"Anchylostomiasis"

(being an enquiry into a prevailing epidemic disease in Assam.)

by

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Anchylostomiasis.

During the last ten years an epidemic disease has ravaged the northern districts of the Province of Assam. In many cases whole villages have been nearly depopulated, and such panic has been caused among the survivors, that they have fled and left their homes entirely abandoned. The disease is known to the natives by the name "Kála-azár".

In the year 1882 the Sanitary Commissioner of Assam drew attention in his "Annual Sanitary Report" to the abnormally large death-rate in the Garo and Goalpara Districts," and pointed out, "that it is largely due to an epidemic disease raging in those parts." During the next three years, it spread widely through the Goalpara district, affecting first those portions near the Garo Hills, and gradually spreading till almost the whole district became dotted with the affected villages. In 1884 the disease became so serious a matter, that Government

organized special measures of medical relief, by starting dispensaries at suitable centres, and employing a number of medical subordinates to travel about and visit the people in their houses. These measures were attended with but little success, owing to the difficulty of inducing the people to submit to European medical treatment, and to their impatience for an immediate cure, even when persuaded to give our methods a trial. They expected to be cured by a single dose of medicine, and seldom could be induced to take a second. The disease being a very chronic one, no method of treatment could be expected to yield appreciable results at once. The treatment, however, was entirely devoted to combatting malaria, and hence, from the point of view of the disease developed in the present study, it is by no means surprising that the efforts put forth were so unsuccessful. Malarial cachexia, always so abundantly met with in Assam, nevertheless, provided many cases capable of material improvement

under the measures adopted; but the spread of Kála-azár was in no way checked by the relief operations. In 1886 the disease appeared in the neighbouring district of Kamrup, and year by year we find the District Surgeons speaking in stronger and stronger language when reporting the ravages caused by the disease in their several localities. In 1888, it appeared in Chaygaon, a place 30 miles west of Gauhati, having taken four years to travel 36 miles. In 1890 it appeared in the town of Gauhati, and many villages around that town were as badly stricken as any of those in the Chaygaon district. It is now to be found well to the eastward of Gauhati, and has invaded the Nowgong district. On the northern bank of the Brahmaputra river it has spread to Mangaldai and Barpeta

It is a noticeable fact that when once the disease has made its appearance in a district, it never leaves it; the weekly returns of the prevalence of epidemic disease showing

it to be present today in every part of the country where it has been hitherto reported.

The progress of the disease is remarkable. It does not appear suddenly, affecting a large area, like influenza and cholera; neither does it spread rapidly from person to person like smallpox, and the other specific fevers; but it creeps slowly and methodically from village to village, so slowly that it has taken seven years to travel from Garo to Gauhati — a distance of less than 100 miles. This shows clearly that its origin cannot be malarial. Assam is, and always has been eminently malarious. It has been suggested that "kala-azar" may be due to waves of "periodic epidemic intensity" (of malaria), but this can hardly be the case, as we never heard of any such periods of intensity before the appearance of this disease. Moreover, once kala-azar makes its appearance, the intensity remains intense, but ceases to be periodic. It is quite

usual also to find villages quite unaffected by the disease, in the midst of villages which have been nearly decimated by it - less than a mile away. If these out-breaks were due to "climatic influence" it is clear that the out-break should attack a large number of people simultaneously, or nearly so, whereas instead of such being the case, we find that the disease always attacks but few of the inhabitants of a village at first, and then spreads with exceptional slowness.

Another fact against the theory that the disease is of malarial origin, is this - that there is no recorded instance of a European becoming infected. Europeans enjoy no such complete immunity from malarial cachexia. They also are exposed to the same climatic influences, and one must therefore conclude, that the source of their immunity is to be found in a difference of habits.

As before remarked, the distribution of the disease is extremely capricious, while the individual

out-breaks are sharply circumscribed; one village being decimated, while another, close by it, remains unaffected. But, wherever the disease does make its appearance, in the course of a few months, large numbers of people are attacked, and the mortality of the place is enormously increased.

The system of Registration of Births & Deaths, is naturally a matter of extreme difficulty in India, and only in a few districts can it be efficiently carried out. The following table prepared in 1884 by the Civil Surgeon, Dhubri gives the mortality in a few affected villages:—

Name of Village	Population	Deaths	Mortality per 1000.
Champasani	219	50	228·3
Reperaghor	61	17	278·6
Thoran Dubi	179	51	284·9
Jira	228	46	201·7
Daman	217	70	322·5
Mamarpara	120	28	233
Khal Mohara	117	26	222
Bhardoki	84	42	500
Dakaidol (Jarpotakpara)	263	53	207.
	1458	389	
Averages:—	148·8	38·3	257.
	165·3		

A remarkable feature, clearly indicated by the above table is, that the villages remarkable for so pestilential a mortality are of small size, and the worst instances occur in very small villages. On the assumption that the disease producing this mortality is communicated from person to person this can be easily understood. Take for instance the village Bhardoki in the above table, where half the entire population of 84 souls died in one year. In the Indian sense of the word "family" the village would consist of but two or three families, and the inhabitants would all be in close and constant association. The high mortality would therefore be inevitable.

The average population of the villages having a mortality of over 200 per thousand in the Gorakpur district, is 148, that of the villages with mortalities between 150 and 200 per thousand is 215, while the average population of the whole number of villages is 443. It seems impossible to explain this fact, save on the

assumption of communicability. For it is perfectly inexplicable that malarial poisoning, or any other non-communicable malady should thus single out the small villages for a disproportionately large mortality, scattered as they are amongst the large ones, over all parts of the affected district. The number of small villages exhibiting exceptionally high rates of mortality is too large to admit of the explanation of its being due to the fallacy of drawing statistical ratios from a small number of data.

The deductions derivable from such statistics as are available all point to the conclusion that, whatever it may be, "Kala-azar" is a disease which affects, intensely, scattered local centres. The distribution of the malady points strongly to the conclusion that it is a communicable disease.

What then is "Kala-azar"? Perhaps the best method of answering the question is to give the notes of a number of cases, as described by

different observers, commenting upon what appears most remarkable in each case.

Case No. 1. Tang, a labourer, age about 30, living at Kalporia in the Garo Hills. Father died when he was a child; mother, a month ago. Is married and has two children, said to be healthy. His village consists of about 16 houses, all inhabited by Garos. A large number of people had died of "kala-azar, in neighbouring villages, but this village was not attacked until a year ago. The disease was believed to have been brought from Kulonbari, which is within sight of Kalpoia. A man called Dang was first attacked, and though ill, he still lives. The man's children have since become affected. Then others fell ill, and about a month ago, Tang began to suffer from diarrhoea and feverish symptoms. Then his spleen became palpably enlarged, and he began to lose flesh.

Present condition is weak, thin, and anaemic. Has slight anasarca, appetite bad, bowels loose. Spleen enlarged to 9 inches below ribs. Pain in abdomen.

The patient remained in the hospital for three months, and in spite of continuous anti-malarial treatment, grew steadily worse, had repeated bowel attacks, and died from an attack of pneumonia.

He is recorded as having "fever"* regularly at some portion of the 24 hours. In spite of this, his temperature, with the exception of one or two slight rises, remained subnormal during the whole of his stay in hospital, often not reaching the normal for a week together. During the last fatal attack of inter-current pneumonia, it rose up to 103°, and remained above normal some days. It had however, become subnormal (95·8°) for some days before his death.

Anti-malarial treatment had a fair trial in this case. Quinine in 16 grain doses, arsenic, iron, iodine were all tried, and persevered in; but the man grew steadily worse instead of better. Considering that the only symptom of malarial poisoning in this case was a moderately enlarged spleen, such as is found in immense

* The word "fever" is used in a very loose way by Native Hospital Assistants, & simply means that the patient seems more uneasy than usual — more restless.

numbers of the natives in India, who are to all appearance enjoying very fair health, this result of the anti-malarial treatment is by no means surprising.

Post-mortem examination twelve hours after death:-

Body emaciated, not darker than usual. Feet oedematous. Section of tissues,- anaemic.

Thorax - left pleural cavity contained fluid of a slightly turbid character; pleura, rough and congested. Posterior part of left lung in a state of grey hepatization. No fluid in right pleural cavity.

Heart - structurally healthy, contains clots.

Abdomen - contains a moderate amount of pale serum.

Liver - of normal size, not congested, of firm consistence.

Spleen - enlarged to three times its normal size, lobulated, section-firm, capsule - thick and corrugated. No infarcts

Kidneys - right - congested, medullary portion slightly granular. Left of similar appearance

Supra-renal capsules appear normal.

Stomach contains thick white mucus adhering to mucous membrane, the surface of which is brown and corrugated, but not ulcerated.
Mesenteric glands of normal size.
Intestines. The duodenum and about two feet of the jejunum were of somewhat slaty colour, which was gradually less marked in the lower parts. Lower down the bowels were considerably thinned with patches of dotted congestion, sometimes extending through Peyer's patches in streaks, but not over the whole surface. Other Peyers patches were quite healthy.

Case No 2 Nagan, a boy cooli from Garo. Age about 15. Gives the usual history of having fever ie and "feverish" for some time. Enlarged spleen, fever, gradual wasting, & inability to work.
Present condition is anaemic, thin and weak, but can get about. Chest-healthy. Bowels-regular. Pulse quick. Tongue pale, appetite fair. Spleen enlarged.
The after-history of the case closely resembles

the preceding one. He was put under a course of anti-malarial treatment, and the spleen diminished in size, but he became steadily worse, and died from a violent attack of diarrhoea, after a stay in hospital of less than three months. A temperature of 101° is recorded once only, and with this single exception there was never any pyrexia during his stay in hospital. The temperature was frequently subnormal Post-Mortem examination showed his death to be immediately due to enteritis. The same peculiar appearances were noticed in the duodenum, and a large number of deposits of pigment of an inky hue, varying in size from a millet seed to a split pea, many of them being quite firm to the touch.

Case No 3. Duchi, a girl aged 12, daughter of a small cultivator in Jura. Was driven away from her village because she had "Kala-azár". Her parents had both died of it. She was found on the road and brought into hospital half-starved.

Present condition — Body thin, feet and face oedematous. Anaemic. Bowels loose. Spleen very large.

The patient rallied at first, but suffered almost continuously from diarrhoea and severe abdominal pain. Her temperature, though not of a malarial type, at one time remained for a few days at 101°. It was probably indicative of a severe attack of enteritis. She lingered on for three months and eventually succumbed during an excessive diarrhoea attack.

Post-mortem examination. Body very much emaciated. Section of tissues anaemic.

A little serum in pericardium and pleura, but thoracic organs otherwise healthy.

Liver, large and somewhat congested.

Spleen, very much enlarged.

Intestines — Duodenum thickened and large. Jejunum has a darkened appearance caused by a remarkably fine dotted pigmented deposit. Not ulcerated. Arborescent patches of congestion in the ileum, but Peyers patches were healthy.

Remarks on the above three cases:- The cases recorded may be taken as fairly representative of a series of twenty five upon which the opportunity of a post-mortem examination was offered. The careful examination made of Peyers patches in each case shows that there was in the mind of the physician some suspicion of a connection with typhoid fever, but in place of the lesions characteristic of that disease, changes were found in the upper part of the gastro-intestinal canal which were quite peculiar, and inexplicable, owing to the non-discovery of the cause or causes of such change.

In the first recorded case we have a typical state of duodenum, and jejunum colored by some coloring of parasites, and in the second case the "deposits of pigment of an "inky hue varying in size from that of a "millet seed to a split pea, many of them "being quite firm to the touch" can be nothing else than the "bites" of a parasite, and are

really nothing else but extravasations of blood into the mucous & submucous tissues forming such bites.

But why were no parasites found in situ? Two reasons are obvious. (1) The preliminary washing necessary for a careful examination of the mucous surface, necessarily washed away the worms, and left only the changes produced by them to be observed. (2) All the dissections were made several hours after death, when the worms would have become detached from the mucous membrane, and so enveloped in the glairy yellow mucus of the duodenum, as to be quite invisible, except to the closest scrutiny. Had a post-mortem been made on a case immediately after death, while the parasite was still fixed to the mucous membrane, it must have been readily observed, and the connection between it & the purulent lesions of the bowel, would have been at once revealed to the dissector.

It having now become practically certain that cases known as "Kala-azar" were really cases more or less due to the ravages of intestinal parasites, particular attention was paid to the evacuations of the patients, and the following cases will show the result

Case No 4. Mohor, an Assamese, age about 32 a native of Nalbari. Complained of diarrhoea, abdominal pain, dyspepsia & general debility. When admitted to hospital he presented every indication of anaemia, weakness and emaciation. Feet pitted on pressure. After the administration of a preliminary saline purgative, the patient was given three 30 grain doses of thymol at 7.0 am. 9.0 am and 11.0 am followed by a second saline purge. At 5.30 pm (i.e. 6½ hours after the last dose), he passed a stool containing 25 ankylostomes. At 6.0 am another stool containing only 2 ascarides At 8.0 pm. 9.0 pm & 10.0 pm more ankylostomes.

Many other cases were treated on the same manner as Case No 4, on some a careful examination of the slides revealed the presence of anchylostomes, on others, no indication of their presence was found. The following case may be taken as typical of many which were only seen when the disease had already nearly done its worst:-

Case No 5. Das, a Hindustani who had lived for some years in the neighbourhood of Gauhati. Age about 30. Much emaciated. Height 5 feet 6 inches, weight 90 lbs. Can scarcely stand, and is too apathetic to give any clear account of his past history.

Face puffy, legs oedematous. No modification of sensory or motor power, apart from extreme weakness. Very anaemic, tongue pale and furred. Skin dry to feel. Area of cardiac dulness somewhat increased, but difficult to define, on account of

being merged below in the dulness extending over the pulmonary area. A weak systolic murmur; marked ascites; but no other abnormality of abdominal organs. He is constipated, but complains of no pain. A saline purgative was given to relieve the bowels, and the stools were found to contain large numbers of ova, but the food was passed in an undigested state.

This man lived only three days after admission. The temperature was persistently subnormal.

Post-mortem examination four hours after death. Emaciated and anaemic but very oedematous.

Chest:- Small quantity of fluid in each pleura. Much fluid in pericardium.

Lungs:- Right 1½ lb. Left 1½ lb. Both extremely oedematous throughout, congested below, very pale above. Ante-mortem clots in the larger branches of the pulmonary arteries.

Heart:- Right side filled with an enormous

ante-mortem clot, which extended into the auricular appendix, and into the pulmonary artery and its branches. Muscular substance thin and pale. Mitral valve and adjacent endocardium thickened. Left ventricle empty.

Abdomen:- Contained a quantity of ascitic fluid. Liver 1 lb 15½ oz pale, but otherwise normal. Gall bladder nearly full.

Spleen:- 5 oz. Tissue of normal appearance, very firm. Kidneys:- each 4 oz. pale but otherwise healthy.

Intestinal canal:- Oesophagus pale but normal. Stomach dilated, pale, with a few reddened patches and petechiae: the walls of the organ are so thin as to be nearly transparent. The bowel was loaded with alvine secretion throughout the upper part, containing mucus streaked with blood. The ascending colon contained soft faecal matter, while the lower bowel was filled with a hard mass. No ova could be

found in the mucus from the upper part of the bowel; they were extremely numerous in the soft faecal matter in the ascending colon, while they were comparatively rare in the hardened mass. The upper end of the small intestine must have contained about a thousand anchylostomes. The first specimens were found adherent immediately below the pylorus, and they were adherent in great numbers to the whole of the duodenum, and upper third of the jejunum. The greatest numbers were to be found in the commencement of the jejunum, where 65 were counted adherent to a piece of bowel eight inches in length; on this piece only 10 more recent bites than adherent worms could be counted. About 3½ feet along the jejunum, only 30 could be counted in a similar length, while at five feet, only un-adherent specimens could be found, and a few such could be found throughout

the greater part of the length of the small intestine. No other entozoa were present.

Although it was impossible to make a very careful clinical study of the disease, there was no difficulty whatever in satisfying oneself of its enormous prevalence. The natives have a strong prejudice against subjecting either themselves or their relatives to European treatment, and it was only as a last resort, after all their "hakims" had failed, and after every sacrifice had been unsuccessfully offered to their gods, that they allowed themselves to be brought to hospital. Even then, if improvement were not noticeable after the first dose of medicine was given, all faith disappeared, and so often the patient also. By making repeated tours through the villages, some half-despondent, others with but a few cases, it was very evident that, although cases of sickness of all sorts were confounded with the prevailing

epidemic by the panic-stricken villagers, by far the greater portion were cases of anchylostomiasis, and that it was this alone that was responsible for the enhanced mortality. One strongly confirmatory fact was that many of the villagers who did not as yet consider themselves as absolutely ill, showed unmistakable symptoms of the disease, and it was easy to pick out such cases from the groups around one. But the diagnosis of the disease can only be made a matter of certainty by the actual discovery of the ova of the parasite in the faeces, and even this test should be carefully repeated before final decision be arrived at. The people could not be persuaded to bring specimens of their dejecta for examination, so specimens had to be collected at random. However, as natives never go very far from the doors of their houses to relieve the calls of nature, there was no difficulty in this respect. Close

by the houses are always to be found a number of small shallow pits, from which mud has been taken for plastering the walls. These pits are usually made use of as latrines, forming miniature but horribly filthy open cesspools within a few feet of the house. There is also much fouling of the ground for the Assamese has a constitutional objection to exertion, and during the rainy season will often foul the ground under the very eaves of his hut, rather than go out into the rain to relieve nature.

By examining specimens haphazardly collected, it was found that in badly-drained villages, three specimens out of every four would contain the ova of the parasite. It was further found that the severity of the outbreak and the proportion of specimens showing ova, were generally proportionate. In these pits, in the soil about the houses (though often

showing no traces of recent defilement), in the puddles in the streets, and in the filthy shallow pools, were repeatedly discovered the free or "rhabditis" phase of the parasite, in all stages of growth.

The more a village is affected with the epidemic, the more easy is it to find evidence of the prevalence of the parasite, - the "Dochmius duodenalis"

The ova, as met with in the faeces, have a very characteristic appearance, & once seen, cannot very well be mistaken for anything else. They measure, on an average $\frac{1}{420}$ inch in length by $\frac{1}{630}$ inch in breadth. They are usually 2-4 segmented. The yelk is of a greyish colour, and is separated from the shell by a comparatively wide zone of clear transparent fluid. The fluid and shell are quite white, and can thus be distinguished from the ova of "Ascaris lumbricoides", which are yellow, and from those of "Trichocephalus dispar" which are deep brown.

The only human parasite with whose eggs it can possibly be confounded is the "Bagnis vermicularis"; but this latter is somewhat smaller, being only $\frac{1}{420}$ inch in length; its outline is unsymmetrical, being flattened on one side only, and, lastly, it always contains a well-advanced embryo. The most advanced Oxyuris ova which can be found in fresh faeces are in the morula stage of development.

Method of Infection. As the parasite under consideration belongs to the species possessing a "free stage", it is clear that the ova themselves are never the infecting agents. Embryos are never found in the faeces, however prolonged constipation may have been. It is the hatched out embryos, and these alone that are in a fit condition to develop into the entozoic stage of the worm. These embryos must gain access to their human host by the mouth, and so must be

introduced either with the food or the drink, or perhaps, in certain instances, by the fingers, when introduced into the mouth for other purposes than eating. Contrary to the usual belief that in the contamination of the water supply, we find the method by which the parasite gains access to the intestinal canal, an analysis of a large number of specimens of drinking water taken from several affected villages, fails almost entirely to give any evidence of the presence of the Dochmius embryo. In the water of the small village pools, however, which the villagers use for bathing & washing purposes, and upon the banks of which they do not hesitate to ease themselves, undoubted Dochmius embryos have been frequently found.

This would account but for a small proportion of cases, for in a number of the most affected villages there is no opportunity for the propagation of the disease in this way.

What then is the usual way in which

the disease is contracted? In what situations are infective embryos to be found in the largest numbers? Not in wells, nor in pools, nor in any collection of water, but in the surface of the soil near the dwellings of patients suffering from, or who have suffered from the disease. Apart from the well known indolent nature of the Assamese who will not take the trouble to go more than a few feet from his house to relieve the wants of nature, it is but natural that a person scarcely able to move from the weakness brought about by anchylostomiasis, and often tormented by diarrhoea, should not trouble himself to tax his strength by going to any distance, especially as his ideas of decency at the best are very rudimentary. No wonder then that a broad circle of ground round the hut of every patient suffering from this disease, is little better than an extensive cultivation ground for the embryos. The

country is essentially a muddy clemate, and only for a very short period in the dry season does the ground become actually hard and dry (even then the morning fogs keep the surface more or less moist). Hence, in entering and going out of his hut, and in his journeys to and fro, the inhabitant of an infected village always carries about with him on his mud bespattered feet, a teeming colony of embryos. Once within a house, it is obvious that there are many ways by which an embryo can obtain access to the intestinal canal of his future host; e.g. food is always eaten whilst squatting on the ground, cleanly eating is impossible under such circumstances. From the position in which natives sit whilst eating, the hands necessarily often come into contact with the feet. Again, in process of time the infected mud which has been introduced into the house becomes house dust. Another native habit

which greatly facilitates infection, is that of cleaning the brass cooking utensils with earth taken up anywhere. Mud is used for so many purposes in the economy of a native household, that it would be impossible to conceive any surer method of favouring infection, than by conforming to the ordinary habits + usages of an Assamese family

The common symptom that characterizes the greater number of cases of Anchylostomiasis is anaemia, and the prominence and importance of this symptom has been noted by all observers. The medical officers of the different districts in which Anchylostomiasis has occured, generally agree as to the general symptoms of the disease. An epitome of these various reports shows that Anchylostomiasis (Kāla-azār) is a disease commencing with pyrexial attacks, ushered in with rigors, and followed by

frequently recurring fits of sweating, which are accompanied by pains in the joints, & headache, resulting in a condition of general anaemia, with splenic & hepatic enlargement. This is followed by extensive anasarca of haemic or renal character, affecting the face, eyelids, abdomen and feet; occasionally by melanaemia, epistaxis, diarrhoea, aphonia, and symptoms of bronchitis or laryngeal catarrh. The disease usually terminates in death from general asthenia. Nearly all these symptoms seem to depend on the anaemia, which shows itself at an early period, and continues to increase in intensity as the disease progresses.

Treatment. The importance of an early diagnosis cannot be overrated. When it was first discovered that "Kala-azar" was actually Ankylostomiasis, one naturally hoped that the expulsion of the parasites would be sufficient to initiate a cure, and

the rest of the treatment would be building up the patients strength and blood-supply, in order to make up for what had been lost in the support of the parasites. This would be right enough, provided the patients retained their powers of assimilation; but further experience of the disease clearly demonstrated that the mere loss of nutritive matter is but a small factor in the causation of the fatal symptoms. The real damage is caused by the destruction of the digestive power. Much of this is caused by the constant recurrence of numbers of the small traumatic lesions of the mucous membrane, — by the bites of the parasites. Add to these the probability of boring + of temporary encystment, and the matter is more easily understood. The gnawing of the parasite produces an erosion, which extends to the submucosa, repair takes place by cicatrisation, not by reproduction of secreting membrane, so that each bite leaves a small area

incapable of ever again performing its natural functions. The total area of permanently injured intestine therefore becomes considerable, especially when we remember that the duodenum and the upper part of the jejunum are one of the most important portions of the intestinal tract. The stomach is also very considerably affected.
Chronic gastritis is followed by imperfect assimilation, and a condition of slow starvation follows, under which the loss of blood, small in itself, rapidly produces the pernicious anaemic condition which we have referred to.

As a somewhat rough guide in the treatment of Anchylostomiasis, it may be useful to divide the course of the disease into three parts & these are well marked in nearly every case. (1) The dyspeptic stage (2) The Anaemic stage (3) The Cachexial stage. Though this division is arbitrary,

it recognises the very essential fact that the dyspepsia always met with is of primary importance and really underlies the anaemia characteristic of the disease. And here we meet our greatest difficulty, for patients afflicted with Ankylostomiasis rarely come under our notice until the second and even the third stage is well entered upon. Dyspepsia is, however, met with all through the disease, and must be met by all the dietetic and therapeutical devices at our command. This is a very different thing in a small badly equipped hospital on the frontier to what it would be in the wards of one of our Glasgow infirmaries. Caste prejudices of the patients themselves are often our greatest obstacles in the treatment of a patient, who will prefer death to the breaking of his caste, by eating food prepared by any other than a man of his particular caste. In the villages, amongst out-patients, treatment by dieting is quite impossible, as no native has

the faintest idea of any special preparation of food for the sick. What is good for the healthy must needs serve for the sick too, and he who cannot eat and digest the native meal of ill-cleaned stewed rice, with a dash of curry spice, must die.

The next thing to do in treatment is "to remove the cause" i.e. the parasites. Microscopical examination of the dejecta will confirm the diagnosis of a case. The difference between the ova of Trichuris dispar and those of other human nematodes has already been pointed out. The best agent we have for expelling the parasites is, without doubt, that excellent anthelmintic Thymol. It must, however, be given in substantial doses, and the most effective method of administration is the giving of it in doses of twenty grains, repeated three times in the twelve hours, with a brisk saline purgative after the last dose. No preliminary purgative should be administered,

but the patient should be kept without food for a few hours before being subjected to Thymol.

The following is a good way of dispensing the drug:-

(a) Stock solution of Thymol.

℞ Thymol 10ʒ
Sp: vini rect 3ij

Dissolve the Thymol in the spirit, and add a sufficiency of Sp: Vini Rectif: to make up to 3iiij. Each drachm of this solution will contain ten grains of Thymol.

(b) Vehicle for administration.

℞ Mucilage (acaciæ) 3ij
Glycerini 3ʃ
Aquæ 3ij

M.

At the time of administration, add one drachm of the stock solution of Thymol to two ounces of the (b) mixture, shake well, and give to the patient.

If the patient has been taken in hand at an early stage of the disease, the dyspeptic troubles overcome, and the parasites thoroughly expelled, generous feeding up will, as a rule, finish the cure, unless, as is too often the case, other complications, chiefly malarial in nature, are in evidence.

Preventive Measures. In the face of such mortality as we have seen in tracing the course of this epidemic, it is but natural that our attention should be directed as much to the prevention of future outbreaks of the disease, as to the treatment and cure of the unfortunate victims of the present scourge. We have shown that the disease is spread entirely by the agency of the dejecta of the patients. From this it follows that there is only one possible way of dealing with the disease, and that is, to no longer allow the general fouling of inhabited sites; viz, in other words, to adopt ordinary

measures of conservancy. We must either do this or let the disease settle itself by the simple method of depopulation. This question of village sanitation is a wide one, and is urgently clamouring for attention in all parts of India, but nowhere, so much as in Assam. This is not the place to do more than merely indicate the simplest methods which in our opinion would do much to stamp out this dreaded disease

(1) The adoption of a simple system of conservancy in every village.
(2) The improvement of the water supply.
(3) The clearing of, and drainage of the land round about each village.
(4) Migration from infected sites, if possible.
(5) Disinfection of infected sites if migration be impossible.

In the preparation of this thesis, the writer has been under great obligation to the

various Medical Officers in charge of the affected districts in Assam; to many native "Hospital Assistants" in charge of Government Dispensaries in Assam; to the Annual Sanitary Reports of the Sanitary Commissioner of Assam; and especially to Lt-Col. G. M. Giles who, (whilst the writer was in charge of the Mowpheang and Jowai Dispensaries and Jaintia Hospital) was specially commissioned by the Government of India to inquire into the epidemic known locally as "Kala-azar."