger number of persons in one house or village seems to have suffered from the fever at the same time in the Bardwan epidemic. The severe remittent fever, which was always preceded by an increase of ordinary fever, is described by Dr. Jackson as follows:—

"From the first the patient had a stupid drowsy, brain-poisoned aspect; was unwilling to talk or answer questions; the intellect gradually became more and more confused; and he lost the power of understanding what was said to him, and it was only by shouting that he could be roused at all. From the first also there was intense prostration and loss of muscular power. In the cases which proved fatal, the stupor passed rapidly into coma, and after 12 to 36 hours of utter insensibility, death occurred. Some of these cases proved fatal in three days; others lasted a week or ten days."

It was these cases that made Dr. Jackson believe the disease to be "typho-malarial fever," but it is worthy of note that they formed only a small proportion of the cases, and Dr. French, in writing about them, says:

"This condition, if not immediately relieved by large doses of quinine, passes into a state of coma with dilated pupils, etc., and then death."

Which implies that if large doses of quinine are given, they may recover. The Bardwan fever was then a very intense form of malarial fever, just as so-called *kála-ázar* is, but the type of fever differs in the two cases, just as malarial fevers differ from one another all over the world. Let us see now how it spread.

As the appointment of Sanitary Commissioner of Bengal was only made in 1868, when the first sanitary report appeared, very few details are on record with regard to the earlier course of the Lower Bengal epidemic fever in Jessore and Nuddea, but ample material is available as to its subsequent spread in Bardwan, Beerbhoom, Midnapur, and Hooghly. Although it was patent to all that the fever invaded Bardwan from the east, or Nuddea side, yet so firm was the conviction that the fever was a malarious one, and *ipso facto* could not possibly be communicable in any way whatever; that numerous theories were started one after the other to account for the spread of the fever on local grounds, as each, in turn, was rendered untenable by the total disregard paid to them by the fever in its 12 years' march through Bardwan. Thus, Dr. Jackson writes—

"Malarious fevers are not infectious or contagious. They are produced, in the neighbourhood where they prevail by the action of the sun on a moist soil. ..... Malarious fevers cannot travel; they may be blown to a certain distance, or may diffuse to a certain distance, which is very limited."

Again Dr. Wilkie says—

"In the writings of those who have maintained the malarious nature of the disease, there is a very general absence of all reference to the subject of contagion, probably because the malarious nature of the fever was accepted as a guarantee of its non-contagiousness."

Thus we see, just as has been the case in Assam with regard to *kála-ázar*, those who looked on the fever as malarial held that it could therefore not be communicable, while Dr. Jackson, who seems to have been the first to recognise that its steady spread could only be accounted for on the supposition that it was communicable, argued that for this very reason it could not be entirely malarial in its nature. Later, when the disease travelled across the Bardwan district into Beerbhoom and Midnapur, it was recognised that, although malarial, it was communicable, but no satisfactory solution of the difficulty was offered; and I believe it is generally recognised to this day that it has never been fully explained.

I hope, however, to be able to show in the next section how this problem, both in the case of the "Bardwan fever" and "*kála-ázar*" which was insoluble in the, from the scientific point of view, dark ages of a quarter of a century ago, can now be easily explained by the light of mordern bacteriology, although it will not be capable of complete demonstration until the malarial organism can be cultivated outside the body in laboratories. The evidence as to the communicability of the Bardwan fever must be first given. It is unnecessary here to discuss the many theories which were put forward to account for this epidemic by purely local alterations of the country affected, either by a hypothetical wave of "gradual elevation of the fever tracts from east to west," or an equally unproved "river elevation above the intervening land," while the various river and railway embankments to which were attributed the outbreak, not only were totally inadequate to affect the large areas involved in which they were but as mole-hills, but they were crossed in all directions by the advancing wave of fever without any relationship whatever to their influence on the drainage of the tracts. It may safely be said that such obstructions as did exist were far too local to account for a tithe of the area affected, while the one theory which approximately covers the area involved, namely, that which attributes it to the successive elevation of the rivers from Jessore in the east to those of Midnapur and Beerbhoom in the west, is not only totally without any proof that such a thing took place during the time of the epidemic, but is entirely negatived by the sequence in which the various areas were affected, bearing no accurate relationship to the supposed cause.

On the other hand, there is abundant evidence that the fever travelled steadily along "lines of communication," subject, however, to the controlling influence of another factor, namely, a soil on which many malarial fevers naturally flourished. This last all-important factor has been already illustrated, and it has been shown that to this alone was due the cessation of

the spread of the epidemic when it reached the laterite and rocky soil of Midnapur and Western Beerbhoom, respectively. The former remains to be illustrated by abstracts from the accounts of the epidemic.

In 1872, Dr. French, after recording it as his opinion that the disease was an intense form of a malarial fever, which local conditions were not sufficient to account for, writes—

"There is one thing certain about the fever,—it progresses steadily, although slowly; in some years it has come east and south-east, regularly west and north-west; it has followed like a rolling wave, the chief roads and lines of communication; and it is steadily going on to the west and north-west."

And he compares it with the epidemic fever in the Mauritius of 1866-67.

In 1875, the Civil Surgeon of Serampore (in the Hooghly district) writes—

"There is no doubt that the fever seems to follow the tracks of the main and branch roads of the district originating at Jehanabad, on the other side of the Damuda; the disease can be distinctly traced along the old Benares road, which ends at Sulkea above Howrah. There is no doubt that the fever has been carried along this road to Howrah."

And he illustrates these remarks by a map. Dr. Barker, of Beerbhoom, considers that—

"the fever could not have been generated where it prevailed, but must have been introduced from Bardwan either through intercourse or the atmosphere."

And again—

"These insanitary conditions came about very gradually; and as until recently the health of the people has been good, they cannot be said to be the existing causes of the epidemic, although they may be predisposing causes. It is hard to disassociate oneself from the early teachings of science, but facts are stubborn things, and plainly point to the fever being communicable."

In Midnapore the Civil Surgeon, Dr. Mathews, did not agree with Dr. Jackson that the fever is propagated by human intercourse.

"He instances the town of Midnapore, where hundreds of people

had fled from the surrounding villages that were suffering from the epidemic, yet this crowded, and by no means clean, village escaped the fever."

But the Magistrate, Mr. Harrison, did not concur with Dr. Mathews, for he writes—

"I do not see anything in this district to confute Dr. Jackson's views, while there is much to confirm them. It is plain in the face of it, that the progress of the fever is directed by other agencies besides contagion."

And he points out that Midnapore is situated on the laterite soil, and that no places, except quite on the outskirts of this tract, had been attacked, the whole of which had been for 15 months in immediate contact with the fever-infected parts, so that "it may be inferred that this tract is at least adverse to its spread, if not altogether fatal to it," as it subsequently proved to be. But he writes:

"Assuming that other agencies which appear to be of some natural character beyond human control, determine its general progress, there are many facts to show that contagion accelerates its march. In every instance that I call to mind, the outposts of the disease, if I may so say, of the fever have been on roads, and constantly traversed by traffic."

This seems to be an instance of a non-medical man, untrammelled by "the early teachings of science," seeing more clearly than the doctor. But it is in the elaborate report of Dr. Jackson that the most convincing evidence of the communicability of the disease is given, and he goes further than the others, and shows how the infection is carried. Thus, he writes, in the account of his tour in the affected districts in 1873—

"Neema I found to be a very insignificant little village of only nine houses, containing at the time of my visit 65 people; but during the preceding year, 1872, there had been 21 deaths. The people's account of the visitation was this: In May 1872, some men belonging to the village, who had been employed in the country south of the More, where fever was prevalent, returned sick with fever to the village. The other inmates of their houses then began to suffer, and in a little time all the inhabitants were attacked. Some of them had friends in

Kotasore (2 miles west), and asked them to come and nurse them; but those who came were also attacked, and returned with the fever on them to Kotasore, hitherto free from the disease, and it began to spread there in the same manner. This was a volunteered statement, and the people evidently believed that the disease had been imported into Neema, and thence carried to Kotasore. In Kotasore, which contains about 60 houses and 300 inhabitants, the people assured me that the disease first appeared in the houses of some persons who, having gone to Neema to see sick friends, had returned with fever, and introduced the disease among them. There had been thirty deaths, and both here and at Neema many people were ill at the time of my visit."

He gives several other similar instances, all of which were volunteered statements. Another very instructive case is the following :

"In Kotulpore, a thana recently transferred from Bankoora to the Jehanabad (and fever) subdivision of Bardwan, a very rapid spread of fever occurred in 1873, which was attributed by the people to importation from the infected subdivisional station to which they were, in consequence of their recent transfer from Bankoora to Bardwan, really compelled to repair for the transaction of official and legal business, and I am assured that this belief was so strong among them, that numbers preferred losing their suits to incurring the risk of visiting Jehanabad."

Again, he writes—

"It must be borne in mind that there is a kind of intercourse between infected and uninfected villages, which is the necessary consequence of the prevalence of the fever. Sickness and death during the outbreak put so many of the labouring classes *hors de combat*, that much of the annual crop would be lost, unless extraneous assistance were procured. For this purpose, the neighbouring uninfected villages are resorted to; a few labourers are obtained from each of the places around, and it is a common history that the people so employed return to their own houses with the fever, and introduce it among their own people."

Again, Dr. Jackson also records much evidence to prove that the houses become infected. Thus, he writes—

"In Sheopore, in twelve consecutive, but separate, houses, only 3 out of 50 died; in the next group of 12 closely-built houses, of which two were empty, 33 out of 55 died."

Once more—

"I have noticed that when deaths from this cause (the epidemic fever) occur during the year of invasion, there are deaths in the same houses in the following year in more than half the number of cases noted."

To turn from the specific to the general, he sums up as follows:

"During the eight years succeeding the introduction of the fever into Culna, the disease spread steadily westward so long as the roads and traffic lines were westerly. When these began to run north and south, the disease took the same course, and its whole history exhibits a remarkable and persistent association with the lines of communication. There has never been any such connection between the direction of its propagation and that of the lines of drainage. Two areas have escaped the disease, of which the distinguishing features are as follows:—One is low, moist, fertile, contains the average district population per square mile, is purely agricultural, belongs to the district of Moorshedabad, and has no road connecting it with the fever tracts of Bardwan or Beerbhoom, and no traffic with those districts. The other is somewhat higher and drier than the average, is unfertile and sparsely populated (492 per square mile) as compared with the average of the district, which is 578 per square mile, and has no road crossing it, save a mere track. North and south of the area, where there are roads and traffic, there has been fever also. West of the former area, along the road line, there is fever also. In the areas themselves, which resemble each other closely in no particular save their isolation from infected portions of Bardwan, there has been no fever whatever. The exemption of these areas under the conditions mentioned, is corroborative of the conclusion that the disease spreads by importation and communication from and between attacked and healthy villages. The fever then is a travelling fever. It appears, spreads, prevails for a certain time, and disappears."

He sums up his opinion as to the nature of the fever thus:

"I believe that a fever originally malarious, acquired either Jessore or Nuddea contagious properties; that in virtue of this contagion, it travelled westward into Western Nuddea."

And he traces it from here to Bardwan, and continues—

"From his time its history is that of a travelling contagious

fever, intensified by crowding, receiving an accession of strength and malignancy in every large town it visited, and proving most fatal wherever people and villages were most concentrated. Wherever there has been active intercourse, the fever has travelled; where there has been little or none, it has died out."

And again—

"It will be seen from the foregoing remark that, while I believe the fever to be malarious in its origin, and to have some malarious characteristics, I blieve it to be not a mere or simple, but a contagious, malarious fever, that is, probably typho-malarial; and that it has not been produced in Bardwan, but imported and again carried from Bardwan into Beerbhoom, and its behaviour under the various conditions observed are quite incompatible with its being simply malarious, locally produced, or non-contagious.

I have quoted thus largely from the reports on the Bardwan fever, because I am so convinced that the epidemic of malarial fever which devastated several districts of Lower Bengal in the sixties and early seventies, is a phenomena so precisely similar to the epidemic fever which has traversed Assam during the last twenty years, that, although I have shown it had a different origin and was also of a different type, yet I am prepared to go so far as to say that any explanation of *kála-ázar*, which will not also be applicable to the "Bardwan fever," will not be satisfactory. Moreover, the facts which have been recorded concerning the Lower Bengal epidemic prove conclusively that the Assam outbreak is not the first time that malarial fever has attained to the power of communicability, and in the former instance, too, the epidemic was started as far as the imperfect records of that time allow of an opinion to be formed on the subject, by extraordinary conditions brought about by an unusually rapid silting-up of the Bhairub river.

SECTION IX.

CONCLUSION AS TO THE NATURE OF THE EPIDEMIC AND THE METHOD OF INFECTION.

We have seen that the epidemic which goes by the name of *kalá-ázar* in Assam is from first to last a very intense form of malarial fever. We have seen that the whole course and distribution of the disease has been that of a wave of increased mortality from fever, which has slowly, but surely, spread up the Assam Valley in the lines of communication, and that conclusive evidence has been given to prove that the fever is spread by being actually communicated directly or indirectly from one person to another: in a word, that it is infectious. We have seen that there was a definite cause for the intensification of the ordinary malarial fever in Rungpore in the early seventies, and that, as a matter of fact, the Assam epidemic did take its origin from the spread of this intensified Rungpore fever across the Brahmaputra by the two main lines of communication between that district and the foot of the Garo Hills, where it first attracted attention under the name of *kála-ázar*, or black sickness. We have also seen that the epidemic of malarial fever which devastated parts of Lower Bengal in the sixties and early seventies, was of a precisely parallel nature to the Assam epidemic, although it was of a different type and had a different origin, and doubtless other historical epidemics of malarial fever for centuries back were of the same nature, although this is the first time that such a phenomena has been examined in the light of modern pathological and bacteriological knowledge, for the latter was still in its infancy at the time of even the Bardwan epidemic, and the malaria germ was then undiscovered. The question remains whether the facts adduced in this report, when viewed in the light of our present knowledge, will allow of an intelligible and comprehensive solution of the problem presented by the disease, such as will

furnish a rational foundation for the consideration of preventative measures.

To make the matter as clear as possible, I will first state exactly what I believe to be the nature of the epidemic, and then discuss the propositions that are involved in this view of the disease.

Kála-ázar is, I believe, an epidemic of malarial fever, which originated in an intensification of the ordinary malarious fever in the Rungpore district in the early seventies by an extraordinary succession of unhealthy seasons, until it attained to the powers of infection; and that it has spread for the last twenty years slowly up the Assam Valley as in a wave of increased mortality due to this intense communicable type of malarious fever, having found in the districts which it has traversed a suitable soil for its propagation.

This view involves the following propositions: (1) that malarial fever may, under exceptional circumstances, become intensified to such a degree as to attain to epidemic properties; (2) that malarial fever when it has become sufficiently intensified and is introduced into a suitable soil, may spread in an epidemic form over considerable areas, and cause an exceptionally great mortality, in the absence of any changes in the physical or sanitary condition of the places affected; and (3) that the germs of the disease must in some way be able to get out of one person into another, either directly or indirectly, after passage through the soil, which is obviously necessitated by the conditions of the second proposition.

The first of these propositions requiries very little argument, as the facts given in the last section regarding the "Bardwan fever," which was of an admittedly malarious nature, is alone sufficient proof of its truth, while many other instances in which malaria has become epidemic are on record. It is also well known that malarious fevers vary very much in their intensity in different places, and in different years in the same place. When we talk of an unhealthy year in India, we usually mean one in which the mortality from malarious

fevers was unusually high. The probability is that such a year will be succeeded by one that is comparatively healthy. But suppose that on the top of one very malarious year another similar to it should succeed, as occasionally occurs, then we find the death-rate from fever increases very greatly both in its prevalence and its fatality, in other words, in its intensity. Now, suppose it should happen that a third bad year should immediately follow the other two—a coincidence that must fortunately be exceedingly rare—would it not be expected, nay, would it not be certain, that an exceptional outbreak of malarial fever of a very intense type would result, and is it not conceivable that it might even attain to such an intensity as to actually become communicable? But I have shown that the Assam epidemic fever originated in a succession of four out of five such unhealthy years.

The second of the above propositions is the most important and essential to my argument. In the case of the Assam and Lower Bengal epidemics of malarial fever, the communicable type was introduced into places where malarious fevers were already only too prevalent, although not previously epidemic, namely, the foot of the Garo Hills, and in Nuddea and Bardwan, etc., respectively, so that the difficulties of demonstrating my second proposition in these instances are well nigh insuperable. If, however, it can be shown that malarious fever has been introduced by human intercourse into any place which was previously free from it, and that, when so introduced, it spread in the form of an epidemic, for which purpose the germs of the disease must necessarily pass from one person to another and be introduced into the soil from the bodies of the carriers of the disease, then both the second and third of my propositions will be demonstrated. Now, there are certain parts of the world, mostly islands, in which malaria is unknown in spite of a climate and soil which appear to be most favourable to its development, and its absence can only be accounted for on the supposition that the malarial germs have never been introduced into the soil. There

is also an instance recorded within modern times where malarial fever did become introduced into two islands that were previously free from it ; I refer to the well known instance of Mauritius and Réunion. Up to 1865, malarial fever was unknown in them, although low-lying, undrained, marshy land abounds, more especially on the leeward side of Mauritius. In 1865, malarial fever was introduced into the island by coolies from India, and this, be it remarked, at the very time when the Bardwan fever, which was an intense communicable type of malaria, was raging in parts of Lower Bengal, and it is recorded that such a fearful epidemic ensued that one-third of the inhabitants of the island are said to have died in the course of four years, while out of a population not exceeding 130,000 in the area affected, the deaths in 1867 were 31,920. Moreover, the disease is there in an endemic form to the present day, although the neighbouring island of Rodrigues is still free from infection in spite of its being under precisely the same climatic conditions. The fact that this island is still free from malaria, points to the conclusion that when the epidemic subsided in Mauritius, the fever lost its communicability, and therefore its power of being introduced into new places, at the same time that it lost its abnormal intensity. Here, then, at all events, the malarial germs must have been introduced into the soil of the island from the imported coolies who brought the disease, and finding a suitable *nidus* for their development, and people who were very susceptible to the poison they were able to rapidly spread themselves over the island, and to cause the worst epidemic of malaria recorded in modern history. This being the case, it is evident that malarial fever is capable of attaining to the power of at least indirect infection through the soil, as it must have done so, in order to infect the islands of Mauritius and Réunion ; and I think the evidence that I have produced in this report proves that it has also done so in the case of the epidemic fever of Assam, and doubtless also in the Bengal outbreak,

so all difficulties in the way of accepting this view of the disease vanish.

It may be at once admitted that this is quite contrary to all the old ideas on the subject of malaria; but, on the other hand, it is easy to show that it is in accordance with all the teachings of modern bacteriology, and is also very strongly supported by analogous instances in the case of some of the other germ diseases. Thus, in the first place, it is an established principle of bacteriology (and one on which both Pasteur's inoculations against hydrophobia and Haffkine's against cholera are based) that any pathogenic germ which can be cultivated or passed through suitable animals, can be artificially intensified without any marked changes in its shape or appearance; and what is of more importance still, when it has once been so intensified, it can be inoculated from one test-tube to another for generations under favourable conditions without any marked loss of its newly-acquired powers. In precisely the same way I maintain that in the case of travelling epidemics of malarial fever, the germs have become intensified, as it were in Nature's laboratory, by unusually favourable conditions, until they attain to the power of being communicated from one person to another, either directly or indirectly, after passage through the soil; and that when once this power has been attained, an epidemic is started, which may go on spreading from one district to another for years without any alterations in physical or sanitary conditions of the affected places, as long as it finds a suitable soil for its multiplication and people whom it may attack, or until it loses its intensity owing to any unfavourable conditions. Unfortunately it will be impossible to scientifically demonstrate this proposition until the malarial organisms can be artificially cultivated (which it would have been only a waste of time for me to attempt under the conditions in which my work was necessarily carried on, seeing that all efforts in this direction, which have been made in the well-appointed laboratories of Europe by the most skilled bacterio-

logists, have hitherto failed to produce this very desirable result). We may, however, consider if there are any analogous instances of diseases produced by other germs, which are not ordinarily infectious, becoming so when intensified in any natural way, for even a single example of this taking place would lend great probability to the same being possible in the case of malaria. Here again, there is evidence at hand, for pneumonia (inflammation of the lungs), which is now pretty generally acknowledged to be caused by Fraenkal's pneumococcus, is not ordinarily an infectious disease; yet every now and then it becomes so, and attacks whole families, or, as has occasionally been seen on the North-West frontier of India, a large number of a regiment, the sick attendants put over cases even contracting the disease. Moreover, when it is thus infectious, it is also very fatal or intense. Again, if the recent views with regard to plague be verified, as seems very probable, then there is a mild non-infectious and seldom fatal form of the disease which goes by the name of pestis minor, while there is also the well-known communicable and very intense and fatal form now so serious in the Bombay Presidency, which arises by an intensification of the mild non-infectious form, possibly through the passage of the virus through the rat or other very susceptible animal.

There is then nothing inherently improbable in this view of the disease, while it is strongly supported both by what is known by analogous germ diseases, and by all the doctrines of recent bacteriological research. Moreover, it fully explains all the mystery surrounding both the Assam and Lower Bengal outbreaks of epidemic malarial fevers, in a way that no other view of either disease does, that is in consonance with the facts.

The way in which the germs get from one person to another, and the reason of the decrease of the affection in any given place after a few years, remain to be considered. Abundant evidence has been given in Section VII to show that it is generally necessary for a person to live in or visit an

infected house or village in order to contract the fever. It was also shown that the disease usually begins in one place by a person who has contracted the fever in another coming to live in the uninfected village, and that the next cases occur in the same house as he is residing in, and then it spreads to others, often those who frequently visit the first cases when ill, being the next to suffer. The majority of new cases begin during the rainy season, and there is always much less fever in the cold-weather months, when the ground is dried up. Further, the infection sticks to houses and the soil of affected villages, and, in a few instances, where the inhabitants of an entire village or coolie lines have been moved to a new site, which may be only 200 yards from the old one, during the cold-weather months, when the fever is at a minimum, then the disease has not broken out in the same way among them during the ensuing rainy season, as it had in previous ones. This shows that they had left the infection behind them in the soil of the old village site. How can all these facts be explained? It is evident that the malarial germs must escape from the bodies of those who are suffering from the fever in some way or other. Dr. Patrick Manson and others have recently suggested that this takes place in ordinary malaria by mosquitoes sucking up the blood from infected persons, and then going and dying in water in which the germs contained in the blood that they have extracted escape and multiply. Although Surgeon-Major R. Ross has recently published some interesting experiments on certain changes which the crescent-shaped form of the parasite goes through in the stomach of the mosquito, there is as yet very little evidence in favour of Dr. Manson's ingenious idea; and, although I have constantly borne it in mind, I have not met with any facts in the course of my investigation that lend support to it. On the contrary, it would be very difficult or impossible to explain the facts that I have adduced as to the infection in *kála-ázar* on this hypothesis, while the crescent-shaped bodies are not met with in *kála-ázar*, and even

if they were, it is much more reasonable to suppose that the changes which they have been seen to undergo outside the body, are only those which naturally take place in such organs as the liver and spleen during life, and account for the relapsing nature of the cases in which they occur. The facts can be easily explained in a much simpler way. We know that in the great majority of cases malarial fever originates from the inhalation of the germs which have obtained access to the air either by being carried up by the evaporation of moisture from drying up marshy ground, or, as I have shown elsewhere, by the displacement of the air from the soil by rising ground water forcing them up into the atmosphere. They pass down into the alveoli of the lungs, and have only about one thousandth of an inch of tissue to traverse, in order to reach the blood in which they live and multiply, and this they can get through by means of the power of motion that they possess. When they have completed that part of their life history, which is passed in the human body, it is equally easy for them to get back again into the air-sac, and it will be easier for them to escape from there into the air, than it was for them to effect their entrance from it, for the fine hair-like cilia, which are constantly waving upwards in the air passages, will assist their exit while they would oppose their entrance. When it is remembered that *kála-ázar* is essentially a very chronic and relapsing kind of fever, and that every day on which the temperature rises each of the malarial germs in the blood will divide up into some ten to fifteen little ones, it is easy to understand that when a man with the fever is constantly exhaling them, not only day after day, but week after week, and month after month, then anyone else living in the same house will be very likely to breath them in; and if his white blood-cells are not very active in killing them as fast as they gain entrance to his blood, then he will fall a victim to the disease and contract the fever. The way in which whole households die of the affection, is thus easily accounted for. But in addition to this, the germs must also gain access to the soil

in the immediate neighbourhood of a house that is occupied by anyone who is suffering from the disease. During the early rainless cold-weather months, when the ground is dried up, the germs which have reached it will lie dormant; and as those who have the disease so often lose their fever at this time of the year, the opportunities of direct infection through the air will be also less than at any other time, and new infections will be at a minimum. As soon, however, as the first few inches of rain fall, as usually occurs in Assam in April, and sometimes even in March, then the ground will once more become moist and warm, and the conditions will become most favourable for the multiplication and escape into the air of the temporarily dormant germs, new infections will again become common, and the ordinary outbreak of the rainy season will recur. When this has gone on for some five or six years in any place, a large number of the inhabitants will have died of the disease, while others may have had a slight attack, which is not distinguishable from ordinary malarial fever, but which protects from a more serious one, while the rest are insusceptible to the disease, or have not come into sufficiently intimate contact with infected persons to get it, and so the epidemic fever declines. The fact that the later cases last a much longer time before they are fatal than the earlier ones, while more of them recover, shows that the most susceptible die first, and later those who have more power of resistance succumb, or in other words, the disease loses its power of attacking the surviving inhabitants of the place, and so it gradually dies out. Whether it leaves the place or district more unhealthy than it was before, is very difficult to say. There is also evidence to show that a site which has been deserted on account of the fatality of this epidemic, may yet be safely inhabited by the people who had previously deserted it, after it has been unoccupied for some years, for this is recorded to have taken place at the foot of the Garo Hills, in the very part where the Deputy Commissioner of the district was subsequently unable to find a single case of the disease. This indicates that the intensified germs

must be frequently passing through the human system in order to keep up their intensity, otherwise they will revert to the ordinary type, just as artificially intensified bacteria require every now and then to be repassed through susceptible animals if their power is to be maintained at a very high pitch for long periods. Whether these are the correct explanations of the facts recorded relating to the spread of the disease, is of much less practical importance than the knowledge of the facts themselves, for they at least may be safely relied on in considering the measures which are necessary to check the spread of the epidemic, and it is for this reason that all controversial matters have been treated of in this section separately from the previously recorded facts which require to be explained.

SECTION X.

RECOMMENDATIONS.

We have seen in Section VI that the nature and past history of the epidemic fever, which has been spreading slowly, but surely, up the Assam Valley for over twenty years, affords no hope that it will spontaneously subside until it reaches the hills that form the easternmost limits of the valley beyond Dibrugarh, for although the Mikir Hills will doubtless delay its spread into the Golaghat subdivision for a longer or shorter time, yet the fact that it has already invaded Bishnath, and the facility with which it crosses the Brahmaputra river, render it certain that even if the tract of jungle between the Nowgong and Golaghat districts forms an insurmountable obstacle to its progress in that direction, it will nevertheless gain admission by the lines of communication along the waterway which forms such a magnificent highway for the trade of the valley.

Nor will the policy, which has hitherto been adopted of watching its progress and building dispensaries to help to mitigate the miseries and death-rate which it causes, suffice to check its progress in the smallest degree, while no sanitary measures short of an entire alteration of the soil of the whole valley into one which is unfavourable to the development of the malarial organism, will suffice to check its spread, though they would doubtless in some degree lessen its severity.

It may at first sight appear that the proof afforded in the earlier sections of this report that the disease is entirely malarial in its nature, and the light that has been thrown on its origin will not help us much in finding a remedy for the evil; but on the other hand the facts given in Section VII, illustrating exactly how the disease really travels from village to village and house to house, indicate the lines on which any preventative measures must necessarily be based. The great difficulty lies in proposing

any means of checking the disease which come within the bounds of practical policy, but the difficulty must be faced. It will then tend to clearness if I first lay down the measures which are likely to be efficient in staying or limiting the ravages of the epidemic, and afterwards indicate the manner in which they can best be carried out. The measures to be taken on tea gardens will be separately considered, as here it is possible to lay down a definite plan of action with a certainty that it can and will be carried into effect.

The only way in which the spread of the epidemic can possibly be checked is (1) by cutting off inter-communication between infected and non-infected villages and districts, and (2) by moving the houses of attacked villages from the infected sites to a short distance only during the cold-weather months, when the fever is at a minimum.

The extreme difficulty of carrying out such measures will be at once apparent, in fact, they will be absolutely impossible without the cordial assistance of the people of the affected districts. If, however, the native population are willing to adopt a few simple measures based on the knowledge that we now possess regarding the methods and ways in which the disease is spread, then very much may be done to check the mortality and spread the disease.

Now I have shown in Sections VI and VII that the people in isolated instances in various districts have themselves, on their own initiative, adopted the very measures which are best fitted to check the mortality and spread of the epidemic, and it is indeed only from the favourable results that have followed their action that clear evidence of the efficiency of the very measures which I am about to propose has been obtained. Moreover, I have shown in Section VII that the people of the affected districts are in such terror of the disease, that they have even broken through their social and religious customs in their endeavours to escape the infection. I am therefore strongly of the opinion that in spite of the essentially lazy, easy-going, and intensely conservative

character of the Assamese people, they are in this instance most willing and anxious to carry out any measures that are in the least degree likely to mitigate the ravages of this decimating disease. I made a special point in the course of my tours of inquiring from the villagers if they were prepared to adopt certain measures for the prevention of the infection of their villages, or the eradication of the disease when once it had broken out amongst them, and found that they were quite ready to adopt any suggestions, even to the extent of moving their villages to a new site, although in this latter case they were confronted with a great difficulty, which was that if they moved unless they gave up the land on which their houses stood and took over the new site on January 1st, which was obviously impossible, for it would take them at least a month to effect the move, they would have to pay a year's rent for both pieces of land, which they were even less able to do when affected by the epidemic than when in health. It is all the more surprising that they should nevertheless have moved their villages to new sites in several instances in both the Nowgong and Mangaldai districts, purely and solely in order to escape annihilation by the disease. In this connection the following extract from the administrative report of the Garo Hills district for 1886-87 showing the good effect of the above measure among the Garos who so extensively adopted it will be of interest. "In other villages it would seem as if a wave of malaria had passed over them, so numerous were the cases. I was urgent with the people in such cases to move their villages at least during the rains, and to betake themselves to temporary houses on the hill tops, and near good hill streams. This the people had done in some instances of their own accord with most successful results." Again, I have shown that in Mangaldai there is good evidence to show that the spread of the disease to certain villages has been prevented by the people finding out for themselves that it was infectious, and cutting off all communication with the affected villages.

I propose then to take advantage of this attitude of the people, and to assist them to help themselves in the following two ways:

(1) By writing a pamphlet, to be translated into Assamese, Kachari, and any other languages that may be thought advisable, setting forth in the simplest words the methods in which the disease is commonly spread, and the precautionary measures which should be taken in order to avoid the introduction of the disease into previously unaffected villages, and the best measures to adopt to prevent its spread through a village when once it has begun, etc.

(2) To give facilities to those villages which are already severely affected by the disease to move from the infected sites to new ones a short distance off, while still retaining the land which they are cultivating, which will prevent their going to a distance and so running a risk of carrying the infection to new places. The move should be made during the dry cold-weather months, from December to February or the middle of March, being the most favourable ones.

As regards the first suggestion, I have written a pamphlet (see Appendix No. 2), which, I hope, will prove suitable for the purpose, and which should be distributed gratis to the heads of villages in both the affected and threatened districts, and to any other persons whom it may be thought advisable to instruct on the subject.

The second proposal is based on the fact that if the people of an affected village move their houses from the infected site to a new one, which need not be more than two hundred yards from the old one, during the cold-weather months, when the fever is at its minimum, then the usual severe annual recurrence of the fever in the following rainy season does not ensue. It must be admitted that so far this has usually been carried out after the village had suffered from the epidemic for some years, and when it may have been on the decline, so that it is not certain if quite as good results would be obtained by moving a village during the

height of the epidemic. There is, however, no reason why it should not, and if carried out in the first cold weather after infection it should even be more likely to be successful than if delayed for some years, and at least it would be certain to very much lessen the mortality during the next year, while if the disease did eventually recur to a severe degree the measure might have to be repeated. It may be objected that such a move would be the very way to spread the disease, and this would be true if it was proposed to remove to a considerable distance, or into a previously uninfected part. Fortunately it is only necessary to go a very little distance in order to escape from the infected area, as is conclusively proved by the examples both in villages and on tea estates, such for instance as those given on pages 154 and 163 of Section VII. In the cases of villages it is impossible to separate the healthy from the sick as is recommended on tea gardens further on in this section; but in the instances I have met with the presence of a few chronic cases has not caused the re-appearance of the disease after removal from the infected site.

The proposed form of assistance to encourage them to move will at the same time ensure that they do only go a short distance, and is moreover one that my inquiries in the villages leads me to believe would be sufficient inducement to them to move, in at least a great many cases. It is in the first place, that the necessity of their paying a year's rent for the land on which their village stands, and also for the site to which they remove, should be done away with, which would be sufficient in case of many of those villages which have already severely suffered from the epidemic to induce them to abandon the infected place in which they are living; but further, in order to get the villagers to move during the first cold weather after their village has become infected, and consequently before they have realised to the full extent the decimation which it will be certain to cause among them, it will be necessary to give them the land for a new site for their village rent-free for one year,

while to prevent their moving to a distance it should be a condition of their receiving this aid that they still retain and cultivate the same land that they previously held.

One difficulty that was pointed out by some of the villagers was that they might not be able to find a suitable high piece of ground big enough to accommodate the whole of their village. This may be true of some of the more thickly-populated districts, such as the southern parts of the Mangaldai division, but is rather of the nature of an advantage than otherwise, as it would necessitate the splitting up of some of the larger villages, which would help to prevent a recurrence of the disease among them, for the smaller communities into which they would have to divide would be less likely to become re-infected by the disease than a single large one would be.

The question remains—what would be the cost of such a measure as this? As I only propose that the rent of the actual sites of any infected villages, which can be induced to move, should be remitted for one year, and the revenue from cultivated land would be untouched, while it is probable that it will only be carried out in some of the districts at first, the initial cost would be small, and would, I feel sure, be ultimately more than recouped by the resulting saving of lives, preventing the great loss of revenue from whole-sale falling out of cultivation of the land owing to the great mortality among the active able-bodied workers, which has always followed the invasion of any district by this epidemic.

The application of the above-described measures to the different districts will be discussed under the following three heads: (1) Those which have been infected for some years, such as Nowgong and South Mangaldai; (2) Recently infected parts, such as Bishnath and Tezpur, the north of Mangaldai; (3) Uninfected, but threatened districts, such as Golaghát and the more easterly divisions of Upper Assam. The Garo Hills, Goalpara, and Kamrup districts do not require consideration, as there appears to be no

epidemic manifestation of the disease in these parts at the present time, although cases of ordinary malarial fever and other conditions are still returned under the head of *kála-ázar*. The nomenclature of these diseases will be discussed later.

(1) (*a*) *Nowgong*.—The epidemic reached the extreme north-east end of this district some three years ago, as far at least as it is inhabited, which is only a few miles beyond Silghat. It has largely subsided in the southern and western parts of the district, and is on the decrease in the central parts also, including the station and surrounding villages, but is still increasing in the north-eastern corner, especially along the eastern end of the Kulung river, and also in a few of the more sparsely-populated parts between the Kulung and the Brahmaputra rivers which have been comparatively recently attacked. This decrease is probably greater than is indicated by the slight fall in the number of deaths returned from fever and *kála-ázar* in 1896, for there appears to have been a distinct improvement in registration during the last year in this district. A further decrease in the mortality of the district as a whole may be looked for during the current year (1897), so that no very active measures are required. There is still some scope for the beneficial effects of instructing the people in the necessary precautions to be taken for preventing the spread of the disease in the more recently infected parts. The villages along the Kulung are practically continuous, so that this would not be a favourable part for moving the villages from the infected sites, but some of the more scattered places towards the Brahmaputra river would be much more suitable for the carrying out of this measure.

(*b*) *South Mangaldai*.—Here again, the disease has greatly decreased in the south-western parts, especially around Sipajhar and west of Rangamati. North of Mangaldai town, and especially in Bor Autola, it is still bad, but has only just begun to affect a few villages in the southern parts of the Kalaigaon tahsil, which is more sparsely populated than the

southern parts of the district. It was here that the people seemed most alive to the infectiousness of the disease, and most ready to adopt measures against it, so that by teaching them exactly how it is really spread and the necessary precautions to be taken against it, and at the same time encouraging and helping them in moving their villages from the infected sites during the cold weather, much may be done to both to check the spread of the epidemic and to reduce the mortality and consequent reversion of cultivated land to a state of jungle, such as I have seen taking place in parts of this division.

(2) *Recently affected parts of the Darrang district.*—Here we have to deal both with a rural and an urban population. Taking the Tezpur division as a whole first, it must be remarked that although both the town and the villages around it have been affected for some three years, the death-rate appears to have been small and the spread of the disease less rapid than it always has been on the south bank of the Brahmaputra. The soil is probably less favourable to the multiplication and survival through the cold, dry weather of the intensified malarial organisms. Nevertheless the disease has undoubtedly gained a footing in the district, but its slower spread should render it easier to check than it otherwise would have been, and vigorous measures are therefore still more imperative, and likely to give better results than in the lower and damper parts of the valley. Most of the villages for 5 or more miles around Tezpur town, are affected by the disease, although not yet very severely; and, as far as I could ascertain, there are also other isolated villages much further out in the district, right among the tea gardens, which are consequently threatened, although I do not know of any that are as yet infected; and, if this is the case, the measures detailed in the latter part of this section should be efficient in preventing any great mortality on them. The measures already described will then also be applicable to this district, only they should be carried out in a more systematic way than is necessary or advisable in districts that are already severely and universally attacked, with a

view to prevent the disease from spreading to the unaffected parts, and to stamp it out of those in which it has already got a foothold. For this purpose all attacked villages on the margin of the affected area, as well as isolated newly-infected ones should be carefully sought out, and the neighbouring ones warned against intercourse with them, while in the cold weather active steps should be taken to move the infected ones, as already described. If this was systematically and thoroughly carried out for two or three successive years, I believe the march of the epidemic would be most materially checked. Similar steps should be taken, as far as possible, in those around the town itself. The same remarks apply to the northern part of Mangaldai, which is beginning to be affected, and as it is largely inhabited by Kacharis, who are less loath to move than the Assamese people, the necessary measures should be easier to carry out here.

With regard to Bishnath, the disease is only just beginning near the river-ghât, although it is present in the more western parts towards Tezpur in the same degree that it is in the Tezpur division itself. Vigorous measures should be taken on the lines already described to stamp out the disease and to check its spread, especially from the recently affected portion near the landing place.

Tezpur town.—It has been pointed out in Section VI how rapidly the disease became widespread soon after Gauhati was badly infected, owing to its travelling along the much frequented lines of communication from that town to the Nowgong and Mangaldai districts. The same was true of the "Bardwan fever" when it reached the town of that name. That the disease is already beginning in Tezpur town is then a fact of the gravest significance, although fortunately it is not yet attained to epidemic proportions, so there may yet be time to do something to lessen the danger of its widespread dissemination from this centre. Now we have seen in Section VI, page 144, and Section VII, page 160, that the disease has been introduced here as elsewhere by means of

persons suffering from it coming to live in the town, usually with relatives. This is quite impossible to put a stop to under the present laws, but the question arises whether the dangerous condition of affairs, both with regard to Tezpur town, and also the threatened spread of the epidemic into Golaghat and the more easterly parts of the Assam Valley, is not sufficiently grave to render it advisable to take advantage of the provision of the Epidemic Diseases Act passed recently, which reads—

"In the event of India or any part thereof being at any time visited by or threatened with an outbreak of any dangerous epidemic disease, the Governor General in Council, if he thinks that the ordinary provisions of the law for the time being in force are insufficient for the purpose, may take such measures, and by public notice prescribe such temporary regulations to be observed by the public, or by any person or class of persons, as he shall deem necessary to prevent an outbreak of such disease, or the spread thereof."

Now there can be no question but that *kalá-ázar* is a "dangerous epidemic disease" within the meaning of the Act, while there is good reason to believe that in the course of its 20 years' uninterrupted spread through the province, it has caused much greater suffering and loss of life than even plague itself would be likely to do, supposing it to gain access to Assam; for the latter, although very virulent while it lasts, seldom remains in a place, especially comparatively small towns, for more than a few months, while it does not cause the terrible dying-by-inches of whole families that *kála-ázar* does. If then powers were taken to send back to their own villages any cases of *kála-ázar* which might be found in other unaffected or recently infected districts or towns, which would also cover the case of whole villages migrating to uninfected parts, as I know to have occurred from Nowgong to both Golaghat and the interior of the Tezpur district, then a very great step would have been taken to prevent the infection of new places and divisions; and the very fact of such powers being in existence, would tend greatly to prevent occasions arising for their exercise. Moreover, I have met with more than one instance

in which the people were most anxious for powers to expel cases of *kála-ázar* from their village, so that such a measure would be welcomed by them. I am strongly of the opinion that the circumstances both warrant and necessitate the use of this measure if the upper parts of the valley are to be saved from the calamity which threatens them.

In addition to this the moving infected collections of houses in the town of Tezpur, or other large places which may become infected, should also be enforced, as I have already recommended in the case of the *busti* opposite the club in Tezpur, the particulars of the infection of which have been given in Section VII, page 160. It is a doubtful question whether this can be done compulsorily under the Municipal Act on the ground that it is a necessary sanitary measure, if not powers of doing so should be prescribed under the Epidemic Diseases Act for towns and any other places where it might be thought to be desirable.

(3) *Unaffected but threatened districts.*—The most important of these is the Golaghat subdivision. For the more easterly parts of the Sibsagar district and that of Dibrugarh, are not likely to be affected except through Golaghat. If then Golaghat can be saved, the whole of the upper end of the valley on the south bank of the river will also be spared, while North Lakhimpur will probably also escape, although this division is too sparsely populated to be likely to suffer severely from the epidemic. This is the most important, and also the most hopeful part of the problem, for, as has already been said, there is a better opportunity of stopping the easterly extension of the disease at the present time than there has ever been before, or is ever likely to occur again if it is neglected now, while if nothing is done the epidemic seems certain to spread to the end of the valley.

The principles on which successful action should be based are still the same as in the previous instances, but require most thorough carrying out if they are to be efficient. The principles are to prevent, as far as possible, the entry into

the district of any persons suffering from the disease by continuing the watch, that has been kept on infected persons travelling up along the grand trunk road from Nowgong into Golaghat, sending back any that may be detected, which appears to have been so far successful in keeping out the disease, and extending it to the lines of communication from infected districts by the river, for which purpose again a special notification under the Epidemic Diseases Act may be required; and secondly, finding out the first villages in the district to become infected by the disease, warning all the people around them against having any communication with them, and in the cold-weather months making them move from the infected site to a short distance where they will still be under observation.

The most essential point then is to detect the very first villages that get the disease, and these are nearly certain to be either in the mauzas adjoining the Nowgong district, or in those through which the traffic from the river passes, and will very likely be first found in some of the larger places which have most active communication with infected districts. The earliest information can be obtained from the mauzadars of these parts, while a careful watch should be kept for any notable increase in the death-rate in each separate mauza, especially during the rainy season.

The best ways of carrying out the proposed measures.

My first proposal, namely, the distribution of a pamphlet, pointing out the usual methods in which infection of villages and families takes place, should be carried out as soon as possible, as the season when the spread of the disease is most active, namely from April to October, has already arrived.

The measure of moving the sites of villages that have become infected can only be carried out in the cold weather, so that there will be ample time for a full consideration as to how and where it can best be carried out, and in the meantime it will be necessary to collect information as to the exact

distribution of the disease mauza by mauza, and village by village, in order that when the short annual time for action comes, a decision may already have been arrived at as to where the measures shall be adopted, by which means no time may be lost, especially with regard to the Golaghat division, where most careful inquiries should be made during and towards the end of the ensuing rainy season (1897), as to whether any villages have already been attacked by the disease.

The advisability of taking advantage of the provision of the Epidemic Diseases Act, as proposed, will also have to be carefully considered both with regard to towns and uninfected, districts, more especially Golaghat.

Registration of deaths.

The subject of the headings under which deaths from this disease have been registered, and the correctness or otherwise of the classification, must be briefly referred to. In this connection I would like to point out that one of the main obstacles to the all-important early recognition of the beginning of the epidemic in any district or part of a district is largely due to the generally acknowledged deficiencies of the present system of registration of deaths by an unpaid agency in all the districts of the Assam Valley, with the exception of Goalpara, which is the only one in which anything approaching accurate figures are obtained. The remedy for the defect is as well known as the defect itself, and the difficulty in its adoption is a financial one. I think, however, it is worthy of consideration whether the present position of the Golaghat subdivision does not warrant a system of paid chaukidars similar to that in force in the Goalpara district, being introduced there for the registration of deaths in order to ensure the earliest and most correct possible information of an increased death-rate from fever in any part of the division being obtained.

Since September 1891, deaths have been returned under the heading anchylostomiasis, which is further subdivided into (*a*) anæmia or *beri-beri* of Ceylon, and (*b*) *kála-ázar*. Nothing could illustrate better the hopeless state of confusion which

ensued on the publication of Dr. Giles' report than this extraordinary nomenclature; for apart altogether from the absurdity of expecting the ignorant village headmen who register the deaths to differentiate between these diseases, which have been such a source of controversy for so many years and the subject of two special investigations, it will now be evident from the facts recorded in this report that two or more totally distinct diseases have been returned under one heading, while, as Dr. Giles himself pointed out, true *beri-beri* does not occur in Assam, although, unfortunately, he used the term on the title page of his report.

It has been shown that before the adoption of these terms cases of *kála-ázar* were correctly returned under the head of fevers, while in the last few years in more especially the Nowgong district, a large proportion of ordinary malarial fevers have been returned under the head of *kála-ázar*. The fact that *kála-ázar* is nothing but an intense form of malarial fever, which is not always easily distinguishable from the ordinary type, even by an experienced medical man, will be sufficient reason for reverting to the previous arrangement whereby these cases were returned under the head of "fevers." The deaths from fevers (and also from *kála-ázar* if the term is retained), in each mauza of the affected districts should be permanently recorded in the office of the Sanitary Commissioner, in order that the yearly progress of the epidemic may be easily and accurately followed in the future.

With regard to the term anchylostomiasis the facts already recorded, together with some further details which will be found in an appendix to this report, prove that the particular form of anæmia which is brought about by the anchylostomum parasite, and is therefore rightly called by the above name, is not distinguishable from anæmia due to the numerous other causes which are in active work in the Assam Valley, except by the use of the microscope or by giving thymol and counting the worms that are passed, and even then it will in the majority of the cases be only one factor in the production

of the anæmia ; so that, apart once more from the impossibility of the diagnosis being made by the registering agents, the condition of vital statistics of the province and our present state of knowledge do not appear to be at present sufficiently advanced to render the retention of this term advisable. If a substitute be required, the simple term "anæmia" would be the most suitable, but even this is open to the objection that many cases of *kála-ázar* would be likely to be returned under it, which is just what is most desirable to avoid, and moreover, severe anæmia is not common among the indigenous population of the province, apart from those who work on tea gardens, except as a result of malarial fever.

The term "anæmia of coolies" should, however, be retained in the returns from tea gardens, but I am doubtful if the term anchylostomiasis is advisable here either, as there is certainly a tendency to enter any case which has been given thymol under this heading, which will include practically every case of anæmia due to whatever cause, whether there were any of the worms present or not, while I have shown in the appendix that this disease is only one of the many causes of anæmia on tea gardens, although it is undoubtedly a very important one.

MEASURES TO BE TAKEN ON AFFECTED TEA GARDENS.

The early recognition of the disease.

The first difficulty which is encountered with regard to *kála-ázar* on tea gardens is the early recognition of the disease, but it is hoped that the illustrated clinical description of the disease, which forms a large part of this report, together with the clearing up of the confusion which has hitherto surrounded the subject, may be of considerable help in this respect. On the other hand, it must be remembered that the disease has been mistaken for a general but unexplained unhealthiness of an affected garden until it had gained a firm hold and had considerably increased the death-rate, which is not surprising, considering that it is but an intense form of malarial

fever, and individual cases are quite undistinguishable in the early stages from ordinary malarial fever.

The two most important points to be kept in mind are firstly the intensity and relapsing nature of the disease, and secondly its distribution in families or houses. If it be noticed that cases of fever are occurring, which are very resistent to ordinary doses of quinine, which relapse again and again, and in which the spleen and liver very rapidly become markedly enlarged, when it will often be found that the patients seem to lose all reaction to the fever, and will even deny that they have any and go out to work when their temperature is anything between 102° and 104°F., then it will be evident something quite different from the ordinary malarial fevers of Assam has to be dealt with. If then inquiry brings out the fact that the first few cases or deaths have been in one or two families, or among old coolies who have connections in neighbouring *busties* which are infected by the epidemic, then the diagnosis will be quite certain. The instance given in an earlier section, in which three brothers were the first three cases who died of the disease all within a few months, is a good illustration of a characteristic outbreak of the affection, while in the North Mangaldai garden, which has recently become infected, no less than three of the family of the first case subsequently contracted the disease. If a suspicion of the presence of the disease should arise, but doubt still remain, it would be much better to get some one who has had a large experience of the affection to give an opinion on the question, rather than wait for its presence to be unmistakably revealed by the increase of the disease, as by that time the best time for eradicating it will have passed, and the necessary measures may have become tenfold more difficult and expensive to carry out. One other point should be mentioned, and that is if on a garden where there is a suspicion of the presence of the epidemic malarial fever of Assam, it be found that fever cases continue to be seen after the end of the rainy season during the early cold-weather months, after they have ceased

on other gardens, and at a later date than is usual on the suspected one, in the absence of unusually late rains or other abnormal cause, then it will be pretty certain that the epidemic has begun.

Measures to be taken if the disease is detected early.

If the disease be recognised on the occurrence of the first few cases, as should in future be the case, then the measures to be taken will be simple, and will be comprised in the following suggestions. If the affected coolies be local labourers, their services will be at once dispensed with, together with any from their villages or other infected ones from which labour may have been derived. In fact, any garden which employs local labour or has infected villages near it, should take care to keep itself informed of any neighbouring villages which are suffering from this communicable type of fever, and all their coolies should be warned against visiting them. If the infected coolies be imported ones, they should at once be sent into hospital, and, if possible, placed in a separate building to other patients, and kept there until the case terminates in death or complete recovery. The other inhabitants of the house from which he comes should move into another one by themselves, and carefully watched for fever, and the infected house destroyed and its site abandoned. It has been proved by actual experience that it is of little or no use to simply remove those who already present well marked signs of the disease, for the houses in which they have lived are certain to be infected before the first case which has occurred in it can be diagnosed with certainty, and some of the other occupants will develope the disease if they remain in them. The infected houses should therefore be burnt down, and not rebuilt. The simple burning of the thatch roof within the mud walls of the house and then re-roofing it, has been followed by other cases occurring in the same house. If only a very few cases have occurred, the above measures may be sufficient to eradicate the disease, and the importance of its early recognition and of prompt measures for getting rid of it cannot

be too strongly insisted on, as when once it has got a firm hold on the lines of a garden it is no easy matter to get rid of it, as will be seen immediately; and when I mention that the manager of a garden which has suffered severely from the epidemic for the last four years, and who is not yet at the end of his troubles, estimates his loss through its ravages at a lakh of rupees, its prevention will be regarded as better than its cure, especially as it has already been pointed out in the clinical section that no method of treatment has yet saved more than a small percentage of cases when once the disease has become established.

Measures to be taken if a coolie line has become severely affected.

If the disease has already got a hold of one or more of the coolie lines of a garden, what is to be done? In discussing this question it must be borne in mind that the soil is infected in addition to the houses of the lines, so that the same principles that hold good with regard to the best methods of getting rid of the infection from a village, which have already been discussed in the earlier part of this section, also apply to the case of the coolie lines of a tea garden; only in the latter case there is the satisfaction of knowing that they can and will be carried out if my conclusions with regard to the nature of the disease are accepted, and the efficiency in preventing or eradicating the disease by the measures here advised is confirmed by further experience. The principle is that the infected site should be moved from during the cold-weather months when the fever is at a minimum, but the particular application of the principle, having regard to the conditions pertaining to a tea garden, remains to be considered.

The following instance illustrates the good effect of this measure. A certain garden in the Kamrup district a few years ago lost some 200 coolies from the disease in the course of four years. The manager having found everything else failed to eradicate the disease, built new lines about a quarter of a mile from the old ones, and during the latter part of the cold

weather he moved his coolies into them, and burnt down the old infected ones. From that date he scarcely had a fresh case of the disease, and the epidemic died out. In this case the disease had been present on the garden for some four years, and it does not necessarily follow that quite as good a result would have followed if the move had been made in an earlier, and perhaps more acute stage of the outbreak, unless the precautions to be immediately mentioned are adopted. The same plan is now being tried in the case of a newly-infected line on a garden in the north of the Mangaldai district, so that by the end of the rains of 1897 more definite information as to its utility in this stage will be available, while in the garden of the Nowgong district, which has been already mentioned as having suffered so severely from the epidemic malarial fever, similar measures have been carried out in one of the lines.

The following measures are those which are to be recommended in the case of coolie lines which have become infected with the disease to a greater extent than that already discussed, namely, when cases have occurred in many of the houses of the lines—

(1) All new coolies should be placed in new lines, and on no account should they be put into the infected lines for a single day. The importance of this is very great, and when it is pointed out that for the cost of a single coolie accommodation can be constructed for thirty or forty, the economy of this measure will at once be evident, and it will also be the first step towards giving up the infected lines altogether. Convincing evidence of the efficiency of this measure and of the bad result of its partial neglect, will be found in the example recorded on page 163.

(2) The infected lines should be evacuated during the cold-weather months when the fever is at a minimum, the best time being in the months of January and February, and it should be completed before the first few inches of rain falls, which usually occurs in Assam in March or April.

Fortunately this is just the time when, if the pruning is well in hand, labour can most easily be spared for the work of building new lines. In carrying out this measure, the following precautions will be advisable in order to prevent the infection being carried into the new lines. All those families who have had no cases of the disease among them, should undoubtedly be moved into the new lines, and this measure will be certain to result in many of them escaping the disease who would otherwise have suffered from it during the ensuing rainy season. The difficulty is to know what to do with those families who have had cases of the disease during the previous rains, but none of whom have any fever at the time of the proposed move. If none of them have been getting fever of a persistent nature during November and December, that is after the ordinary fever season is over, they may be safely moved, but any families who have definite cases of the disease among them, or who are continuing to get fever during the cold-weather months, should not be moved into the new lines, but if they can be got out of the infected ones and put in separate ones, it would be the best thing for them. It would be advisable not to build very elaborate houses in the new lines in case the disease should break out in them and a second move be necessary, although this is not at all likely to be the case, especially as any early cases which might occur there would be detected and dealt with as suggested in the recommendations to be taken when the infection of a garden is found out early.

CONCLUSION.

I regret the great length of this report, but the confusion in which I found the subject involved has necessitated a full and systematic clinical and pathological description of the disease, while it was also essential to record ample and convincing evidence as to the communicability and origin of the epidemic. The facts have, as far as possible,

been left to speak for themselves, and discussion has been reduced to a minimum. It is hoped that a sufficienlty intelligible and comprehensive account of the epidemic has been given to serve as a basis for ameliorative and preventative measures.

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Surgeon-Captain, I.M.S.

SHILLONG, 31*st March* 1897.


सत्यमेव जयते

APPENDICES.

Photo-etching. Survey of India Offices, Calcutta, April 1897.

GROUP OF CASES OF ANÆMIA OF COOLIES.


सत्यमेव जयते

APPENDIX No. I.

ON THE ANÆMIA OF COOLIES.

In the course of my investigation into the origin and nature of *kála-ázar*, much work has been done on the subject of anchylostomiasis and other conditions in which anæmia is a marked feature, in order to differentiate these from the former disease with which they had been confused. In the course of this work, some results have been arrived at, which do not enter into my general argument on the subject of epidemic which has, for so many years, been spreading up the Assam Valley, but which may be worthy of record. It is therefore proposed to embody them in this appendix. As, moreover, my work has necessitated visiting nearly every district in the province of Assam, and has afforded me unique opportunities of learning the views of those best qualified to form an opinion on the difficult subject of the causes of the anæmia, which is still very prevalent among tea garden coolies, the conclusions which I have arrived at, after a perfectly unprejudiced examination of the question, may be of some interest to other workers on the subject, even though there is very little that is original in my observations.

The adoption of the heading of anæmia of coolies rather than the term anchylostomiasis is due to my belief that the latter is only one out of many causes of the disease, and it is largely with a view to pointing out some other factors to which I think sufficient attention is not paid that the following notes have been written:

The groups of cases of anæmia of coolies in the photograph opposite this page illustrates very well the differences both in type and degree of the class of cases which are grouped under this heading. The two cases on the right represent moderately advanced cases of the dropsical type of anæmia, while the two on the left show advanced cases of the same, the extreme left one being the worst that I have met with. The central man's anæmia was due to chronic dysentery, and the absence of dropsy in this case imparts to him a very different appearance to the other cases. The type of his anæmia was also different to that of the others, as his hæmoglobin was less reduced relatively to the number of the red corpuscles. The whole group presents a very different appearance to that of the *kála-ázar* cases shown in the illustrations opposite pages 33 and 57.

That anchylostomiasis can and does cause anæmia which, unless detected and properly treated, will by itself cause death, is an undoubted fact, but the liberal use of thymol has, to a very great extent, robbed this disease of its terrors, while the nearly universal introduction of pure water supplies on tea gardens has largely reduced the chances of very large numbers of the parasites being introduced into the digestive track within a short time, for, although the infection may be due in some cases to the swallowing of earth containing the encysted or rhabditic form of the worm (whichever may be the correct view of the development of the parasite), it can only occasionally happen that several hundreds can be rapidly introduced in this way. That anæmia of coolies due to anchylostomiasis is much less common than it was a few years back, is the opinion of both the most experienced Civil Surgeons as well as of the private practitioners in charge of gardens whom I have met. This improvement has taken place in spite of the fact that, although latrines are now frequently supplied as a result of the recommendations of inspecting officers, they are little if at all used (for to force the coolies to resort to them against their wills, is quite impracticable), which seems to show that the decrease in the disease is due chiefly to the improved water-supply, and hence affords an additional argument in favour of the more commonly-held view which attributes the main part of the infection to water.

Now it must constantly be borne in mind that the best writers on the subject of anchylostomiasis admit that as many as five hundred anchylostoma must be present for from six months to a year in order to cause anæmia in a healthy man. Although it is also true that at the time of his death very few, or even none, may occasionally be present, especially if he has had repeated doses of thymol, yet, on the other hand, if a series of cases be examined, a large number will be found in the majority of them. Again, it must be pointed out that the worms cannot multiply within the body, but every one must be introduced through the mouth in a partially developed form, for I find this is not always understood by planters.

My work on the blood changes given in the fourth section of this report proves that the essential factor in the production of the anæmia of anchylostomiasis is the daily loss of blood, while dyspepsia is a late and comparatively unimportant symptom, for it is a common experience to see a coolie in a fairly advanced stage of the disease with marked dropsy regain his appetite within a very

few days of his worms being removed by thymol, and to be quite free from the discomfort after meals, which used to trouble him greatly before.

That anæmia is the main cause of death in anchylostomiasis is proved by the fact that in five successive fatal cases, in which I estimated with accurate instruments the exact state of the blood within a few days of their death, the percentage of hæmoglobin (the colouring matter and most essential part of the blood) was, respectively, 8, 16, 10, 8, and 7 per cent. of Gower's standard, while it is generally admitted that if this necessary element falls below 10 per cent., recovery is scarcely possible, so that we see in all but one of this series the anæmia was in itself fully sufficient to account for the fatal termination, while in the other case death was caused by hypostatic congestion of the lungs and extreme dropsy of the whole body, including the chest wall and abdominal cavity. The fact that within the last few years the disease has been described more than once as pernicious anæmia by workers, who have overlooked the presence of the anchylostoma, is enough in itself to show that this is the most important feature of the affection. The beneficial effect of absolute rest in bed in cases which are apparently in a hopeless state, as will be more fully pointed out presently, taken with the well-known fact that complete rest is essential for the recovery from the worst forms of anæmia, also points in the same direction.

Granting then that the anchylostoma, when present in large numbers and for a long period, are able by themselves to cause a serious, and if not properly treated, fatal anæmia, the question remains—How much of the anæmia now seen on tea gardens is of this nature? And can these cases be distinguished from those due to other causes?

But before passing on to consider these questions, I should like to write something on the use of thymol in Assam at the present time. I have already mentioned my belief in immense value of this drug, and when I came up to Assam, nearly a year ago, I was quite prepared to find that whatever *kála-ázar* was, anæmia of coolies was practically entirely anchylostomiasis, and that the only thing needed to cure it was thymol. A little practical experience soon dissipated this beautifully simple idea, and I am now of the opinion that there is more to be feared from the abuse of the drug in Assam than from its neglect. To give an instance of what I mean. In the hospital of one of the most up-to-date gardens which I have visited, as far

as concerns good water-supply with pumps, cisterns, etc., I saw some six or eight cases of advanced anæmia which were all regarded as hopeless by the doctor in whose charge they were, and who subsequently died. They had all been treated with liberal doses of thymol, but instead of getting better as other cases on the same garden who had been similarly treated had done, they became the victims of an intractible form of dysenteric diarrhœa, became reduced to living skeletons, very like the man in the centre of the group of anæmia of coolies cases opposite page i. Now what struck me about these cases was that some of them did not present an extreme degree of anæmia, while some had a considerable enlargement of the spleen, and others had formerly suffered from dysentery; in short, their anæmia was due to different causes, but they had all been treated alike with thymol. Now I would not be understood to blame any one for this state of affairs; all I wish to point out is that in our present state of knowledge, it is difficult to distinguish quickly and certainly between those cases that are likely to benefit by the administration of thymol, and those in whom it will only cause disastrous results (for the exigencies of tea-garden work will not permit of the frequent use of the microscope), and I find in certain quarters the drug has for this very reason fallen into undeserved discredit. In fact, nothing has struck me so much as the divergent views which I have heard as to the value of this drug, and what I would plead for is a greater discrimination in its use. I had hoped to have found time, while investigating the epidemic malarial fever of Assam, to try and differentiate by means of the blood changes between those cases of anæmia which are due to the anchylostoma, and are consequently amenable to the thymol treatment, and those which are not, and although I have not been able to go into the subject nearly sufficiently to obtain any very definite results, yet the very few observations that I have been able to make are so encouraging as to lead me to record them here, in the hope that some of the private practitioners of Assam may find time to make use of their opportunities of research in this direction, which, I feel sure, will amply repay careful study.

It is a well-known fact that anæmia of coolies is much more common in the first two years after their arrival in Assam, than it is subsequently, 33 per cent. of the cases occurring in the first year and 17 per cent. in the second. Now Dr. Dobson has found 100 or more anchylostoma in 1·3 per cent. of 547 healthy coolies on their way up

to Assam, and between 50 and 100 in 3·7 per cent. more, although he was only able to examine the stools which were passed in the first five and a half hours after the last dose of thymol had been given, so that it is certain that had he been able to examine all the stools passed for twenty-four hours after the use of the drug, he would have found considerable larger numbers, for Dr. Sandwith, as a result of some special observations on the time after the administration of the drug at which most anchylostoma are passed, came to the conclusion that "if it is impossible to examine several motions, it would appear more important to examine, specially those which are passed from six to eight hours after the thymol."

In Dr. Dobson's cases then the stools were only examined before the most favourable time for finding the worms, so that it may be safely concluded that at least double the number that he found must have been present in these healthy men, for, moreover, all the worms are not expelled by one day's administration of thymol. It is then evident that at least 5 per cent. of the healthy coolies who are imported into Assam, have 100 or more anchylostoma in their intestines on arrival at their destinations—a number which, although too small to cause anæmia by itself, must nevertheless cause a considerable strain on the reparative powers of the system. The same must also be true of the coolies in their homes; but it is easy to see that while they are under favourable conditions in a climate to which they are accustomed, and getting the food on which they have been brought up and while their work is of a light, intermittent and congenial nature, they may be able to stand this strain as long as they are not attacked by any other illness. On the other hand, when a coolie, who has such a number of these blood-sucking parasites in his intestine, comes up to a different, and in the case of most of them, a much damper and in every way unhealthier, climate, when he is obliged to subsist on a diet composed almost entirely of rice, although he may previously have been a wheat-eater (in which connection it is worthy of note that it is the North-Western and Central Provinces wheat-eating coolies who suffer most from anæmia, so much so that some managers will not take them at all in spite of the fact that they are easier and cheaper to get than the jungle coolies of Chota Nagpur, who stand the Assam climate best of all), when he is obliged to work steadily, although the task exacted is by no means a very heavy one, and when in the busy and unhealthy rainy season he works for long hours, in order to make extra money,

and perhaps leaves himself too little time to properly prepare his meals, then he is unable to stand the loss of blood which formerly the system was able to repair, and he consequently becomes anæmic. If in addition he suffers from malarial fever, dysentery, or venereal disease, any one of which is capable by itself of producing severe anæmia, then an aggravated form of the affection is certain to ensue. In this class of cases, again, thymol will be indicated, but it must be given with greater care, especially if dysentery has previously been present, and should not, I think, be repeated within a short time, as a second administration may do much more harm by starting dysenteric symptoms than it can possibly do good by the removal of the very small number of anchylostoma which may have escaped the first doses of the drug, and it is of far greater importance that after treatment of the anæmic condition by drugs and rest should be much more prolonged than it very often is on gardens.

The latest views of Dr. Ruddock, who was the pioneer investigator on the subject of anchylostomiasis in Assam, will be of interest in this connection. In a very courteous answer to some questions I put to him recently, he writes—

"Of course, some of the cases (of *kál-ázár*) had the anchylostoma, but so has nearly every native of Assam, whether indigenous or imported, and will continue to do so until their habits are very different; but the harbouring of a few worms does not constitute anchylostomiasis. A healthy person can support a fairly numerous colony of them without damage, and I think it is only when he suffers from some other disease which reduces his powers of resistance that the presence of the parasite assumes importance."

The majority of the cases now met with on tea gardens of Assam are probably of this nature.

Among the various other factors in the production of anæmia, malarial fevers are perhaps the commonest and most important. When I was examining two hundred healthy coolies for enlargement of the spleen and liver on a garden during the rainy season, I also made a note of the amount of fever from which they had suffered, and found that the great majority of them had had fever at some time or other within the previous year, while a quarter of them had some enlargement of the spleen, although this was usually very slight. From an examination of the blood of apparently healthy men in the Nowgong jail, I also found that those who had suffered from even one week's fever within the last two years, had distinctly poorer blood than those who had none or less

than this amount, so that it is evident that even a slight attack of fever has a material effect in causing anæmia. It is evident then that if all coolies who suffer from it to any degree, and especially if it is of frequent occurrence, could be given a tonic containing arsenic and iron for a week or two, but more especially the former, because in the case of anæmia due to malarial fever there is plenty of iron in an organic form in the liver, etc., but arsenic is required to increase the formation of new red corpuscles, than I am convinced a very potent cause of anæmia would be largely removed.

It is not necessary to discuss such common causes of anæmia as venereal disease and dysentery, while all tea garden practitioners are fully aware of the important part that the accidents and complications of childbirth play in the production of the condition among women.

There is one other factor which, I am sure, plays a very important part in the production of anæmia of coolies, to which sufficient attention is not paid, and that is the question of diet. It is not the quantity or the quality of the diet that is at fault, for these are well looked after, but it is not always borne in mind that the first principle of dietetics is that a proper proportion of each of the primary food-stuffs is necessary if the diet is to sustain the body in a healthy state. Now these primary foods are of three classes: firstly, the proteid or nitrogenous, which will include the albuminous constituents of flesh, and the nitrogen containing portions of the cereals in which latter class they are contained in very different proportions in different grains, being in largest quantity in dal (over 20 per cent.) and wheat (13·5 per cent.), and least in rice (7 per cent.); secondly, the carbonaceous substances, such as the starches and sugars, which form the great bulk of such cereals as rice (78 per cent.) and wheat (68 per cent); and thirdly, the fats, which are represented by oils, butter, and ghee, and are only present in very small quantity in most of the cereals. If any one of these is deficient in quantity, then the diet will not be physiologically correct. Again, it is quite possible to give enough rice to supply all the nitrogenous material that is necessary; but in order to do so, it would be necessary to give three times as much as would be required to supply the necessary amount of the carbonaceous element, which would be a great waste. For practical purposes, the problem may be still more simplified, as it has been laid down that in a proper diet there should be one part of nitrogen to every fifteen of carbon, and if the necessity of a certain quantity of fatty food be also borne in mind, this will be a good

rule to work on. Now there is no fear of the amount of carbon falling short in an Indian diet, in which rice, which contains so much of it, figures so largely, but I am inclined to think that the amount of nitrogen often falls short of the necessary amount in the diet on which tea garden coolies often feed themselves. When it is also mentioned that it has been found that a deficiency of nitrogen alone in a diet is sufficient in itself to cause a marked form of anæmia, the importance of this question will be at once seen. To give an example which illustrates very forcibly this factor. Some few years ago anæmia was very prevalent in the Japanese navy under the name of *beri-beri*. (The term *beri-beri*, however, is a misnomer when applied to anæmic conditions, as true *beri-beri* is nervous disease, which is common in the Straits Settlements, and is also seen in Chinese people in Calcutta, but has never been met with in Assam, and in which there is no anæmia whatever. The sooner therefore the use of the term in Assam is dropped altogether, the better it will be, as it is only a cause of confusion.) Various remedies, both hygienic and medicinal, were tried in order to reduce the sick-rate, but without material improvement, until it was found that there was only one part of nitrogen to 22 of carbon in the diet in use, instead of 1 to 15. This was then remedied first by the use of meat and eggs, etc., but subsequently it was found that all that was wanted was the substitution of a certain quantity of barley (containing 11·5 per cent. of albuminoids) instead of a certain quantity of rice, as the former contains considerably more nitrogen than the latter. This was done in the year 1884, but I do not know in what month of the year the change was effected. The result was as follows : in the years 1881 to 1883, that is, before the change, the sick-rate per thousand from this cause had been respectively 205, 404, and 231. In 1884, it was 127, and in 1895, the first year in which the new diet was in force throughout, the sick-rate was 6 per thousand ; in 1886, it was 0·35, and in the next two years it was nil. These figures speak for themselves, and similar results have been obtained in the Singapore Jail by Dr. Rowell, by a reduction of the quantity of rice and an increase of the *dal;* but in this case I am not sure how many of his cases were of the anæmic type and how many were nervous or true *beri-beri*. I had hoped to have been able to have worked out the nitrogenous value of the diets of coolies on a series of gardens, but time has not permitted of my doing this; so I must be content to call attention here to the point, and hope it may be taken up by some of those who have many coolies

under their care suffering from anæmic conditions, as I am convinced that if the consumption of a proper proportion of nitrogenous foods, such as *dal*, could be ensured in the case of all coolies in Assam, a great step would have been taken towards the reduction of the great loss in life and labour from anæmia of coolies.

In this connection the great prevalence of anæmia among North-West and Central Provinces wheat-eating coolies is of considerable interest, for, as already mentioned, they suffer so much from this disease, that many managers will not employ them at all. Now Dr. DeRenzy pointed out, when he was Sanitary Commissioner of Assam some years ago, that coolies from these provinces had just as high a death-rate, and sometimes even a higher one, in the plantations of the West Indies, as they still have in Assam, until compulsory dieting for two years was enforced by Government. From that time their death-rate fell until it reached only 2 or 3 per cent. It is also a commonly-held opinion in Assam that the North-West coolies suffer owing to their great love of saving every pice they can, even to the extent of underfeeding themselves. This is certainly true inasfar as they will live on the cheaper rice, rather than incur the much greater expense of obtaining the higher-priced and more nitrogenous grains to which they have always been accustomed. But we know that the Bengali will subsist and keep his health on a diet which consists nearly entirely of rice, and the quantity that he consumes is extraordinary. This means that he is eating a much larger quantity of carbonaceous food elements than his system requires, in order to obtain sufficient of the nitrogenous food elements to keep him in health, and, although this is a physiologically wasteful method of feeding, yet he is accustomed to it, and can in this way supply all his wants. When, however, he gets any illness, such as fever, etc., which will prevent his digesting these enormous masses of rice, he very rapidly runs down, which shows that he has not much stamina. The wheat-eating coolie, on the other hand, is not accustomed to consuming such great quantities of food owing to the amount of wheat and *dal* that he eats, taking the place of a much larger quantity of rice, and so when he suddenly finds himself in a country where wheat can only be obtained at a prohibitively dear price, then he has to fall back on eating a larger bulk of rice, which he is scarcely able to hold in his stomach, and can only digest with great difficulty. This throws an extra strain on his digestive powers to which he only too often succumbs. He is now unable to get sufficient nitrogen out of his

diet, and soon becomes anæmic, and unless he is caught early and fed up in an "hotel system," he goes from bad to worse. In fact, I believe it would pay any garden which employs a large number of this class of coolies, to feed them on the "hotel system" for at least the first six months, or better still until after the first rainy season is past. This is I know done in some few cases, but it might with advantage be carried out much more extensively. In one or two cases in which I have seen this done, the cost of the diet was deducted from the pay of the coolie with his consent, and the balance was ample to supply him with the little comforts which he loves, and both parties were greatly benefited by the arrangement.

The following is a good example of anæmia, which was largely, is not entirely, due to bad feeding:—A coolie woman, aged 19, from the Assam-Bengal Railway, was admitted to the Nowgong dispensary during the rainy season of 1896, suffering from marked anæmia and dropsy of the feet and face.

The history of her illness was that she got on all right until her father died, after which she lived with another coolie girl, and, according to her story, she only received about one rupee a month from the contractor, and was consequently unable to feed herself properly. She soon became ill, and as she did not improve, she left the works and begged her way some 40 miles into Nowgong, living on what she could pick up on the way. On her admission she was fairly well nourished, but very anæmic, her feet and legs were swollen, and her face was puffy and had been swollen under the eyes shortly before. Bowels somewhat loose. She had not suffered from fever. Liver and spleen normal. Conjunctiva of a dead-white colour. She thus presented all the signs of anæmia due to anchylostomiasis, and was given thymol. The motions that were passed during the next twenty-four hours were washed and examined by myself, but no anchylostoma were present. On examining her blood the following results were obtained:—Hæmoglobin 11 per cent., red corpuscles 1,260,000 per cubic millimetre; white 3,250, or 1 to 384 red; sp. gr. 1·030; hæmoglobin value ·43. If these figures be compared with those given in the table on page 95 as typical of *kála-ázar* and anchylostomiasis, respectively, it will be seen that it differs from both, although it resembles that due to anchylostomiasis, except in the comparatively high hæmoglobin value, that is, the proportion of hæmoglobin in each red corpuscle of the blood. Now it has been shown that a low hæmoglobin value is characteristic of

the anæmia due to anchylostomiasis and of other conditions which are essentially produced by loss of blood, while the high hæmoglobin value of the anæmia produced by malaria is accounted for by the fact that the hæmoglobin of the red corpuscles which are destroyed by the fever is not lost to the system. In anæmia, which is brought about by improper or insufficient diet, it is only to be expected that the hæmoglobin should be reduced more than it is in malarial anæmia, but less than that due to hæmorrhages. This is exactly what we see in this case, and I have also met with it in one or two others in which the anæmia was also produced by bad diet. These observations then confirm the importance of improper diet as a cause of anæmia, and also seem to point to the probability of an examination of the blood being likely to be useful in a study of anæmia of coolies. The case above described improved rapidly under treatment with iron combined with a liberal diet; but unfortunately she died of cholera two or three months later, just before my return to Nowgong after a long tour, so that I did not have an opportunity of recording the extent to which her blood improved while she was under treatment.

While on the subject of the blood changes found in anæmia of coolies, it will be well to record some observations which prove the primary importance of proper after-treatment of this class of cases. I re-examined five cases of anæmia due to anchylostomiasis in garden coolies after an average of eight weeks from the date of the removal of the worms by thymol, but they had only been treated for a few days in hospital and then had returned to work, although in two of the cases iron had been continued for three weeks in all, including the time they were at work. The average gain in the hæmoglobin in the eight weeks since the previous examinations was 7 per cent. only, and on enquiry they were all doing half or less than half work, and would have continued doing so for a long time. Now Dr. Sandwith, who has recorded the result of the after-treatment of 173 cases of anchylostomiasis, found that during an average stay of thirty days in hospital after the administration of thymol, the gain of hæmoglobin varied "from 22 to 32 per cent., and probably continued when the men became out-patients." The advantage of the complete rest in hospital, combined with treatment with iron, etc., is very marked here, and when it is considered that the anæmic tea garden coolies were only doing half or less than half work, it is obvious that if they had been given complete rest for one month, together with

proper medicinal treatment, the resulting improvement in their blood and consequent increase in their power of working would have been so great, that in another month or so they would have made up for the lost time, and after that there would have resulted a great gain of labour, to say nothing of the doing away of the considerable risk of a coolie in such a state dying from some inter-current disease from which he would recover if in fairly good health. These observations then show the immense importance of suitable after-treatment in cases of anæmia of coolies, whether produced anchylostoma or other cause, in addition to the mere giving of thymol, and also that rest is of just as much, and in some cases of even more importance than medicines in the treatment of anæmia of an advanced type.

One other point has been brought out as a result of my investigation which must be referred to before passing on to see what practical lessons can be learnt from the points that I have mentioned. I was much surprised to find how poor the natives of Assam were in hæmoglobin as compared to the European standard of Dr. Gower's instrument (the instrument which was used was checked with another one, and found to be correct, so there is no doubt about the accuracy of the observations), and that they were much poorer in this element than Europeans living under the same climatic conditions, although this latter point is readily explained by the fact that the meat diet of the latter contains much more iron than the diet of natives does. Still more important is the fact that in some cases the hæmoglobin may be reduced to one-half without the mucous membranes showing any sign of anæmia, so that in any case where pallor of these is well marked, a severe degree of anæmia is present, while the slightest appearance it denotes a very considerable deficiency of this the most essential element in the blood. This is a point the practical importance of which cannot be too strongly insisted on, for if only all cases of anæmia could be caught in the early stage and treated properly until the blood is restored to something like its normal state, then advanced and fatal cases could never occur, and instead of getting but half or less work from an anæmic coolie, or in the more advanced stages having to spend money on both food and medicines for him, with no prospect of any return, a few weeks' or even days' rest and medicine in the early stage would prevent all this, and would eventually save much more than it would cost.

Now I am fully aware that only too often the coolie does not come to hospital until he is suffering from a marked form of anæmia,

and also that some of the worst cases are among those who have come up to the province in an anæmic condition, and who have never done a day's full work on the garden since their arrival; but this is obviously a mistake in the recruiting, and only confirms most strongly my point that prevention is better than cure, and that rest in time saves much labour in the end. Although the ideal aimed at in the following suggestions based on the foregoing facts may not always be attainable in every instance, yet I am convinced that the more nearly they are carried out on any garden, the less will be the loss of life and labour due to what should be, in its more severe forms at least, largely a preventible disease, and the greater will be the gain on any expenditure that may be incurred in carrying them out. Most of them are in active use in one or other garden, but I know of none in which all of them are carried out. It will simplify matters if anæmia cases are divided up into three classes, namely, slight, well marked, and extreme.

In order to detect the cases of anæmia while they are still in an early stage, it is highly desirable that all coolies should be examined for anæmia, say, about once a month, more especially during the rainy season for their first two years on the garden, as it is during this period that quite 50 per cent. of the cases begin. This inspection could be carried out in the lines at the morning roll-call or other convenient time, and would not take very many minutes. All that would be necessary in these cases, would be to treat such of them as it might be thought advisable with thymol, and to see that they took an iron tonic for a few weeks, which might be given in the lines before and after going out to work, as has been done in one or two gardens with some of the cases which had previously failed to improve after the thymol treatment as detailed above, with the best results. Special efforts might be made on these lines at the end of the rains when the busiest season is past.

In well marked cases of anæmia, these simple measures will not be sufficient to secure the most rapid, and consequently the most economical, results. Rest from work must also be given for a time, and it is here that improvement is most to be desired, and will doubtless be brought about when its importance is more fully recognised. The difficulty is that anæmia most often begins in the rainy season, which is just the busiest time, but a week or two of rest combined with medicinal treatment as soon as well marked anæmia is discovered, will be very soon made up for by the increased power of work,

which will result instead of a gradual but sure diminution of the small account of work that is being done to be followed by sooner or later by its entire cessation for a much longer period than would have been necessary in the first instance. Anyone who will estimate the hæmoglobin in a few cases of apparently slight anæmia will, I am sure, be speedily convinced of the necessity for rest in addition to medicine in their treatment, while the figures which I have given above, show the practical importance of it in the anæmia due to anchylostomiasis after thymol has been given. It must not be forgotten that when once a coolie has become anæmic, he will be utterly unable to earn any extra pay by doing more than the allotted task, so that if he had been previously doing so, he may become unable to afford as good a diet as he has been accustomed to, and that just at the very time that he is in urgent need of a better one. For this reason, if it is not quite certain that any particular coolie who has become anæmic, is supplying himself with a sufficient diet,—and the proper amount of nitrogenous food must be taken into account as well as the actual quantity,—then it will be of the utmost importance that he should at once be put on the "hotel system," and a full and physiologically complete diet ensured. This is, I am sure, of just as much, if not of even more, importance than any medicine not excluding iron itself, for what is the use of pouring iron into a man who is living on a diet which in itself will be an efficient cause of anæmia.

In the most advanced stages of anæmia the above measures will again be of primary importance, but one or two others may also be indicated. Thus, if dropsy be present much good may be done by the use of Southy's tubes for the removal of the œdema from the legs and other parts of the body. Caution must be used in the administration of thymol in such cases, and cardial tonics, of which strychnine will be the safest and best, will be indicated for the dilation of the heart, which is nearly sure to be present. It is in these cases that the microscope is of the greatest use in deciding if anchylostoma are present in any numbers before thymol is given. In these cases once more the primary importance of absolute rest, even to the extent of putting the patient to bed, and, if necessary, placing a sick attendant over him to see that he stays there, must be insisted on, and I was much interested to hear from Dr. Elliot, of the Jorhat district, that he had recently carried this out in two cases of very advanced anæmia, in which the patients had done no work for months, and yet had made no progress at all, with the

result that at the end of two months he could not find a trace of anæmia in them; and these people who would otherwise have had their names cut and been lost to the garden after great expense had been incurred in feeding and treating them for many months, became once more healthy working coolies. This is a measure which should be carried out in all very bad cases of anæmia, and together with good feeding would make the loss of either the permanent labour, or the life of a coolie from anæmia of much rarer occurrence than it is at present. I feel sure that these various points only need be brought to the notice of such as do not already carry them out, in order that they may be given a fair trial, which could only lead to their more general adoption than is at present the case.

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APPENDIX No. II.

ON THE SPREAD OF KÁLA-ÁZAR, AND HOW TO CHECK IT.*

As the disease called *kála-ázar* is still spreading slowly, but surely, from one village to another and from one district to another, and as it has been doing so for many years, the Government caused an inquiry to be made into it. As a result of this inquiry, many facts about the way in which the disease is carried from one place to another, have been found out. It is therefore desired to instruct and teach the people how the disease is spread, so that they may be able to know how to prevent the infection being introduced into their villages. This little book has been written and distributed for this very purpose, and if the people will be careful to carry out the instructions contained in it, very much may be done to check the further spread of this deadly disease.

How to recognise the disease.

In order to prevent cases of the disease being brought into villages which have been previously free from it, it is necessary that the villagers may be able to recognise it and to take the proper steps to prevent it before it gets bad in the village. It has been found that villagers do very soon recognise the disease as a different kind of fever to that which they have always been accustomed to before the introduction of *kála-ázar* into their villages. It may be told from the ordinary fever in the following ways: the ordinary fever lasts usually for a few days only, generally not more than ten days, and then the sufferer gets well again, and it is only in a few of these cases that death occurs, and then it will take place within a few days. This is the kind of fever that the people have always been accustomed to as long as they remember. Now, the fever of *kála-ázar* is quite different to this, for it goes on and on for from two to four weeks at a time, and then after stopping for a time it begins again and lasts for several weeks more. By this time the spleen will have become enlarged, and the sufferer will get thin, especially in the face and arms. He will often look fairly well, and not be in distress, and may eat fairly well, and yet all the time his skin will be hot. He will also become gradually bloodless, yet his face and skin will get darker than it was before his illness. His feet may

* This is intended to be translated and distributed in the villages in the form of a pamphlet.

now begin to swell, and he will continue to get fever, and will waste away until he dies. Instead of only one or two cases dying out of a great many, as in the case of the ordinary fever, nearly all of the *kála-ázar* cases will die, and only very few will recover.

But there is another way in which it may be known that *kála-ázar* has begun in a village, because when one man dies in a house another is nearly sure to get the disease, and one who lives in the same house, or who often visits the first case during his illness will soon fall sick, and after some months will die in the same way. This tendency to attack several members of the same family is the most definite characteristic of the disease, and one which marks it out as something quite different from the ordinary fever.

The way in which it is introduced into villages.

It is very important to know how the disease first begins in a village, as then it will also be known how it may be kept out. There are several ways in which it is introduced. The most common one is by some person who is suffering from the disease coming to live in a village where there were no cases previously to his arrival. He generally comes to live with some relation, often in the hope that the change of air from the village in which he contracted the disease will do him good, and perhaps cause the fever to leave him. He will generally therefore have suffered from the fever for a long time before he comes, and so he will present the typical appearance of the disease, and his face and extremities will be thin, while his belly will be prominent owing to the enlargement of the liver and spleen, by which characteristics it will be known that he is suffering from *kála-ázar*. Another common way is for a person of a healthy village to go and live, perhaps for a week or two, or even for a few days only in an infected village, and then to return to his own village with fever, which goes on and on until it is evident that he is suffering from *kála-ázar*. After he has lived in the village for some weeks, especially if it is during the rainy season, and he is still suffering from fever fairly regularly, one or more of the inhabitants of the house in which he is living will also begin to get fever of the same persistent type, and then others in the village, especially those who visit this house will begin to suffer, and there will be more deaths than usual from fevers during the rainy season, while during the following cold-weather months it will be noticed that the fever runs on later than it usually does at this time of the year. It will then be certain that the

dreaded disease *kála-ázar* has broken out in the village. Another common way in which the disease is introduced into a village is by either one of the residents of this village going to visit a relative who is suffering from the disease in another neighbouring village, and perhaps stopping a day or two in his house with him. He then returns suffering from fever, which at first is thought to be ordinary fever until it goes on and on, and he begins to get the symptoms of *kála-ázar,* while others of his household and others in the village get the disease in the same way as in the previous instance. Sometimes it may be introduced by a person who has the fever coming to visit a relative in a neighbouring uninfected village every now and then for a day or two, although he does not regularly live in the village, but during his visits some one in the house contracts the fever, and it spreads as before. Instances have been met with in which the disease was introduced in all of these various ways. Sometimes the disease does not appear in a household until some time after a person, who had the disease and had been living in this house, had left it again and returned to his own village, so that in such a case the house itself must have been infected during his stay, and later on the affection broke out in the house, usually during the rains.

The course of the disease in an infected village.

As soon as several cases have occurred in a village, it will spread steadily through it, as there are plenty of opportunities for infection to take place. It will be worse during the ordinary fever season, which is in the rainy months, but as soon as the cold weather sets in, and the ground becomes dry, the fever will become less, and those cases which have not died will often lose their fever for a time. As soon as the rain begins to fall again in April of the next year, fresh cases of fever will begin to occur again. This shows that the infective agent has got into the soil, but it is dried up during the cold season, but comes to life again and causes a new outbreak of the disease as soon as the ground becomes moist and warm. Both the houses and the soil around them then have now become infected, and as long as the people remain in this infected place, so long will they continue to suffer severely from the epidemic until it has been present among them for six or seven years, and a great many of them have died of the disease, while others of them are very ill with it. Whole families will die of the disease, or only one or two children may be left, and when these are taken into another house, the fever may appear in that house also.

How to avoid infection of a village.

Now we know in what way the disease is carried from one village to another; we also know how to prevent the infection being introduced. When the disease is first beginning in a district some of the larger places, which have much traffic with those parts where the disease is bad at the time, will be the first to become infected by it through persons with the fever coming to live in these places from the infected districts. As soon as it gets bad in these larger places, it will begin to break out in some of the smaller villages near them by persons from these villages going to stay for perhaps only a few days in these infected places, and returning to their villages with the fever on them, and subsequently others in the village will get it. The infection spreads in this way chiefly during the rainy season, extending from the fall of the first few inches of rain in April or May up to the end of October. If then a village wishes to prevent the disease being introduced among them, it will be necessary for them to find out what villages near them are affected by the disease, and to cut off all communication with them as far as possible. To carry this out they should not allow any one from the affected villages to visit them, nor should they visit any one in these villages, even if they are relatives of theirs. It is especially important that any of the villagers who may have to visit a neighbouring large place or market town in which the disease is present, should be very careful to avoid sleeping in this infected place if they can possibly avoid it, and this is especially the case during the rainy season when the disease is most active. When any village has many other affected villages around it, it is still possible to prevent it being introduced into their village by cutting off all inter-communication with the infected villages, and even when one part of a village is attacked by the disease and the other parts cut off all intercourse with the infected part, the other parts may escape the introduction of the disease into their part of the village for several years, or even entirely. Several instances of this have been met with during the course of the Government inquiry into the disease, so that it is easy to prevent the spread of the disease if only the proper precautions are taken by the people themselves, as they have successfully done in some instances. In one of the *paras* of a village which so escaped, the headman forbade those of the villagers who lived in his *para* to have any communication with the other infected *paras*, and would not allow them to visit their own relatives who were sick of this infectious disease, in those parts of the

village, and the result was that although the other *paras* had suffered from the disease for several years, his *para* had entirely escaped owing to his wise precautions.

The two important things then are—firstly, to prevent those who are suffering from the disease from coming into the village to live, even with their own relatives; and secondly, to stop any of the inhabitants of the village from visiting and staying, even a day or two, in any village or place in which the disease is prevalent.

If a case should occur in a village who has come from another already infected village, he must be sent back to his own village without any delay. If, however, the first case is one of the regular inhabitants of the village, then it will not be possible to send him away unless he is willing to go into a Government hospital, which would be the best thing for both himself and the village in which he lives. If he will not do this, then it will be necessary for the other villagers to avoid going into his house to visit him, lest they also get the fever. All the persons who are living in the house with the sufferer will be in great danger of contracting the disease from him. It will not be of much use to move him out of the house in which he has been living, especially if he has been ill for several months, as will most likely be the case before it is recognised that he is suffering from *kála-ázar*, for the house will have already become infected, and others in it may get the disease after the first case has left it. The best thing to be done then is for those in the infected house, who are quite free from fever, to move out of it at once, but they should not be allowed to go to live in any other house in the village which is inhabited by healthy people lest they may have already contracted the disease, although they may not yet show the well marked symptoms of its presence. They should build a new house a little distance away from the infected one, and go and live there. In this way it may be possible to prevent the disease spreading in the village.

How to get rid of the disease when it has become bad in a village.

When the disease has become widespread in a village and deaths have occurred in several families during the rainy season, then it will be too late for the use of the measures described under the head of—how to avoid infection of a village, for it will be already infected. The disease will become less prevalent during the next cold-weather months, and some of the few who may have not have died during or towards the end of the rainy season, may now lose their fever for a time; but as sure as the rains will return in the

next year, so surely will many of these cases get the fever again when the rain comes, and many fresh cases will occur, chiefly in the same houses in which persons had died of the disease during the previous year. In this way the fever will break out year after year for six or seven years until half, or perhaps two-thirds, of the people will have died, and many of the rest will be too weak from repeated attacks of fever to till the ground, so that the whole village will be reduced to a most deplorable condition,—the houses of whole families who have died of the disease will be tumbling down, and the land which may have been cultivated by these people for generations will lie untouched, such is the desolation produced by this terrible epidemic.

When once the disease has got a footing in a village and has been prevalent throughout the rainy season, or a whole year, can anything be done to stop its ravages and to prevent the calamity which threatens its very existence (for in some cases whole villages have died of the disease)? To answer this question it must be remembered that by this time the houses, and even the very soil on which they stand, will have become infected by the fever-causing poison. There is then only one way of saving a village which is severely infected by the disease, and that is no less than moving the whole of the houses from the infected site. There is also only one time of the year when this can be successfully carried out, and that is during the dry cold-weather months from December to the end of February. Fortunately, this is the very time when it is easiest to carry out this measure. It has been carried out by several villages both in the Mangaldai and Nowgong districts of their own accord and with success. For instance, a *busti* in the Mangaldai town which had lost two-thirds of their people from *kála-ázar*, moved their houses during the cold-weather months from the old infected site to a distance of only about two hundred yards. During the year before they moved, they had lost twenty persons from *kála-ázar*, but in the next year after they moved there were only two deaths from this disease, and one of these had got the fever before they moved, so that it is evident that they left the infection behind them in the old village site, and it is also important to notice that they only moved a very short distance, but it was far enough to enable them to escape from further destruction by *kála-ázar*. In this case, and also in another one, it took the villagers about one month to effect the move, and the usual outbreak in the rainy season did not occur after they had moved to a new site.

If then any village which gets the disease severely were to move from the infected site during the following cold-weather months, when the fever cases are at a minimum, then they may be sure that they will not suffer in the same way during the next rainy season as they had done in the last, and if they also carry out the advice previously given for avoiding the infection of a village and prevent all communication with other infected villages around them, they ought to escape from further attack by the epidemic, and thus avoid being destroyed by the disease.

If then the people of infected or threatened districts will only carry out the advice which is given here, and which the Government have gone to great trouble and expense to obtain for them, then the spread of the disease and the great suffering and death-rate which it causes will be greatly diminished; while if the headmen and people of the villages refuse to carry out this advice, then it will be their own fault if they are annihilated by the terrible epidemic which goes by the name of *kála-ázar*.

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GLOSSARY

OF MEDICAL AND SCIENTIFIC TERMS.

Agar-agar.—A glue used for cultivating organisms on—

Albumen.—A nitrogen containing organic constituent of the body.

Albuminoid.—Albumin containing—

Amphistomum hominis.—An uncommon intestinal parasite.

Anæmia.—Deficiency of blood.

Anchylostomum.—A small worm about half inch long, which lives in the small intestine.

Anchylostomiasis.—Anæmia caused by anchylostoma.

Anterior superior spine of the ileum.—A bony process on each side of the lower part of the abdomen.

Aortic cartilage.—Second right costal cartilage.

Arachnoid.—One of the membranes of the brain.

Ascites.—Dropsy of the abdomen.

Aspirating.—Drawing out.

Assimilation.—The act of absorbing nutriment.

Asthenia.—Loss of strength.

Atrophy.—Wasting of a part from lack of nutrition.

Autopsy.—*Post-mortem* examination.

Beri-beri.—A disease with nervous symptoms which is common in the Straits Settlements, but has never been found in Assam.

Buccal.—Pertaining to the cheek.

Cachexia.—A depraved condition of nutrition.

Cancelous.—Resembling lattice work.

Cancrum oris.—Gangrenous ulceration of the mouth.

Cardiac.—Pertaining to the heart.

Cerebellum.—Inferior back part of the brain.

Chlorosis.—A form of anæmia common in young girls.

Coagulability.—Clotting power.

Conjunctiva.—The white mucous membrane of the eye.

Corpuscles.—The solid particles in the blood.

Corpuscular.—Pertaining to the corpuscles.

Crepitations.—A sound heard over a pneumonic lung.

Cyst.—A sac containing fluid.

Disk (optic).—White spot at the back of the retina.

Dochmius duodenalis.—Another name for the anchylostomum.

Duodenum.—The first fixed part of the small intestine.

Emphysematous.—Tissues distended with air.

Endocarditis.—Inflammation of inner lining membrane of heart.

Endemic.—Peculiar to a place.

Epidemic.—Prevailing disease.

Fluke.—A small intestinal parasite.

Gangrene.—Mortification of a soft tissue.

Gartric catarrh.—Inflammation of inner coat of stomach.

Hæmocytometer.—Instrument for counting the corpuscles in a certain quantity of blood.

Hæmoglobinometer.—Instrument for estimating the percentage of hæmoglobin in the blood.

Hæmic murmurs.—Sounds due to alteration in the state of the blood.

Hæmocyte.—A small blood corpuscle.

Hæmoglobin.—Coloring matter of the red corpuscles.

Hepatisation.—Conversion into liver-like substance.

Glossary.

Ilium.—Lower part of small intestine.

Inferior vena cava.—A large vein at the back of the abdomen.

Jejunum.—Upper two-fifths of the small intestine.

Kála-ázar.—Black sickness; the Garo term for malarial cachexia.

Knee-jerk.—A tendon reflex.

Leucocytosis.—Transient increase in the number of white corpuscles in the blood.

Macroscopic.—Visible to the naked eye.

Medulla oblongata.—Enlarged upper end of spinal cord.

Mesenteric.—Pertaining to the peritonial attachment of the small intestine.

Metabolism.—Intimate chemical changes in cells.

Millimetre.—$\frac{1}{25}$ of an inch.

Mucous membrane.—One that secrets a viscid fluid.

Neucleated.—Having a vesicular body in its substance.

Œdema.—Accumulation of clear fluid in a tissue, or dropsy.

Optic commisure.—A part of the under-surface of the brain.

Palpation.—Exploration with the hand.

Pericardium.—A fine membrane lining the abdomen.

Perihepatitis.—Inflammation of the capsule of the liver.

Perisplenitis.—Inflammation of the capsule of the spleen.

Peritoneum.—Fine serous membrane lining the abdomen.

Pernicious anæmia.—xtreme or fatal anæmia.

Pestis, minor.—A mild non-infectious form of plague.

Peyer's patches.—Clustered glands in the small intestine.

Phosphate.—A salt of phosphoric acid.

Pigmentation.—Deposite of pigment in—

Plasmodium malaria.—An ameboid parasite, which is the cause of malarial fevers.

Pleura.—Fine membrane surrounding the lung.

Pleurisy.—Inflammation of the pleura.

Pneumonia.—Inflammation of the lungs.

Pons.—A part of the under-surface of the brain.

Porosis.—A thinning or absorption of bone.

Pulmonary.—Pertaining to the lungs.

Retina.—The internal sensitive membrane of the eye.

Ruga.—A wrinkle or crease.

Secretion.—The product of glandular activity.

Spicule.—A small spike-like piece of bone.

Subcutaneous.—Beneath the skin.

Suprarenal capsule.—A small ductless gland above the kidney.

Systolic.—Pertaining to the contraction of the heart.

Therapeutical.—A science concerned with the application of remedies.

Thoracic duct.—The main lymphatic vessel through which fats are absorbed.

Thyroid gland.—A ductless gland in the neck in front of the wind pipe.

Tricocephalus dispar.—A thread-like worm found in the large intestine.

Valvuli conniventes.—Folds of mucous membrane in the small intestines.

Ventricle.—Small cavity.