SEVENTY-THIRD ANNUAL MEETING
OF THE
British Medical Association.

Field at Leicester, July 25th, 26th, 27th, and 28th, 1906.

PROCEEDINGS OF SECTIONS.

SECTION OF TROPICAL DISEASES.

Professor Robert Boyce, F.R.S., President.

PRESIDENT'S INTRODUCTORY REMARKS.

THE YEAR'S PROGRESS IN TROPICAL MEDICINE.

The record of the progress in tropical medical science, if perhaps not so rich in discoveries as last year, shows both a great increase in the amount of scientific observation and the practical good of the application of scientific knowledge to the prevention of disease over an ever-widening area of the world.

At last year's meeting of this Section three remarkable discussions took place: (1) Upon the Prophylaxis of Malaria, (2) upon Trypanosomiasis, and (3), and for the first time, a discussion upon the newly-discovered Leishman-Donovan bodies. During the last twelve months further progress has been due to these three directions in particular. My opinion that trypanosoma fver was probably only an early stage of sleeping sickness. Extended experiments upon the lower animals with trypanosoma gambiensis (Dutton), notably by Thomas and Breini, further confirm the view, and still more recently we learn that some of the natives in the Gambia, in whose blood Dutton originally detected the parasites, have since died of sleeping sickness.

I had indeed looked forward to seeing Dr. Dutton at this meeting, and to his laying before you with his colleague, Dr. Todd, the results of their two years' labour in the Congo. You are all, however, aware of his sad and sudden death. how, although much weakened by an attack of the disease—tick fever—which he was investigating, he pushed on with increased and impatient eagerness, and finally succumbed at his post, after the most careful and benevolent workers who have lost their lives in the cause of that wider medicine which owes its inspiration to the establishment of the Schools of Tropical Medical Research. At last year's discussion Bruce stated that there was nothing to record with regard to the treatment of sleeping sickness. This year Thomas has conducted an exhaustive inquiry into the action of the compounds on the various trypanosomiasis diseases, and he demonstrates the remarkable curative action in the lower animals of an (atoxy) aniline arsenic compound.

It now proceeds to the efficacy of this drug in cases of sleeping sickness in man.

During the year much valuable work has been done in connexion with the new parasite, with the discovery of which the names of Leishman, Marchand, Donovan, and Wright are so honourably connected, by Rogers, Leishman, Donovan, Christopher, Balfour, and others. Further light upon protozoology is furnished by Bentley, followed by James and Fallon, who describe leucocytes in certain mammals, and by Balfour and Christophers, who find haemogregarines in the red cells of certain mammals.

By Dr. J. 1902, Christy—working in Uganda—described and figured a tick harmful to man, which he sent to England. It was identified as Ornithodoros moubata, and has since been proved identical with similar ticks sent from the Congo and India. Christy regarded the symptoms of the tick fever which he described to be due to the result of a primary inoculation with filaria by this tick, known in Uganda as the "bito." Ross and Milne communicated to the medical world shortly afterward the interesting discovery that spirochaetes were present in the blood of cases of tick fever. Previously Marchoux and Salamblin had succeeded in infecting foxes in France. Lastly, Dutton and Todd, working quite independently in another part of the world, confirmed Ross and Milne, and succeeded in infecting the monkey and rat by means of ticks infected by feeding upon dogs coming from the fever in man. They conclude that tick fever is clinically identical with relapsing fever, and that the transmission in the tick is not merely mechanical, but that some developmental process is carried on in the body of the tick. These are but a few examples of the present year's progress in the domain of tropical medicine. In conclusion, there is a large group of workers who have recently returned or are actively engaged in studying tropical disease problems upon the spot, and the result of whose labours we are looking forward to with eagerness.

Medical and Gray have been working in sickness in Uganda, Dutton and Todd have followed both it and tick fever along the water ways and trade routes in the interior of the Congo. Thomas and Breini have left to again undertake the Amazon the zoological research of the tick fever. Renewed energy, under Bruce, has been directed to the elucidation of the manner of infection in Malta fever.

Dr. Annett has just returned for Uruguay the native diseases of animals, and Professor Minchin in Central Africa is bringing his zoological knowledge to the support of the medical investigator. Surely no other departments of medical science can show such activity as this, nor does there exist one that deserves to be more strongly supported. It is indeed most interesting to note that as the result of all these investigations there has sprung up amongst the medical workers a demand for the co-operation of allied sciences. A very large amount of the work in tropical medicine consists in the study of the life-history of parasites and of insect hosts, and the medical investigator constantly finds himself in need of knowledge in these subjects. The help must come from the zoological side, and it is coming; and the dipterologist, helminthologist, or protozoologist are finding more and more that they are coming in for a very large share of attention; and, lastly, the study of ticks and fleas, so neglected in this country, is now, thanks to the medical investigator, being taken up in earnest by the zoological specialist. The discoveries of "red water" have driven the Americans to closely investigate the tick, and to their investigations we owe a great deal of the success of Dr. Bruce in his work on tropical medical laboratory. The study of the mosquito has engaged the attention of numerous dipterologists, and these insects are being worked out in all parts of the globe.

The biting flies—mosquito, tick, and to animals— are coming in for a very large share of attention; and, lastly, the study of ticks and fleas, so neglected in this country, is now, thanks to the medical investigator, being taken up in earnest by the zoological specialist. The discoveries of "red water" have driven the Americans to closely investigate the tick, and to their investigations we owe a great deal of the success of Dr. Bruce in his work on tropical medical laboratory. The study of the mosquito has engaged the attention of numerous dipterologists, and these insects are being worked out in all parts of the globe.

The section of zoology to be vivified by tropical medicine is helminthology. Medicine owes a great debt to active zoologists like Looss of Cairo, and Shipley of this country, but we do not give them sufficient encouragement. We require their cooperation, for there are vast fields which wait their investigations. To my mind, Dr. Bastian was one of the very first in this country to emphasize the importance of helminthology to medicine, and I hope after all these years he will see his wishes realized. If the zoological specialist is necessary to the medical investigator in the fields of dipteroLOGY and helminthology, he is equally indispensable in protozoology.

The necessity for exact knowledge in this branch of science goes without saying—everything depends upon it. The zoologist trained to study-life history in minute forms of animal and vegetable life is better equipped than the medical investigator. The two sets of workers require to co-operate, and above all to work side by side in the laboratory. I do not think that either group yield their best when working in opposition to another; for the maximum amount of good to be obtained they should work in close co-operation in the laboratory. Far too much departmental isolation appears to me, and no true advance existed in the past in the higher branches of scientific training.

The advantage of co-operation with allied sciences is again seen in another direction, and in a field of supreme interest to the medical scientist. It is well known that the greatest medical observations of all are being made by men trained in botany and zoology.

Dr. Dutton has recently acknowledged our indebtedness to these investigators, and we have gladly invited their co-operation to guide us, and we look forward with confidence to uniting cytology to medical science in the same way as we have united pathologic zoology.

Few will have failed to observe a further most interesting outcome of the tropical medical movement. I refer to the
remarkable activity of the Indian Medical Service, the Royal Army Medical Service, and the Colonial Medical Service. At no time do these services appear to me to have been so mentally alert or so active as they have been in recent years. The mosquito has been given to prosecute researches into tropical diseases, and now that the role of the mosquito and the biting insect in the transmission of disease is fully accepted—thanks in part to the discovery of Ross—the medical officers of these services have found abundant material and occasion both to make observations and to engage in practical antimalarial measures. A new, better-trained generation of officers has been given the opportunity in these wars, and has been abundantly proved by the sanitary reports which reach us, they certainly see the success of their prophylactic measures. This increased scientific activity is not, however, confined to these services; it is also seen both amongst the young doctors on the tropical coaling stations and amongst those in charge of mines and industrial concerns in the tropics.

At last years’ meetings of the Royal Society the sense of the need of a better prophylaxis was most enthusiastic and full of practical bearings. This year it has been decided not to hold one—not that there is not abundant material, but for the reason that the case has been so abundantly proved that the necessity for discussion is not urgent.

In all parts of the world practical effect is being given to the newer methods of prophylaxis. In the Federated Malay States there has been published by Dr. Malcolm Watson a most encouraging report of the success of antimalarial measures at Klang and Port Swettenham. Dr. Watson shows that in these towns, whilst the number of cases of malaria has been reduced, the disease has not taken place in the districts around, where no antimalarial operations were undertaken. He further points out that the results are not only taken place in malaria, but also in the other diseases due in many instances to the local state of health consequent on malaria. He finally claims, and with justice, "for the Government of Selangor the honour of being the first British Government to follow" Dr. Ross in an attempt to be on those entrusted to its care the benefits of his great discovery."

From Hong Kong we have recently received the report of Dr. A. F. Kimble for 1904. He shows, as the result of antimalarial measures, the following marked decrease in malaria cases, based on the admissions into the three hospitals. The total admissions in 1901 were 4,593; in 1902, 816; in 1903, 644; and 1904, 490. In Ismailia and Hana the results are well known. From Italy, Algeria, Mexico, Brazil, and from many of our own Colonial possessions, most satisfactory reports are being constantly received.

A most striking feature of the reports of many of the South American towns is the remarkable decrease of yellow fever as the result of organized campaigns against the <i>Aedes aegypti</i>. In many towns the bite of greatly improved municipal yellow fever still made headway, and there were, in consequence, numerous critics found to draw attention to the necessity of spending money on sanitary reforms. With the discovery of the true cause of yellow fever, criticism was soon disarmed by the remarkable reduction in the cases of this disease brought about by getting rid of the mosquito.

Although these results are most encouraging and are increasing in number, yet, after all, they form only a small beginning, and there yet remains an immense amount to be accomplished and a vast sea of prejudice and apathy to be got over. At the discussion last year, Dr. Stephens stated that he did not believe that one in a hundred laymen in the tropics knew that malaria was a contagious disease, or suspected the source of infection to reside in the natives around him. That is true in a great measure to-day. My experience is, for the most part, amongst those living in and trading with the West Coast, and, undoubtedly, the most striking improvement has been that improvement in the health and in the position in the Gambia, Sierra Leone, and Lagos, there still exists a high percentage mortality; the young and the old trader and the miner is still indifferent, antimalarial measures are not pushed on with, and still the sanitation of some of our oldest coast towns is a blot on our administration. But, after all, it gives little satisfaction to any one to complain. The problem is now being grappled with, and we have a large number of the sanitary officers of the coast, especially trained, increased regard is being paid to the quarters of the white man, the use of the mosquito net is being pushed on, and whilst native supplies are coming into practice, and a greatly-increased number of investigations are at home and in the tropics are focussing attention on the tropics.

India has done great strides, and we have learnt from that country how it has done it and what to avoid. We can, therefore, make use of the West Coast of much of India's experience, and the one thing that appears clear is the necessity for a more organized system of sanitary control for the separate colonies, to supervise and organize the mortality and sick rates, to make sure that antimalarial measures and general sanitary reforms—a system which would enable the Colonial Office to remain in touch with the sanitary work which is being accomplished locally—to publish accurate data, and to afford local governments a skilled referee in sanitary matters.

**THE NATURE OF TICK FEVER IN THE EASTERN PART OF THE CONGO FREE STATE,**

WITH NOTES ON THE DISTRIBUTION AND BIONOMICS OF THE TICK.

BY

J. Everett Dutton, and

John L. Todd,

M.B. Vetr.,

Late Walter Myers Fellow of the Liverpool School of Tropical Medicine.

**Abstract.**

The paper was read by Major John; Ross, F.R.S.

On November 26th, 1904, Dutton and Todd announced that they had discovered that a spiroplum was the specific agent in the causation of human tick fever in the Congo Free State. The disease was proved to be identical with that caused by monkeys with the disease by allowing monkeys to be bitten by ticks. In the British Medical Journal of November 26th, 1904, a note by Drs. Ross and Milne on tick fever made the same announcement, and Drs. Dutton and Todd are very cautious in the claim of priority in the discovery in favour of Ross and Milne. During their travels up the Congo to Kasongo, Dutton and Todd found the horse ticks of <i>Oxynothus maculata</i>—a veritable pest, and saw cases of tick fever caused by its bites. Amongst the natives of the district the tick had an evil reputation, and to its bite severe illness was ascribed. From their investigations on the spot they came to the conclusion that tick fever is a relapsing fever produced by a spiroplum identical in all probability with the spirochaeta ombremleri, and that this organism can be transmitted by the bite of the <i>Oxynothus maculatus</i>. An interesting experiment, although only one is recorded, showed that the spiroplum can be transmitted by the bites of young ticks newly hatched in the laboratory from eggs laid by infected ticks. The period of the incubation of the disease does not exceed one week.

**Signs and Symptoms.**

Tick fever declares itself by frontal headache, bone ache, backache, vomiting, and distaste for food are the usual symptoms present at the commencement of the illness. Diarrhoea is the rule, but constipation may occur. The temperature during an attack is high, such as 104.5° F., or even to 105.5° F. Febrile attack lasts three or four days, and usually ends in profuse perspiration. As a rule there are three or four febrile attacks with intervals of as many as five to nineteen days between each. The spleen may be found enlarged; and other complications recorded are herpes, epistaxis, and hicough. By far the most pronounced feature of the ailment is the prostration and the terrible feeling of depression of the patient during the febrile attacks; whilst equally marked is the sudden return to feeling well when the temperature falls.

The mortality, except under circumstances of great fatigue and exposure, is not great; that, however, under adverse conditions it can be serious is shown by the fact that in one caravan journeying from Kasongo to Kumbambare out of twenty carriers no less than ten died.

**The Spirillum.**

The spirillum is readily stained by the Romanowsky method or by one of its several modifications, but the staining process varies greatly in the length of time it takes to prove effective, and uneven staining is the rule. Of the numerous spirilla of the size measured by the sanitarial officer the longest spirillum measured 43 μ in length, the shortest 13 μ; the average length of the longest was 35 μ, and of the 10 shortest 16 μ. The spirilla group themselves at times into twos, threes, or fours, and what appear to be division forms, inasmuch as they presented a Y-shaped outline, were encountered.
Experiments on animals showed that for monkeys (Cercocebus) alone was the spirillum uniformly pathogenic. Rats, rabbits, and guinea-pigs were tried, but in none of the inoculated animals were signs or symptoms of constitutional disturbance. A large guinea-pig after a large dose of infected blood showed a slight rise of temperature, but in two weeks it died. In the blood of three rats infected the spirilla were believed to have increased for a time, and division forms of the parasite were seen.

Distribution and Biomecs of the Tick.

In the Congo Free State, where Dutton and Todd made their observations, the tick which attacks human beings, the Ornithodoros moubata, is met with almost solely along the main roads and especially at the entrances to the villages even where the roads do not meet with. In the rest houses, which are placed at convenient distances along these roads for ling such a matter did not drop off until before two and three hours—so that it is unlikely a human being would tolerate the presence of a tick for more than a few seconds during waking hours, thus preventing the tick obtaining a meal. Dutton and Todd believed the tick attacks large dimensions, and increasing in bulk to many times its size when empty. A full-grown female filled with eggs and blood may measure as much as 12 by 7 mm. Whilst feeding there passes from the anus the whitish secretion or liquid that the tick has engorged; the female being more or less distended, but not engorged. The amount of eggs laid at one time vary greatly. The number of eggs observed when laid at one time was 139. The eggs mature slowly with the body of the mother, and 70 eggs only were observed to be laid during three days. When deposited, the eggs adhere to one another in groups, and under a low power of the microscope resemble bunches of grapes, and are of a glistening golden-brown hue. In about twenty days the eggs hatch. The following observations were made: The larva of the Ornithodoros moubata is inactive, a new scientific fact; the larva is hexaped in shape; in about seven days the larva can be seen forming within the translucent egg-shell in the nineteenth day the egg-shell splits along the posterior sagittal plane, as a rule; the larval skin splits at the junction of the dorsal and ventral surfaces. The eight-legged nymph, when it emerges, casts off both the egg-shell and larval envelope; as a rule, the nymphs do not feed for three or four days after leaving their shells.

Ticks would seem to retain their power of infecting for some considerable time; ticks caught before November 23rd at the Royal Naval Hospital, Haslar, and kept in a moist chamber capable of holding monkeys with spirilla from fifty to fifty-five days later.

Sir John Kirk states that after tick fever the patient was very nervous and became prima, so that the freedom of adults from the disease might be accounted for by the fact that they had tick fever in their youth. The natives when tick capture as many ticks as they can, burn them, and rub the ashes into their bodies. It is possible that they may be made to think of the bite with the idea of preventing tick fever. This is a natural enemy of this tick, and ants attack the eggs and the newly-hatched ticks with avidity.

Dr. Louis W. Samson said Dutton and Todd had established the presence of relapsing fever in the Congo Free State, and had shown that the disease was propagated in that part of Africa by Ornithodoros moubata, a widely infected bug, belonging to the subfamily of the Argyasinae, and presenting life-habits very similar to those of the common bed-bug. They had, moreover, suggested that the disease was transmitted by Dr. Livingstone and Sir John Kirk in the Zambezi valley, and attributed to the natives of a tick (Ornithodoros moubata), might be nothing more or less than relapsing fever. Certainly, suggested by Dr. Livingstone was probably correct, but no one could have guessed it solely from the scanty description of the disease given first by Livingstone and Kirk, and more recently by Dowse and Bentley in the Lower Congo. The Ornithodoros moubata seems to have entered the Congo Free State from the east, being carried from the east coast by Arabs, and from the south by way of the Portuguese territory. Livingstone found these bugs a pest in his time, and states that before the Arabs came bugs were unknown in the regions; and, furthermore, one may know if a man's walk from off the principal roads the tick is not met with. In the rest houses, which are placed at convenient distances along these roads for ling such a matter did not drop off until before two and three hours—so that it is unlikely a human being would tolerate the presence of a tick for more than a few seconds during waking hours, thus preventing the tick obtaining a meal. Dutton and Todd believed the tick attacks large dimensions, and increasing in bulk to many times its size when empty. A full-grown female filled with eggs and blood may measure as much as 12 by 7 mm. Whilst feeding there passes from the anus the whitish secretion or liquid that the tick has engorged; the female being more or less distended, but not engorged. The amount of eggs observed when laid at one time vary greatly. The number of eggs observed when laid at one time was 139. The eggs mature slowly with the body of the mother, and 70 eggs only were observed to be laid during three days. When deposited, the eggs adhere to one another in groups, and under a low power of the microscope resemble bunches of grapes, and are of a glistening golden-brown hue. In about twenty days the eggs hatch. The following observations were made: The larva of the Ornithodoros moubata is inactive, a new scientific fact; the larva is hexaped in shape; in about seven days the larva can be seen forming within the translucent egg-shell in the nineteenth day the egg-shell splits along the posterior sagittal plane, as a rule; the larval skin splits at the junction of the dorsal and ventral surfaces. The eight-legged nymph, when it emerges, casts off both the egg-shell and larval envelope; as a rule, the nymphs do not feed for three or four days after leaving their shells.

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this kind, giving one the opportunity of investigating the characters of the blood as taken from the peripheral circulation and also by means of splenic puncture.

In both the cases there was a history of being abroad late, with excessive and peculiar anaemia and great splenic enlargement. In both splenic puncture gave negative results for the particular bodies sought for, and in both cases cultures made from the splenic blood showed it to be quite sterile. In neither was there any history or evidence of malaria. Unfortunately, owing to the chronic character of the disease and the rules of the service it was impossible to keep them permanently under treatment and observation, both having been invalided and lost sight of.

**CASE I.**

J. W., aged 21, stoker. 
**History.—**While out on the South African station he landed at Zanzibar, never slept on shore, and never had fever. On July 16th he vomited some blood, and had several similar attacks during the last eight months, with loss of appetite and occasional pyrexia; on the passage home he once vomited up coffee-ground fluid. On admission to Haslar he was an extremely anaemic though not emaciated man, complaining of breathlessness on exertion, pain and weight in left loin and side, and slight evening fever. On examination his temperature was normal; pulse 88, regular; heart's impulse diffused with a soft systolic bruit at the apex; liver normal; spleen extending across middle line and down to the iliac crest, edge sharp and hard, not easily felt, some tenderness on deep pressure; urine 1020, acid, no albumen.

He was under treatment for three months, during which time there were two slight attacks of pyrexia, regular slight evening pyrexia, occasionally up to 101°F. He maintained his weight, and his anaemia appeared to decrease in severity, but there was no decrease in the size of the spleen; there was also a marked tendency to the formation of boils.

**Treatment.—**Iron, arsenic, injection of pot. iod. high-frequency currents, and generous diet.

The following is an account of the blood changes observed:

<table>
<thead>
<tr>
<th>Date</th>
<th>Red Cells</th>
<th>White Cells</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 16th</td>
<td>2,050,000</td>
<td>1,650</td>
<td>Invalided from Africa.</td>
</tr>
<tr>
<td>Aug. 1st</td>
<td>2,592,000</td>
<td>2,400</td>
<td></td>
</tr>
<tr>
<td>Sept. 1st</td>
<td>3,040,000</td>
<td>2,902</td>
<td></td>
</tr>
<tr>
<td>Sept. 16th</td>
<td>2,780,000</td>
<td>3,303</td>
<td>Red cells irregular and very pale.</td>
</tr>
<tr>
<td>Nov. 1st</td>
<td>2,880,000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dec. 15th</td>
<td>2,400,000</td>
<td>2,800</td>
<td>Haemoglobin, 49 per cent.</td>
</tr>
<tr>
<td>Dec. 22nd</td>
<td>3,500,000</td>
<td>3,700</td>
<td></td>
</tr>
<tr>
<td>Feb. 2nd</td>
<td>2,400,000</td>
<td>2,803</td>
<td></td>
</tr>
<tr>
<td>Feb. 3rd</td>
<td>2,580,000</td>
<td>3,200</td>
<td></td>
</tr>
<tr>
<td>Feb. 7th</td>
<td>2,520,000</td>
<td>2,900</td>
<td></td>
</tr>
</tbody>
</table>

**Relative Counts.**

<table>
<thead>
<tr>
<th>Date</th>
<th>Neutrophiles</th>
<th>Small Lymphocytes</th>
<th>Mononuclears</th>
<th>Eosinophiles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan. 17th</td>
<td>74</td>
<td>28</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Feb. 3rd</td>
<td>60</td>
<td>34</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Feb. 7th</td>
<td>75</td>
<td>40</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

On two occasions the spleen was punctured and 3 c.cm. of blood drawn off, free from splenic pulp. After both he experienced for two days an increase of splenic pain, but there was no sign of haemorrhage. A specimen was sent to Lieut-Col. Leishman, who kindly examined it, failing to find, as I did, any evidence of either Leishman bodies or other blood parasites. This blood did not markedly differ from that taken from the fingers, except for the rather larger relative number of small lymphocytes.

**Spleenic Blood.**

<table>
<thead>
<tr>
<th>First puncture</th>
<th>Neutrophiles</th>
<th>Small Lymphocytes</th>
<th>Mononuclears</th>
<th>Eosinophiles</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>47</td>
<td>40</td>
<td>5</td>
<td>4</td>
</tr>
</tbody>
</table>

The citrated blood was also incubated at blood heat, with negative results for these bodies, and was also cultured on agar, proving it to be quite sterile. An examination of the blood from the circumference of one of the bolls which formed, did not show any increase in the number of leucocytes, though one expected that with a local suppuration the phagocytes might have been considerably increased, which was not the case, showing an absence of reactive power to local stimuli.

**NOTES ON TWO CASES OF FEBRILE TROPICAL SPLENOMEGALY (KALA-AZAR) AND A SUGGESTION.**

By Sir Patrick Manson, K.C.M.G., M.D., F.R.S.

The following are details of two cases of kala-azar:

**CASE I.**

The patient, Mr. M., had resided for nineteen years on the borders of Assam, and was exempted from the commission of a mild continued fever some three months ago enjoyed good health. He came home on furlough during 1904, returning to Eastern Bengal in July. About a week or ten days after his return his present illnesses began with anorexia, malaise, and fever. As he did not improve he went to Calcutta, and remained in hospital there till August 14th. Under large doses of quinine (60 gr. daily) he
apparently recovered and went to Ceylon for a month. On his return upper respiratory trouble was a recurrence of the fever, accompanied by nausea, irregular rigors, and prostrating night sweats; an erythematous patch developed extremely feverish and died after nine months' illness.

One of his native servants died in May, 1903, with bleeding of the gums after a three months' illness, and another suffering from enlarged spleen and constant fever died after nine months' illness. When he joined the ship at Calcutta, Dr. Webster, under whose care he was, reports that the liver was enlarged downwards 2 inches below the costal margin in nipple line, uniformly resistant, but unaccompanied by pain or tenderness. The spleen extended forwards as far as the nipple line. On the voyage home the temperature was remittent in character, 96 to 96° F. in the evening; the hepatic and splenic enlargements both diminished. Treatment consisted in regulated diet and the intramuscular injection of arsenic.

He was admitted to the Seamen's Hospital, Albert Docks, on December 15th, his condition being briefly as follows: Well nourished but weak, skin muddy, spleen enlarged—extends a 1/2 in. below costal margin—firm, but not tender. Liver enlarged, extends 1½ in. below costal margin in nipple line, smooth, slightly tender. Temperature, 101° F.; pulse, 88; respiration, 24. Erythrocytes, 3,600,000; W. K., 1,000; haemoglobin, 60 per cent.; L. 39 per cent.; E. M., 11 per cent.; P. N., 45.5 per cent.; T. 3.5 per cent. E. 9 per cent.

The history of irregular fever with splenic and hepatic enlargement, progressive anaemia, and the locality in which the disease was acquired, were suggestive of tropic spilomengitis. This diagnosis was confirmed by finding Leishman bodies in spleen and liver juice obtained by puncture, which was put on increasing doses of arsenic and on January 27th his condition was as follows: Weight, 11 ft. 2½ lb. A gain of 1 lb. Erythrocytes, 3,410,000; W. K., 6,300; haemoglobin, 63 per cent.; L. 57 per cent.; E. M., 27 per cent.; P. N., 57 per cent.; E. 11 per cent.; T. 1.5 per cent.; E. R. 7 per cent. M. C. 1 per cent. General condition slightly improved; occasional epistaxis; temperature slightly above normal at night; liver and spleen unchanged.

From January 27th to February 5th the arsenic was discontinued and treatment by X-rays tried, but without benefit; in fact, he lost weight.

On March 3rd the administration of arsenic and trypanoth hypo-dermically was commenced, and continued until May 3rd, but without apparent benefit; bone marrow was then substituted.

On May 5th examination of blood was: Erythrocytes 3,000,000, W. 3,000, Hb. 40 per cent.; there had been slight haemorrhage into right retina, and there was occasional oozing from the gums.

He left Calcutta for Bombay on May 25th, and on his return from a four days' shooting expedition to Jafargudah, Nagpur; it began with fever (temperature up to 106° F.), enlargement of liver and spleen. He was accompanied on the expedition by his father and a younger brother; the party shot during the day and slept in a railway carriage in a siding at night. Both father and brother had attacks of fever four days after returning to Calcutta (November 28th); in their cases there was also enlargement of liver and spleen, and the temperature reached 106°, but in them the fever subsided in about sixteen days, and there was no recurrence. Though the patient improved and was able to be about on Christmas Day, the temperature in his case never fell below 100°.

He took to bed on January 4th, and has been ailing since, in spite of liberal dosing by quinine. Owing to the persistence of the fever (varying between 100° and 104° F. in evening), and a descent of the spleen, he was sent home in the end of February. He picked up a little on the way, but caught cold in the Mediterranean. Whether the illness was cured or not with the subject of this paper, it is interesting to have to record that another brother suddenly, while on board ship on the voyage to England, developed symptoms of what was diagnosed as cerebral meningitis, and died at Marseille.

The patient was seen by Dr. T. S. Kerr, to whom I am indebted for the notes of this case and Illustrations, on April 4th, and the following day by myself. His condition was then briefly as follows: Anaemia, clean tongue, spleen enlarged, exhibiting marked similitude to all euchromatoid lines both forwards and downwards, liver dullness about 6 in. no irregularities on palpation and no tenderness; a small amount of fluid in pericardium; bronchopneumonia; anaemia, and emaciation; yet, withal, a clean tongue and good appetite. The termination, too, by some intercurrent affection—in this case bronchopneumonia—is usual in such cases.

Both cases illustrate the value of spilomengitic puncture as a diagnostic test, though unfortunately it is a proceeding not devoid of danger, as shown by Donovan's statistics recently published in the Madras Hospital Reports—3 deaths are reported from this region, as compared with seven from the central parts of the Congo. Cases of spilomengitis are usually fatal, death being followed by a long interval between diagnosis and death, as seen in our case, and in that of Dr. Kerr. The remarkable feature of this case is the rapidity of onset of the disease, which was so severe that it was not possible to make a blood count until five days after the appearance of the rash, and this was unsatisfactory, the white corpuscles being but 4,000,000, and the erythrocytes only 2,800,000; the hemoglobin was only 55 per cent., and the temperature 104° F.

Reports—3 cases of Leishmania bodies, large splenic cells, also free in the plasma in small numbers (a); in some their appearance was not quite like those usually met with. The nucleus had become diffused (b), in others it was partially dislocated and the bodies themselves were larger, swollen in some instances and vacuolated.

In addition to the Leishman bodies and in fair numbers—some beneath the corpuscles, but mostly lying free amongst them, for the most part singly (c), but also in chains of 2, 3, 5, 4, 5 (f) elements—were to be found oval, in some cases pyriform, bodies. Some were homogeneous and without a nucleus; others had a well-defined round nucleus, appearing as a round or oval ring with a clear centre. In addition to the definite nucleus, many possessed a diffused though well-stained spot at the narrow end, whilst in a very few there was a distinct rod-shaped micrornucleus (g). The figures in the reports show these bodies in blood films from the spleen and liver, but none in those from the lung.

Remarks.

These cases illustrate one or two points in connexion with the inoculation, diagnosis, course, and treatment of this disease. In Case I the patient, after a four months' holiday in Europe, returned to India in robust health. Within ten days or a week after his arrival in Eastern Bengal he developed a fever, which was accompanied by enlargement of the spleen, from which, with but few intermissions, he continued to suffer. The fever was uninfused by quinine; emaciation and anaemia were slowly progressive. The incubation period, therefore, in this case can be fairly accurately determined, and lies somewhere between the period of his return to the endemic area and the onset of fever—a period of almost ten days.

Case II, in its clinical features, is typical of the disease—namely, a chronic fever, never very high, uninfused by quinine; enlarged liver and spleen; progressive anaemia and emaciation; yet, withal, a clean tongue and good appetite. The termination, too, by some intercurrent affection—in this case bronchopneumonia— is usual in such cases.

Since the foregoing was written I have heard of this patient's death.
in 170. Possibly liver puncture would be less dangerous, and for diagnostic purposes equally reliable.

Both cases—"more especially Case 1—illustrate the beneficial effect of comparable injection treatments administered to overcome the anemia, and in retarding, if not actually arresting, for a time the downward course of the disease.

The nature of the bodies found in the slides taken after death is not the subject of this paper; these are but mere conjecture; their occurrence relatively proportional to and with the Leishman bodies is suggestive of a hitherto unrecognized phase—if it be a phase—of the Leishman body. On the other hand, they may be merely spontaneous contaminations of the slides, though every precaution was taken to avoid this.

The life-history of the Leishman body has not yet been satisfactorily worked out, and in illustrating and describing these bodies I would only suggest—and by no means assert—the possibility that these bodies may in some way or another belong to it.

Just as in the Leishman body in culture, they vary in size considerably, the smallest single body measuring 23 mm., whereas the largest measured 5 mm. In one specimen (g) there was a definite nucleus, and an equally definite rod-like micronucleus, and fro its periphery there appeared an oval non-nucleated body in appearance identical with many similar but isolated bodies found in the plasma.

The identity of the Leishman body and that found in Oriental sore is an interesting one, possibly having an important practical bearing. In tropical epidemicso the Leishman body is found in the spleen, liver, lymph, gland, intestinal glands, etc.; in Oriental sore it occurs only in the sore. In the former case there appears to be a general infection, and certainly profound constitutional disturbance; in the latter the infection is strictly localized, and there is no constitutional disturbance. But although there is no clinical evidence of constitutional disturbance in the case of Oriental sore there certainly must be some constitutional recognition of its presence; it is well known that the inoculated disease is protective. This fact has been acted on for generations by the Jews of Bagdad, who anticipate the natural development of the disease—it might be an innocent part of the face, where it would produce unsightliness or deformity—by the practice of inoculation on some less inconvenient region.

Assuming that the Leishman body of kala-azar and the corresponding germ of Oriental sore, which are morphologically identical, are also specifically identical, and that whereas the former is malignant in its course and the latter benign, it follows that the malignity of the kala-azar germ has been got rid of in some way or other. It has long been believed that Oriental sore is a disease of countries in which the camel is a prominent domestic animal, and it is maintained by some that the disease may be caught from the camel.

These facts suggest to my mind that Oriental sore may bear the same relation to kala-azar that vaccinia does to small-pox, and that the virulence of the kala-azar germ has been got rid of during its passage through the camel, just as the small-pox germ is deprived of its virulence by passage through the cow. This is conjecture, but seems to be a point worth working at, for should the conjecture prove to be correct, we may have ready to hand a means of preventing kala-azar.

GUINEA-WORM AND ITS HOSTS.

By W. M. Graham, M.B., B.Ch., B.A.O., B.A.,
Medical Officer, West African Medical Service.

In 1904, while studying the incidence of disease at Gambaga, in the Hinterland of the Gold Coast Colony, I was struck by the comparatively low number of guinea-worm cases as a cause of disease; and a collation of the year's statistics gave the following results: In a force of native troops, averaging 500 monthly, which had been under observation during the year, 57 men, or 16 per cent., had suffered from the disease, and they had been incapacitated for duty for an aggregate of 1,304 days, or for, an average of 22.8 days each. These figures do not, however, represent the whole loss of the men's time, for a number of them, when discharged from hospital were only fit for light duty, and attended hospital daily to have their ulcers dressed.

These figures, however, that guinea-worm was the most prevalent disease and cause of incapacity for duty among the native force employed by the Government.

My attention having been thus directed, I tabulated my results, intending to include them in my annual report on the Northern Territories; but while endeavouring to reduce the statistics to a few succinct generalizations, so many points of difficulty appeared, and so many questions demanding answers, that I abandoned my intention and began a fresh inquiry.

The tabulation of statistics of guinea-worm for 1904 had revealed the following facts demanding explanation: 1. That there had been one month in the year that had been the month of greatest incidence. 2. That from this month backwards and forwards incidence had declined to zero.

Table showing the Monthly Number of Guinea-Worm Manifestations among the Native Troops at Gambaga, Northern Territories.

<table>
<thead>
<tr>
<th>Month</th>
<th>1904. Strength Average</th>
<th>1905. Strength Average</th>
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</thead>
<tbody>
<tr>
<td>January</td>
<td>sick: 4</td>
<td>sick: 4</td>
</tr>
<tr>
<td>February</td>
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<td>sick: 1</td>
</tr>
<tr>
<td>March</td>
<td>sick: 4</td>
<td>sick: 2</td>
</tr>
<tr>
<td>April</td>
<td>sick: 4</td>
<td>sick: 5</td>
</tr>
<tr>
<td>May</td>
<td>sick: 4</td>
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</tr>
<tr>
<td>June</td>
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<td>sick: 4</td>
</tr>
<tr>
<td>July</td>
<td>sick: 2</td>
<td>sick: 6</td>
</tr>
<tr>
<td>August</td>
<td>sick: 3</td>
<td>sick: 1</td>
</tr>
<tr>
<td>September</td>
<td>sick: 3</td>
<td>sick: 1</td>
</tr>
<tr>
<td>October</td>
<td>sick: 3</td>
<td>sick: 2</td>
</tr>
<tr>
<td>November</td>
<td>sick: 3</td>
<td>sick: 4</td>
</tr>
<tr>
<td>December</td>
<td>sick: 3</td>
<td>sick: 1</td>
</tr>
</tbody>
</table>

REMARKS.—The figures in this column (sick) represent the number of men primarily manifesting guinea-worm during the month, and not the number of guinea-worm cases under treatment. As the treatment of guinea-worm may extend over two or three months the cases under treatment in any month would be in excess of the number showing symptoms for the first time during the month. In the case of July, 1905, the numbers given are those of men under treatment, for as the month of June, 1905, were not available, I was unable to estimate from July the cases beginning in the previous month.

A further tabulation of the existing statistics of previous years proved that this generalization was true, not for the year 1904 only, but also for the three previous years, beyond which unfortunately no statistics were available.

I have arranged the figures in a table to show the monthly incidence for 1901, 1902, 1903, 1904. They are, I regret to say, incomplete, but they conclusively support my generalization by showing that in all four years the month of June was the month of greatest incidence.

Fig. 1.—Diagrammatic representation of the three wells at Gambaga.

Now the guinea-worm, requiring for its propagation the alternate services of two hosts, man and cyclops, the induction appeared to me legitimate that the month of maximum manifestation in man is the month of maximum infection of cyclops. It will be evident from the table that this month at Gambaga is the month of June.

It has been shown by Fedchenko that in Turkestan guinea-worm embryos require five weeks for complete development in cyclops. Embryos, therefore, beginning this development in June would become capable of infecting man in July or
August of the same year. Further, as the parasite acquired by man in July or August would become manifest by discharging embryos in the following May or June, it would appear a legitimate inference that ten months represent roughly the period required by guinea-worm for its complete development in man.

To this hypothesis the following statements appear to me necessary corollaries:

1. That the infection of man by guinea-worm takes place during a few months of the year.
2. That it does not occur during the other months.

To prove the truth of this corollary, it would be necessary to show that at Gambaga cyclops contained the fully-developed parasite in the months of July and August, and not at other periods of the year. I am unable to furnish such evidence, for I left Gambaga, on leave, at the beginning of May, and I have only the negative evidence to offer that during the months of December, January, February, March, and April, arrival at the surface of a lower extremity and by the extrusion of her embryos.

What, then, becomes of the unsuccessful travellers? I believe some die and others are delayed and arrive late at their destination, and that it is these latter which give rise to those belated manifestations that seem difficult to reconcile with my hypothesis. Thus by my failure to procure living embryos during the first three months of the year, was raised the important question: During what months of the year do female guinea-worms extrude living embryos, and this question awaits investigation. If, then, the infection of the cyclops with guinea-worm occurs during the months of May and June it becomes worth while to inquire whether during this period any natural facilities are afforded to the process of infection which are subsequently withdrawn.

To answer this question an intimate acquaintance with the habits of cyclops and a minute knowledge of the locality is necessary.

Fig. 2.—Negro women washing on edge of well at Gambaga.

![Image of women washing](image1)

Fig. 3.—Negroes drawing water from Well II at Gambaga.

![Image of people drawing water](image2)

The following observations are therefore of only local significance. At Gambaga almost all the water used by the natives is drawn from three wells situated about half a mile from the town. These wells contain subsoil water only, and are very greatly influenced by drought or rainfall. They are connected by streams that flow during the greater part of the year. During the last few months of the dry season these streams dry up, and then the communication between the wells ceases, in some cases altogether and in others during the daytime only.

I began my search for cyclops in December, when the streams were full of water. During this month I readily found cyclops in all three streams, but I failed to find them in any of the wells. This is also true of the month of January no cyclops could be found in any of the wells, but they were readily found in the streams still flowing, and in the pool into which the interrupted stream from well No. 1 had emptied itself. In the case of this latter stream, therefore, cyclops had retreated downwards in the direction of the current and had entered the pool before the stream became dry.
In the first week of February cyclops could be readily found in the streams 2 and 3 and in the above-mentioned pool, but not in well 1. On February 14th I caught two cyclops in well No. 3 after long search.

On February 23rd the stream between wells 2 and 3, which had been cleaned temporarily interrupted, ceased to flow in the daytime. As the natives ceased to draw water from the well after sunset, communication was frequently re-established between the wells during the night.

On February 29th cyclops could be found in every cupful of water from well 3, and more sparingly in well 2, but were absent from well 1.

On February 27th cyclops could not be found in the streams from wells 1, 2 and 3, and on the same day I caught three female cyclops carrying ovisacs in well No. 3.

In the third week in March the stream from well 3 became intermittent and the level of the water in all the wells was greatly reduced.

From these facts it appears likely that increased facilities for the infection of cyclops are afforded at a certain period of the year. For these observations seem to prove:

1. That cyclops inhabit the streams and the wells alternately.

2. That it breeds in the wells.

This photograph, which is a picture of well No. 2, shows how the natives stand in the water of the well from which they draw.

If many cyclops were always absent from the wells, the difficulty of its infection with guinea-worm embryos, discharged into the well from the legs of those drawing water, would be greatly increased. Secondly, if the young cyclops (Nymphs) had been found in large numbers in the wells where they undergo development, very greatly-increased facilities for penetration would be afforded by them during ecdysis, and to this facility of penetration by the guinea-worm embryo during the month of May and June I am inclined to attribute the greatest importance, for it would limit the infection of cyclops to a few months of the year.

The following considerations make it probable that certain natural aids to the infection of man are withdrawn after September in each year.

1. August and September are the months of greatest rainfall at Gambaga. At the end of September or earlier—according to rainfall—the wells become full, and the increased current would carry some of the cyclops of the wells out into the streams and thus diminish the chance of catching them when drawing water.

2. In a pool of clear water cyclops keeps mostly close to the bottom. When the level of the water in the well is low the calabash used for drawing water is swept near to or against the bank. This facilitates the penetration of free-living cercariae, but at the end of September the volume of water in the well is large, and, water being drawn from the top, the cyclops are less easily caught by the calabash, which came most frequently in contact with water, but waited for the actual contact with water before extruding its young.

This fact was well known to the natives of highly-infected regions and had been acted upon from time immemorial for the extraction of the worm. In the case of fasciola hepatica it was interesting to note that in very dry seasons the rediae harboured within the liver of livestock had to be caught in pools and to a second generation of rediae which continued to live in the host of its parent awaiting a more favourable moment to venture into the outer world of free-living cercariae. This penetration of the body of man through the skin was a very old one and certainly had much in its favour notwithstanding that it is derived from an erroneous conception as to the reason of the fact (that the cercariae are carried in the skin from the region of the back). In the case of fasciola medinensis, Dr. Sambo doubted very much that the infection of man could be carried by the intermediate host to the man either through the skin or the alimentary canal. Professor Leuckart, from analogy with the life-history of a similar parasite, the caliculinae elegans of the perch, suggested the avoidance by the intermediate host, and cyclops as the probable alternative host. Fedchenko in Turkestan had actually proved the truth of Leuckart's wise suggestion, and Sir Patrick Manson had confirmed it. The larvae of fasciola medinensis, having reached some pool or well,
attacked the young cyclops, penetrated into their bodies, and there underwent a certain amount of growth and development. It was a decided step in our knowledge of the life-history of *F. medinensis*, but it was only a step. So far, the experiments made by Fedeschenko and others to bring about the finding of the real host of *F. medinensis* proved unsuccessful. Possibly cyclops was not the real host of *F. medinensis* notwithstanding that the nematode might attain a certain stage of development in its body, or perhaps the species of cyclops could bring about the complete development; or, again, a second intermediary host might be necessary, or there might be another free living stage with or without subsequent encystment, as in fasciolae hepaticae after an intermediate host. We have evidence from the peculiar limitations observed in the geographical distribution of *F. medinensis*, and by the fact that this nematode had not become established in many places within the tropics which seemed to offer the most favorable surrounding conditions, and in which it had been repeatedly and plentifully introduced during a long number of years, as, for example, in the Malay Peninsula, as already stated by Dr. Brown.

Dr. F. M. Sandwith, after corroborating Dr. Graham's remarks with regard to the periodicity of the disease in man, said: Observers in the Egyptian Sudan find that there are two or three months when human beings suffer from *F. medinensis*, while during the rest of the year the worm is not met with. It is now so many years that some of us have thought that there was a certain cycle of the disease which had to be proved. I urge those who dwell in guinea-worm countries to try and prove the point as to whether the embryo can or cannot get into man's body without passing through any species of cyclops.

Dr. W. Carnegie Brown (Penang) said that in the Straits Settlements a unique opportunity for studying the subject exists, the fact that the life-history as presented to the physician by the circumstance that in the Malay Peninsula there was no endemic infection of dracunculus. No one, so far as he knew, had ever acquired guinea-worm in the Malay Peninsula; but migratory cases were seen there. Immigrants, natives of the Coromandel Coast, where the disease was endemic, were frequent sufferers. Some idea of the length of time that it took for a female dracunculus to mature—or, more correctly, to arrive at the stage of discharging her ova and thereby setting up an infection—was to be had from observation of the length of time after the immigrants' arrival before the assistance of the surgeon had to be sought to evacuate a dracunculus abscess. He had seen a case so late as eighteen months after arrival, and he had observed no periodicity—no evident helminthic periodicity. The most frequent periodicity of the disease was all the year round with fair regularity. In such cases it was certain that infection took place in India before the patient reached the Malay Peninsula, and if eighteen months had elapsed since that time, the disease had to be considered which, if not exact, was at least definite. Another point was the peculiarity of the fact that, though cyclops was found in the rivers and canals of the Malay Peninsula, he could not find it anywhere else, and that it was a considerable number, and though undoubtedly embryo guinea-worms were discharged into these waters from infected persons, the cyclops seemed unable to develop the embryo, or at least to render it hurtful to man. It did not appear to act as a host. The fact was peculiar and worth further study.

Dr. Gray was apparently of opinion that the certain method of prevention was to be found in seeing that drinking water was pure. That excluded the popular, and he might also say, the scientific, belief that entry was gained by the skin. From analogy with other worms this method was at least probable, and the fruit idea was that the circumstance that in nineteen-tenths of all cases of guinea-worms the site of maturity was in the leg, because infection took place while the patient was standing in water, which, even if not immediately infected by the dracunculus, was not to be resolved by trying if dracunculus embryos could pass through the skin. They could not; but it was possible that infection could happen in the cyclops they might be able to do so.

Dr. Albert J. Chalmers (Colombo, Ceylon) regretted that he was unable to add anything of value to the subject of the life-history of the guinea-worm, on the spot, but he had noticed one case of what he considered to be infection by way of the skin. He did not think that the authorities of the London and Liverpool Tropical Schools were sufficiently alive to the importance of this disease. They were frequently prevented from marching and carrying out their duties owing to the prevalence of this complaint amongst them, and Dr. Graham had earned their gratitude by drawing attention to it as a serious detriment to health which this widely-spread disease entailed.

Dr. W. T. Proux (Principal Medical Officer, Sierra Leone) was the first to emphasize the importance of the disease. He had noticed something of particular study as to the methods and entrance of this disease; on the Gold Coast there was an enormous amount of inefficiency due to this cause, more especially amongst the natives. In the Gold Coast and Sierra Leone, and had been struck by the fact that while guinea-worms were very common on the Gold Coast they were very rare at Freetown; it was not a disease of Sierra Leone. There was one condition in which the Gold Coast differed from Freetown, and that was that in the latter there was a very rare and plentiful water supply derived from the hills behind, but on the Gold Coast the water was mainly derived from surface water collected in pools and shallow wells. This would appear rather to support the theory as to the entrance into the body of means of drinking water.

Dr. Claude Schilling (Berlin) remarked that one of his friends, a district commissioner in German West Africa, had many cases of guinea-worm amongst the native soldiers. After opening good wells and prohibiting the natives to take water from the brackish or stagnant ponds to bathe, the cases of guinea-worm disappeared almost entirely. This experience is in support of the opinion that the infecting guinea-worm enters the body through the skin. Dr. C. F. Harford said that the comparative infrequency of guinea-worm on the banks of the Niger as compared with the neighbouring territories of Lagos and the Gold Coast might be due to the fact that the guinea-worms obtained their water supply chiefly from the river itself, or from flowing streams rather than from wells.

### TROPICAL DISEASES OF THE SKIN

**By J. M. H. Malcolm, M.A., M.D., M.R.C.P., Lecturer on Skin Diseases to the London School of Tropical Medicine**

**Assistant Physician for Dermatology, Skin, Charing Cross Hospital, etc.**

The subject for consideration of this morning's session is the tropical diseases of the skin, and it is with feelings of great diffidence that I find myself in the highly-honoured position of opening the discussion upon it. We had hoped to have been favoured to-day by the presence of Professor Jeanesime, of the Institut de Medecine Tropicale at Brussels, but he has been prevented from coming. I am delighted to have another opportunity of referring to the subject of tropical dermatology a special study, and to have had this duty performed by him. Unfortunately he has been prevented from coming, and Dr. Robert Crocker, who has been invited to take his place, has also been unable to do so.

I must ask you, therefore, gentlemen, to be more than usually tolerant, for I cannot but feel the grave responsibility which, if the subject enters the body, the disease is made. I am a substitute for so well known an authority as Professor Jeanesime.

The only consolation I can find in my present position is the fact that the subject of tropical dermatology is in its infancy, and, with the exception of the work of several Continental observers who have had special opportunities of studying it, it is a field of medicine which is only partially explored and to which the art similar may be applied of a map on which the large countries alone are marked out, while within these lines much still remains blank and unknown. Though various tropical skin affections have been more or less minutely described it is only comparatively recently that the subject as a whole has attracted the attention which it deserves, and the publication of Jeanesime's *Cours de Dermatologie* in 1899 may be said to be the textbook upon it. At present it is in a state of disorder almost amounting to chaos, and this is due to several causes: in the first place because we are indebted for the description of various tropical diseases to observers who have lived in foreign parts and who have not had the facilities for working out the pathology of these diseases. A great deal has still to be done, and tropical skin diseases is confusing, since many of the names applied to them are native generic names, and used indiscriminately for a variety of affections which differ in their clinical details, etiology and pathology, and on the other hand various skin
Parasites of various kinds are responsible for a large number of tropical skin affections. Hot climates, the more or less insanitary habits of the natives, and the want of protection of the skin render the inhabitants of warm countries exceptionally liable to the attacks of parasites, of which the chief bacteria, streptotrichae, yeasts, moulds, and a selection of animal parasites belonging to the groups of the acarida, insects, and vermes.

1. Due to Bacteria.

Most of the skin affections which occur in the tropics, and are caused by bacteria, exist also in sub-tropical and temperate climates. Tuberculosis cutis, for example, is widely distributed both in tropical and temperate countries. In warm climates lupus vulgaris is, however, rare, though scrofuloderma is comparatively common. This subject requires further observation, as for our knowledge of tuberculosis of the skin becomes more exact, we have come to recognize the fact that the skin manifestations of tuberculosis are almost as multiple as those of syphilis, and what the exotic clinical types of all are still to be shown.

Of the infective granulomata of bacillary origin one of the most familiar to us is leprosy. In 1871 Hansen discovered the bacillus leprosis, and much careful work has been done on the subject since then, but there are still several problems which require solution, and which form favorite subjects for controversy. Although the bacillus is generally admitted to be the cause of the disease, and is found in greater profusion in the skin lesions than are associated with any other member of the infective granuloma group, attempts to cultivate and inoculate it have so far been unsuccessful. From time to time the scientific world has been startled by announcements that pure cultures had been obtained, or inoculations had been followed by positive results, but these have invariably proved to be immaterial.

Only last year Captain Rost, M. S., asserted that he had been able to cultivate the bacilli on an aseptic medium, and had succeeded in making a “leprosin” on the same principle as Koch’s tuberculin but these assertions have been proved since to be premature.

There have been numerous theories with regard to a possible source of infection being through the alimentary tract, but articles of diet and water have been repeatedly scrutinized without success. The theory of infection has been generally credited. In the present state of knowledge, it will be necessary to demonstrate the bacillus in suspected flh, which I fear is a somewhat hopeless task in the present state of the subject.

2. Due to Streptotrichae.

The best known example of a streptotrichae inoculation of the skin in the tropics is madura foot. Two chief varieties have been described accordingly as the granules are white or yellowish, or black. The white variety has been shown to be due to a streptotrichae which differs from the actinomyces. The black variety still requires elucidation; Kanthack regarded it as the result of a degenerative type of the white fungus; recently Oppenheim has declared it to be due to an oldium. The latter observation requires confirmation.

3. Disease of the Skin Caused by Blastomyces.

Yeasts have been shown to be a factor on the skin in tropical countries in moist regions of the body, such as the axillae and about the genitae, and to be a frequent contamination of ulcerated surfaces. Recently a skin affection has been described in America, which both clinically and histologically bears a certain resemblance to lupus vulgaris of the verruca type, and from which a yeast has been recovered. This yeast has been shown to be pathogenic in animals and is believed by a number of observers to be the cause of many of the affections, and not simply a secondary contamination of a lesion due to tuberculosis, syphilis, or other such cause. About forty of the forty yeast type under the heading of “blastomycetic dermatitis,” and in several of them a systemic invasion has occurred and a fatal issue. In California and Buenos Ayres a variety of this condition has been described in which is a sporulating organism, believed also to be a yeast, has been demonstrated. To these cases the name of dermatitis coccidioidei has been given. The study of these cases is of the greatest interest, and it is highly probable that, were they recognized, they would have a wider distribution than we are yet aware of.

4. Diseases Caused by Hyphomycetes or Moulds.

The most important examples of exotic diseases due to hyphomycetes are the various types of tropical ringworm, of which there is a known variety in tropical and sub-tropical climates. The nature of the mould which causes it and which can be so easily demonstrated in the scales is at present under dispute. Tribondeau recently asserted, in connexion with this disease as with the saprophytic variety, that “an infection is a frequent contamination of ringworm cultures.” In tropical countries the gliobus skin, especially about the crust and scalp, and there is doubtless a variety of different fungi which cause the disease, but they still have to be worked out. It is most probable that just as the fungi of ringworm vary in type and relative proportions in the different countries of the world, so the fungi of ringworm will vary in type and relative proportions in the different countries of the world.
Europe so will they also be found to differ in tropical countries.

With regard to pinta—a novo hyphomycetic disease—various forms have been isolated from the lesions such as pellucida, aspergillosis, monilia, and recently a trichophyton by Bodin. But which of these is responsible, or whether there are several fungi, as there are several clinical types, of pinta, is a question which requires solving.

II.—DISEASES DUE TO ANIMAL PARASITES.

These naturally divide themselves into those which are due to acarids, insects, and vermes. Of these only a few have special interest.

Scabies is a common disease in the tropics, and has a variety of names attached to it according to the district in which it occurs. Under the heading of crab-crab, O'Neill describes an erision on the West Coast of Africa which clinically resembled vesiculose scabies, which he found to be caused by a filarial parasite which Sir Patrick Manson considered to be the F. perstans. It is doubtful whether the filaria is responsible for the affection. The name crab-scarb is employed indiscriminately by natives to a variety of skin affections, chiefly of the posterior type.

The affection known as pani-giso, ground itch, or sore feet of Asam., is of special interest, owing to the discussion which has arisen with regard to its etiology. It is an itchy papule-vesicular affection of the feet, which attacks coolies in the wet season. The virus evidently inhabits the macerated skin and is carried in consequence of the macerated condition of the feet. This can be prevented by wearing specially-shaped wooden shoes, known as kurrums. It is prevalent at Sylhet in India, and has been caused by an acarus. Recently it has been shown to be more probably due to the penetration of the larvae ofankylostoma through the skin.

In 1895 Looss demonstrated the fact that these larvae could penetrate the skin, and his observation has been corroborated since then by Dr. Sandwith, Dr. Bentley, and others; and they have produced a disease identical with ground itch by rubbing in a soil infected with the larvae.

Another disease which has found its way into the group of skin affections due to animal parasites, but in which the view to this position has been taken in at least certain of the cases, is elephantiasis arabum. The prevailing view with regard to its aetiology, and one with which the name of Sir Patrick Manson has come to be associated, is that it is a manifestation of filarial disease, and is caused by the larval form of the filaria bancrofti, namely, the F. nocturna. Clinical evidence based chiefly on the facts that the geographical distribution of this filaria and elephantiasis occur together, and that it frequently occurs in individuals suffering from undoubted filarial disease, favours this view.

On the other hand, where it occurs without concomitant filarial disease, or without the presence of filarial embryos demonstrated in the blood, it is difficult also to understand how a lymph stasis, which is an essential precursor of elephantiasis, can be caused by a blocking of the lymph glands by filarial overgrowth. The agent in this case is probably some kind of micrococci, either of the staphyloccocci or streptococci type, seems to be responsible. The most prevalent of these are the localized tropical lesions known as Oriental sore, and the other forms.

The disease has received a variety of names, of which Biscra button, and Buga button, in English, and Bicra soro, in African, are the most common. Some few examples, and seem to describe the clinical entity. To recall the clinical characteristics—after a varying incubation period, the lesion begins as an itching papule like a mosquito bite, which in a day or two forms a small crust, and on its removal a shallow ulcer is revealed, from the floor of which ooze a thin exudation. As the ulcer increases peripherally, the border becomes red, raised, and oedematous. In the interval pus may be noted on the surrounding inflammatory area.

Left to themselves, the lesions may undergo spontaneous involution, and heal in course of six months or a year; on the other hand, they may persist indefinitely, and are most resistant to treatment. The disease is contagious and auto-inoculating.

It affects both sexes and is independent of race and age, but an important factor in its etiology appears to be the state of the general health of the patient. It most readily attacks those whose health is below par from climate, insufficient or unbalanced food, concomitant infections, or any cause which lowers the vitality of the body, diminishes its power of resisting micro-organisms from without, and inhibits the natural healthy reaction of the skin to the virus, and so recruits the lesions indestructible.

Microscopical examination of the lesions show that it is a reaction of the skin against some virus of low virulence, which is probably a gram-positive micro-organism, of the streptococci, or staphyloccoci type, or staphyloccoci and streptococi 1268/10. It has been demonstrated that the virus is of an extraneous nature, and probably is a specific microbe associated with various others, or with a specific type of virus, or even with several micro-organisms as virulence, which is due to the virus of syphilis, and independent of tubercul bacilli, seems certain.

Various micro-organisms have been found in the lesions, and put forward as the cause, such as micrococci by Duclaux and Charlemesee, and streptococci and staphyloccoci by Dr. Le Dancet and Anthé, and recently Leishman bodies by Dr. Homer Wright and others, but none has yet been established. It has to be decision, also, whether the different Oriental sore are due to different micro-organisms, or to a specific microbe associated with various others, or to a specific state of virulence of the same micro-organism.

Some of these problems are by no means easily solved; when we recall that Thomaseli has been able, in at least ten different micrococci on the healthy skin, which under ordinary circumstances are harmless demeseces of it, but under altered conditions may become pathogenic; so that in any purulent skin affections micrococci are certain to be detected.

We have in this country an allied condition which is familiar to certain observers, and is known as a septic ulcerative granuloma. These lesions were at one time considered to be due to a botryomycosis, and were named botryomycosis pyrogenym, but they are now believed to result from the inoculation of a certain pus organism, but the precise microbe has not been isolated.

There is another local affection which bears a close relationship to Oriental sore—namely, the condition which is prevalent among members of the British army, and was known as veld sore, gitzeer, in the Transvaal, and brandzer in the Orange Free State. For an excellent description of these lesions and their pathology we are indebted to a recent research of Prof. Cyril Baly, of Cape Town. As the sequelae of events in elephantiasis arubam somewhat corresponds with that above described, the possibility of, at least in certain cases, an allied cause is suggested.

III.—DISEASES DUE TO A PARASITIC ORIGIN, BUT IN WHICH A SPECIFIC MICRO-ORGANISM HAS NOT YET BEEN ESTABLISHED.

In this group there are various skin affections for which various micro-organisms are suggested, such as a specific microbe of the disease, and
not an attenated form of the staphylococcus pyogenes aureus.

The vesicular nature of the lesion and its subsequent course suggest a close though not a direct relation to a staphylococcus. It differs from the Oriental sore in being more superficial, and not having the same tendency to be associated with granulomatous changes.

The phagedenic ulcer of warm countries is another example of a parasitic affection due most probably to some form of spirochaete, which has a low virulence and may yet be isolated. This lesion has a wide distribution, as its synonyms indicate, such as Aden ulcer, Mozambique sore, CochIN China ulcer, etc. It is, however, somewhat ill-defined, and has formed a disturbed ground for various affections which differ in their etiology and pathology. Jeanselme has suggested that the name be reserved for those erotic ulcers which have a tendency to slough, and are covered by a pustulous diptheroid membrane. A depressed state of the general health resulting from malaria, dysentery, or scurvy, is an important factor in its etiology; indeed, it has sometimes been designated malarial ulcer. It begins as a small nodule, and possibly results from the inoculation of the integument by means of a false membrane. According to the virulence of the infection and the power of the pathogen, it can be in the form of an ulcer with a slightly infected base, or of a somewhat pustulous base, or may take on fulminating characters, destroying vessels and nerves, denuding tendons and muscles, exposing joints, and causing necrosis of bones, and occasionally ending in a fatal issue of pyaemia, haemorrhage, and the like. The lesion in severe cases has a close relation to the now fortunately rare infection, hospital gangrene.

L. Dente has obtained from the false membrane a non-motile straight bacillus, and Boinet has also recovered a straight aërobic bacillus from the lesions which he believed to be pathogenic. These observations are of interest since they are in connexion with the term "bacillus," and in hospital gangrene. Matzenauer has also described a somewhat similar bacillus.

The pemphigus contagious which Manson has described as an affection frequent in South China, Straits Settlements, etc., also belongs to this group. It seems to correspond with our P. acuta or with P. necatrix, and has been shown to result from a virulent micrococcus, in the case of the latter of the streptococcus type.

Ketoids are particularly common in black and yellow races, and lesions resulting from them occur through abrasions and wounds of some micro-organisms still to be isolated.

To pass on to several of the infective granulomata in which the same granuloma undifferentiated type of infection—bacillus pyogenes aureus—appears, and which occur in various stages in their evolution, they are not polymorphous as the spirochaetes and the spirochaetae, and, as yet, the fact that the histo-pathology of the disease in a well-marked case is difficult to obtain from that of syphilis, all go to prove that we have to deal with two separate processes. The details of the microscopic difference of the two affections have been described at a previous meeting of this Association, and the observations have been corroborated by several observers that the two diseases are identical, or that syphilis is yaws modified by life in a temperate climate, or vice versa, that yaws is syphilis of the tropics.

Till the micro-organisms of yaws and syphilis are discovered it is difficult to say whether syphilis is yaws modified by life in a temperate climate, or vice versa, that yaws is syphilis of the tropics. The latter view seems to be the one which is most generally accepted, as those of Taguchi; but these are uncommon, and several observers deny their existence altogether and maintain that such result from the concomitant infection of vapors with bacilli inoculated with the disease suffered from "malarial phenomena," including pains in the limbs, evening pyrexia, and other symptoms of general disturbance. The histo-pathology of the lesions shows the disease to belong to the group of the "infective granulomata." Its relation to syphilis is still under discussion, since it has been maintained by several observers that the two diseases are identical, or that syphilis is yaws modified by life in a temperate climate, or vice versa, that yaws is syphilis of the tropics.

Allied in some respects to yaws is the disease of the western slope of the Andes known as verruga Peruvana. There are three distinct periods in this disease, namely, stages of invasion, eruption, and recovery. The eruption begins as red vacuoles or vesicles which evolve into purpuric exsiccations varying in size from a millet seed to an apple. The microscopical structure of the skin lesions suggested, in tissue which were brought over for examination to the London School of Tropical Medicine, a staphylococcus, and there is no doubt that we have in this disease, a peculiar type of staphylococcus. This is the disease of the Andes of Peru, and it is almost similar to the disease of the western slope of the Andes of Peru. The disease is not usually vascular. This was so marked in some of the lesions as to suggest an angiosarcoma, or a cavernous tissue made up of dilated blood vessels and lymphatics with a multiplication of leucocytes. It is a disease with a definite stage of invasion, as there has been said to result from the diminished atmospheric pressure, for the disease occurs on an altitude of from 3,000 to 10,000 ft. The locality of the eruptions is determined by the disease, but the virus has not been yet detected, though several micro-organisms have been found in the exsiccations.

The last of the infective granulomata of the tropics which I wish to refer to is the local intratable skin affection known as "ulcerating granuloma of the pudenta," or granuloma ulcerans of British Guiana. Daniels first described the affection in British Guiana, but so many cases have been recorded since then from the Solomon Islands, West Africa, Fiji, etc., affecting such diverse races as the aboriginal peoples of New Guinea, the Papuans, and the negro of the West Coast of Africa; it has been shown to be characteristic of the Negro race. It is a disease which is found in the short discharging a few hours, and has been termed by several observers that the disease was inoculable, but the virus has not been yet detected, though several micro-organisms have been found in the exsiccations.
Diseases in which Micro-organisms are Generally Present, but are not the Primary Factors in the Evolution of the Disease.

I will only refer to two diseases in this category—namely, prickle heat and dyshidrosis.

The etiology and pathology of prickle heat still remain uncertain. Some authorities believe that the vesicles on an inflammatory basis were simply sweat cysts no longer holds good, since Török showed that many of the vesicles were independent of the sweat glands. That, however, there is a causal connexion with the miliaria there can be little doubt, and it seems probable that the sweat which, when excessive, is alkaline in reaction, forms a suitable medium for certain other microbes to flourish in, and to cause the inflammatory changes, and the production of vesicles in the epidermis apart from the sweat ducts.

Dyshidrosis or chério-pompholyx is closely related to prickle heat, and affects the hands or feet or both. The double vesicular lesions of this disease were also regarded once as sweat cysts, but have been demonstrated at times to have an independent existence in the epidermis.

In warm countries this affection is frequently associated with an exanthematous condition about the crutch and occasionally on the umbilicus. These lesions suggest a secondary infection on a moist skin by micro-organisms of low virulence.

IV.—NON-PARASITIC SKIN AFFECTIONS OF THE TROPICS.

To the various skin lesions probably due to toxins, such as those of pellagra and acrodermatitis, time will not permit even a passing reference, and I must also refrain from referring to the fascinating and mysterious affections, such as the so-called erythema multiforme, syphilis, and vitiligo.

These brief and imperfect remarks may serve, gentlemen, to show the limited abilities and limitations of the present time, but if they should arouse a discussion and awaken an interest in this wide, inadequately-explored and all-important branch of medicine, they will have more than fulfilled their purpose.

PINTA.

By F. M. Sandwith, M.B., F.R.O.P., Consulting Physician, Kaiser-Ally Hospital, Cairo; Lecturer at the London School of Tropical Medicine, etc.

Although I was expected to prepare a paper upon this subject for this meeting, I must confess that my personal experience of the disease is chiefly based upon 4 cases, seen in Egypt, which have already been described by Mr. Madden and Dr. Goodall. The last six years I have been much interested in pintas, and, besides reading the French literature on the subject, I have been in profitable correspondence with many colleagues living within the pinta zone.

Symptoms.—The early spots grow to a size of 1 cm. and color into caraté.

Definition.—A tropical disease caused by a fungus, which produces various discolorations on the uncovered parts of the skin and sometimes on the mucous membranes. It belongs to the group of specific skin diseases caused by vegetable parasites.

History.—The disease has been known in Mexico since at least 1775, and has been described by various authors since Alibert, who wrote of it in 1829. According to R. F. Juan de Velasco, black African slaves imported "le caraté" into Colombia, where the people used to say it "whitens the blacks and blackens the whites." The fungus has been known since the writings of Montoya in 1896.

Distribution.—Jeanselme says there are no less than 200,000 cases in the Republic of Colombia, which includes the isthmus of Panama. This means epidermic in every 25 inhabitants, and in some parts 1 in every 10 is affected. Dr. Cram says that 60 per cent. of the adult Caribs in British Honduras are affected, and considers that it is very rare among Europeans. Pinta is also endemic in Venezuela, Peru, Bolivia, Chili, Central America, and Mexico. Isolated cases have lately been reported from Tripol (1897), Egypt (1899), and Chile (1900). The disease flourishes best in hot, damp, low-lying countries.

Causes.—The disease is rare among well-to-do people, for dirt and poverty seem to be predisposing causes. Among Indians and aborigines of personal hygiene is often left to the women, in agricultural labourers, miners, muleteers, and boatmen. Both sexes and all ages are liable, but patients are most commonly met with between 15 and 25 years of age. The disease is not hereditary, nor directly contagious from man to man, for there is no personal transmission in families or among members of barrack. The disease is not introduced by his neighbours, who mistake this disease for leprosy.

Montoya has obtained moulis of the violet and the violet-black varieties from moquitoes, and it is quite possible that these insects may be the vectors of the parasite into man's skin. It is suggested that flies and bugs may also introduce them. Montoya has found fungi of the different varieties in the diseased inhabitants of Colombia.

Rabbits can be inoculated with pintas.

Nine varieties of the fungus are given by Barbe:—dulvi violet, pure violet, blue-violet, brown-violet, blue, violet-black, Chinese violet, white-violet, and yellow. Montoya is of opinion that the different varieties of the fungus accounts for the different colours of the eruption, which, when present on the same individual, give him a very grotesque piebald or tattooed appearance.

The violet varieties are met with among negroes and mulattoes of rural and mining districts. They generally begin on the face, or forearms and legs, and may appear in the mucous membranes; loss of pigmentation occurs later.

The red variety attacks white people living in towns, who complain of dryness and smarting of skin rather than of inflammation. The colour is at first pale, but becomes brick red, and there is no secondary stage of loss of pigment. The skin of the palms and soles, when affected, is thick, and crossed by deep, painful fissures.

The red and white varieties attack the reticulo-mucous and corium, while the others affect chiefly the epidermis.

The Incubation is uncertain, but Uribe found that about a month passed between the insertion of the fungus into the epidermis of mulattoes and the first appearance of the spots.

Symptoms.—The prodromal symptoms, such as fever, vomiting, and diarrhoea, of some of the older authors have, apparently, no connection with this disease. The eruption is generally preceded by slight itching, and is confined to uncovered parts of the body. It may begin on the face, neck, cheeks, nose, lower forehead, or upper front part of the ears and occurs later on the forearms, wrists, legs, ankles, and upper part of the chest, but avoids the palms and soles.

The eruption is usually not symmetrical, and is accompanied by furfuraceous desquamation. There are two stages. The first active in increase of pigment, the second characterized by disappearance of pigment or pseudo-vitiligo, of which the traces are said to be permanent.

The initial spots grow to a size of 1 cm. and colour into caraté. The second stage is a complete remission, but in some cases the eruption will recur. In the later stages the eruption is divided into irregular patches, which in two or three years develop the characteristic colours. A patient may have an eruption of one or several colours. The nails may be affected, and are of a red and chalky color. The hair of the head and face in adults is not attacked, and protects the skin beneath, but if a man shaves, the skin may be attacked by the fungus, and the hair will then become white and will fall out. Moreover, when a boy's face is infected before puberty, he will remain more or less beardless all his life.

The mucous membrane of the mouth, tongue, prepuce, and vagina, in long-standing cases of the brown-violet variety may become coloured (Barbe).

In the second stage the pigment disappears always from the centre of the spot to the periphery, and the earliest sites to become epidermoid are the external surfaces of the bony prominences, which are rubbed—for instance, the right olecranon loses pigment earlier than the left if more exposed, and vice versa. The red variety is easily curable, but this is not so when considerable tracts of skin are involved.

Pathology.—The fungus (trichophyton pictor Blanchard) consists of pseudohyphae and oval spores from 8 to 12 μ in diameter, and tapering, branched mycelial threads to which the conidia are attached. It produces what may be called an aspergillosis of the skin.

For demonstration Montoya recommends that the scales be
soaked in ammonia for some minutes to remove fat, and that when the ammonia is poured off, there be added a saturated alcoholic solution of picric acid; after five minutes the scales should be washed in distilled water and then mounted dry. The picric acid hardens the mycelium and colours it yellow. The absence of spores in long-standing cases probably means that the parasite is dead.

Diagnosis.—Primary parasitic leishmaniasis caused by the white variety of fungus appears in large patches on the abdomen and extremities, and is not confined to the neighbourhood of joints, like the secondary leishmaniasis caused by disappearance of pigment in the second stage of the disease. This white variety is often confused with leishmaniasis (villigera) and is, indeed, the basis of the published illustrations of pinta which seem to be nothing but leishmaniasis. In the latter, which seems to be a trophic eruption, not dependent on any fungus, the white patches are symmetrical, with convex borders, and are surrounded by increased pigmentation. Moreover, leishmaniasis is sometimes hereditary.

By the photographs of the two diseases which I now show, it can be seen that the white patches in pinta are much more speckled without convex borders, they are often not symmetrical, and not surrounded by increased pigmentation. The pinta patches are never anaesthetic, which is enough to distinguish them from the macules of anaesthetic leprosy.

Pityriasis versicolor, which, on a colourless skin, looks silver gray, is usually absent from the face and limbs, and on scratching the patches with the nail much of the discoloration can be removed in scales or rolls, because it chiefly affects the superficial epidermal layers.

The coloured sketch now exhibited shows appearances on the skin, of something like lupus erythematosus, and it illustrates the difficulty of differentiating pinta from the scars of favus, with which it is often associated.

Treatment.—Tracture of iodine and Viemncky's solution applied too early causes them to disappear, but both are useless in later cases, where chrysarobin ointment or nitrate of mercury ointment may be tried. The bedding and clothes of patients should be sterilised, and cleanliness is all essential. Mosquito punctures in an infected district should be carefully avoided.

References

TROPICAL FORMS OF PITYRIASIS VERSICOLOR.

By Aldo Castellani, D.M.D.,
Colombo, Ceylon.

Pityriasis Versicolor is extremely frequent in the tropics. It is generally said that the affected parts in coloured races appear of a yellowish tint, much lighter than the surrounding healthy skin. It is also stated that the abundant growth of the fungus conceals a coat of yellow paint—to use the graphic description of Manson—the dark underlying natural pigment of the skin. This description applies perfectly to many cases, but, in my experience, not to all. In my opinion, several forms of the affection must be distinguished. In Ceylon, according to my observations, at least two principal types, each of which may show several varieties, can be found.

First Type: Pityriasis Nigra.

The affected parts are of a completely black, dull, lustreless colour, much darker than the surrounding healthy skin, which never is quite black in the natives of Ceylon. The patches are practically round, of various dimensions, and desquamating. When small they are apt to be slightly elevated. Little, if any, itching is present. This type, in my experience, never affects the face, though it may be found practically on any other part of the body. The neck is apparently the region most frequently affected. On the neck sometimes a very few small roundish patches may be observed, or in other cases the lesion may be diffuse, in the whole side anterior and lateral parts. The fungus found in this form of pityriasis is represented in Fig. 1. The mycelial threads are rather short, 18 to 20 μ, non-branching; they are about 23 μ in breadth. Sometimes they may be irregular in outline, bent, banana-shaped, etc. The spores are globular and most of them very large, 5 to 7 μ; the spores are frequently arranged in clusters. For this micropson I propose the name "micropsonia mansonii," in honour of Sir Patrick Manson.

Second Type: Pityriasis Versicolor Flava.

The affected parts are yellowish, of much lighter colour than the surrounding healthy skin. The patches are of various sizes, generally roundish, with sharply-defined but not elevated margin. Sometimes the patches are irregularly festooned, and may encircle areas of healthy skin. Occasionally the encircled healthy skin appears to be intersected by so many yellowish ribbon-like lines originating from the surrounding yellow patch. There are, in my opinion, several varieties of what might be provisionally called pityriasis versicolor flava. Two of these varieties are very easily recognised.

First Variety.—The face, neck, and upper part of trunk are the regions generally affected: the patches are yellow, not elevated, and absolutely smooth. There is no desquamation
Pellagra was first described by Gaspar Casal, in 1750, under the name of "mal de la rosa." In Spain and Italy the disease has been known as hysteria, and in 1784 it was first recognized in 1818, in Roumania in 1833, and in the island of Corfu in 1839. More recently other endemic centres have been discovered in Northern Portugal, in Austrian Transylvania, Dalmatia, Rumania, Turkey, Greece, Bessarabia, Kherson, and Poland. In Africa pelagra was first recognized in 1837 by Pruner, who had studied the disease in theраницer's statement of facts and figures and in that of others, but quite recently Sandwith has shown that pelagra is very prevalent in Lower Egypt, and is present also, although to a less extent, in Upper Egypt. Pellagra has been reported from Algeria, Tunisia, and the coast of Morocco. It has been found amongst the Kaifirs and Zalufs, and Sandwith saw two cases from South Africa, in 1900, among the coloured inunates on Robben Island. From Asia we have very scanty information. We believe it has been reported from places in Asia Minor, and Ray stated, in 1902, that he had noticed several cases of pelagraceous affections of the skin in one of the districts of Burmah in India. Sandwith had already pointed out that several medical officers to whom he had shown photographs of pelagra patients declared having seen similar skin lesions amongst their out-patients in India. In America pelagra has been recognized in Mexico, in Brazil, and in the Argentine Republic. Low, who had the opportunity of seeing cases of pelagra with me in Italy, informed me that he had discovered the disease in India not so much on account of its frequency, and in another case for arms depicted small roundish lustreless black areas alternating with areas of a very light yellowish colour and with areas of healthy skin. The various fungi were easily found.

Conclusions.

There are in Ceylon several forms of pityriasis versicolor. Two principal types may be distinguished: one black, pityriasis versicolor nigra: one yellow, pityriasis versicolor flava. As regards the microscopic appearances of the fungi, that of the pityriasis versicolor nigra is very abundant, has a mycelium of rather large size, and very large spores which run in clusters. In pityriasis versicolor nigra the fungus is scantly, the mycelium being thick, irregular in shape, the spores few, not large, and not running into clusters. In pityriasis versicolor flava of the second variety the mycelium is thin and regular in outline, the spores small, oval, and may be grouped in clusters. As regards the cultural characters of the various fungi found the investigation is yet unfinished, and will form the subject of another communication. From the few experiments made, however, it would seem that the fungi are completely different also culturally.

REMARKS ON
THE GEOGRAPHICAL DISTRIBUTION AND ETIOLOGY OF PELLAGRA.

By Louis W. Samson, M.D., Naples,
Lecturer to the London School of Tropical Medicine.

Hitherto pellagra has been known chiefly on account of its ravages in Spain and Italy, but the disease has a wide geographical reach, and I believe it is far more common in tropical and subtropical regions than we are aware of at present. When we speak of the geographical distribution of any disease we imply our limited knowledge of its distribution. This is especially the case with regard to pellagra. No doubt pellagra is still unrecognized in some of its endemic areas, its nervous symptoms, gastric derangements, and skin lesions being ascribed to other and separate diseases. There is no cause for surprise in this. A parallel and instructive example is that of kala-azar, a disease which was at one time confined to the Assam valley until the discovery of its specific agent in 1931 showed that it had a very wide distribution not only in India, but throughout the world. As regards pellagra, the recognition of the disease in Europe is easy enough for those who are familiar with its symptoms and course. Indeed, some authors have even affirmed that pellagra is altogether a fictitious disease, a summation of three concomitant, but quite distinct, morbid entities.
insanitary dwellings, and poverty. Neither will I take up your time to prove that syphils, alcohol, irritable oils, onions, and garlic are not the causal agents of pellagra; but the main theory rests most serenely because it is the theory now almost unanimously accepted.

Lusana, Fras, and others endeavored to prove that maize contains a definite and specific poisons. This theory is now quite untenable. It has been proved that maize stands in a high position as regards alimentary value. Besides, insufficient nourishment may bring about inanition and death. All of us have lived through the well-recorded state which is distinctive of pellagra. Whole populations who live solely on rice or potatoes remain quite free from pellagra, although these articles of food are far behind maize in nutritive value.

Some writers surmised that maize, like dannel (Lotium temulentum), contained normally some specific toxic substance which gave rise to pellagra. Some, of course, they were compelled to add that a special susceptibility was necessary to favor the toxic effects of sound maize. Yet, this theory is far from foundation whatever, and he may add that the seeds of maize grain are not poisonous in themselves, but owe their acrid and narcotic properties to an associated symbiotic fungus.

Neufer believes that there is nothing directly harmful in maize when ingested, but that poisons may arise from it within the intestines. He points out that the fungi are a peculiar form of autointoxication. A similar theory is held by De Giara (1909), who ascribes pellagra to a poison elaborated by the common bacillus coli from sound maize ingested by the animal. He has inoculated the animal and found the anaerobic bacillus in dogs by feeding them on porridge made from sound maize, and also to have obtained the same symptoms and lesions in animals inoculated with a toxin produced in vitro by the colon bacillus cultivated on maize media.

The majority of writers have ascribed the disease not to normal maize, but to damaged maize, some believing the symptoms to arise from certain toxic substances arising in the course of the decomposition of the grain under the influence of vegetable organisms, others attributing them to the organisms themselves. Consequently, the various fungi and bacteria which have been isolated have all been incriminated in turn as the causative agents of pellagra.

In 1844 Ballardini ascribed pellagra to sporisorium maydis, a mould which he had found in the greenish stain (verdorame) so frequently seen in the granular rinds of maize grains. He made several experiments with this fungus, and produced gastric and diarrhoeal symptoms in man, loss of feathers and general wasting in fowls. Lombroso pointed out that sporisorium maydis could not be the cause of pellagra because of its extreme rarity. He believed, however, that Ballardini had confounded the sporisorium with penicillum glaucum, which is very common on maize. Monti and Tisselli suggested that Ballardini’s organism was probably an innoxious stage of myroceousae. But whatever Ballardini saw under the microscope was not very unlike the many fungi, because his experiments, like those of Ellis, Michelasci, and others, who confirmed his researches, were carried out with a carefully-isolated organism, but simply with damaged grain. Leclercq and Cournand appointed a Special Commission to test Ballardini’s discovery, and its report, after much deliberation, was unfavourable chiefly on the ground that the verdorame was very often found on very different grains in other regions, such as Northern Italy and Sardinia, which were quite free from pellagra. However, Ballardini’s theory was adopted in Italy and also in France, especially by Rousell and Costallat. In 1880, Rousell inoculated the maize smut (Ustilago maydis) as the cause of pellagra. He pointed out that the dark olive-green spores of this fungus are invariably found in the dust of the wretched hovels of the peasants, who store their maize in the rooms in which they sleep and feed. Generally, at the first infection, fed two horses on fodder mixed with the maize smut, and after seven months one of the animals presented a skin eruption on the parts of the body most exposed to the sun. Professor Imhof made some experiments on himself, and proved that the maize smut is harmless to man.

In 1871, Lombroso claimed that pellagra is not an infection, but an intoxication and that certain toxic substances are formed in the pores in the decomposing corn through the activity of saprophytic organisms, and that these poisons cause the disease. His experiments on the decomposing maize established the poisonous nature of the spores. He observed that pellagra could be induced in man and animals. His theory is that there are two different poisons, and that their combined action gives rise to the complex symptoms of the disease. In human beings, acidophilus anaerobic bacilli and the spores of the special molding fungus are believed to give rise to ergotism. The two toxins are (a) a symbiotic contaminant found in the mould, and (b) an alkaloid resembling strychnine which is contained in the alcohoholextract and also in the oil, to which he gave the name of “pellagraxin.” Lombroso made experiments with these toxins, diarrhoea, loss of feathers, and death; in rats, wasting, choreiform movements, contractures, and death; in men, vomiting, diarrhoea, desquamation of the epidermis, giddiness, dilatation of the pupils, and malnutrition.

But Lombruso’s experiments, it has been pointed out, have been based on the acute symptoms he obtained by means of toxic substances extracted from fermenting maize; and, as the same identical results follow the administration or inoculation of analogous products extracted from wheat and other harmless foods when subjected to decomposition.

Very different substances have been extracted from damaged maize by various investigators. Hakesmann found a narcotic tetano poison “maizin,” which resembles the well-known poisoning of ammoniacal ascorine. In 1876 Peligio extracted from diseased maize a bitter substance which produces paralytic symptoms. In 1881 Moninolé, a French botanist, in his researches on pellagra, was able to find a similar poison. He particularly remarked that artificially fermented maize and ordinary damaged maize are two very interesting things.

In 1881 Majocchi found in both normal and diseased maize a very motile micro-organism which he named bacterium maydis. He claimed that, when the spores of this organism are inoculated into the granule of maize, in the position of the corn, a special infection is produced, but the spores of the bacteria itself do not produce effects similar to those of pellagra.

In 1890 Monti and Tisselli studied the flora of Indian corn and found that the most frequently occurring saprophytic organisms of the bacteria of maize are: Bacillus pellagruis, rhizopus nigricus, mucor ramosus, and aspergillus, a yeast similar to that of beer, several bacteria amongst which baclillus subtilis, a bacillus of the baclillus family, and fungi such as Rhizopus, Kamys, and Cenomyces, which are very closely related to Penicillium. In 1894 Pelizzi and Tisselli made experiments on dogs and rabbits, administering per os, or injecting subcutaneously or endovenously, the toxic substances obtained from cultures of the bacteria of maize. They observed spasmodic pains of the posterior limbs and other symptoms which they considered characteristic of pellagra.

But Carrizoli claimed that he had found a bacillus in the blood, saliva, and stools of pellagra patients. He stated that he had inoculated the toxic products of this parasite subcutaneously into various animals and that he had invariably produced symptoms resembling those of pellagra. He therefore named the parasite bacillus pellagrius. In 1900 Serenas remarked that the toxic substances which give rise to pellagra are the product of ascercinomycetes and not hyphomycetes, because the latter invariably disappear in putrid infusions of maize.

Corto, having observed that the commonest saprophyte of maize is penicillum glaucum, prepared pure cultures of this fungus and extracted a substance belonging to the amoticemic series. Ferrati made some experiments with the mixture of penicillium penicillium and damaged maize, but he was never able to induce toxemia in rats; the animals died in a few hours.

We must notice that only a certain variety of penicillum glaucum has toxic properties. The poisonous substance is not present in cultures before the third day; it is a glucoside, and is found in the mixture of the fungus only. It has nothing in common with the ordinary penicillium. It is a toxic glucoside and is not transformed into penicillin. It is poisonous for guinea pigs, dogs, cats, and rabbits, with fluoresences from pure cultures, produced symptoms very different to those obtained by the pure cultures of the acute pellagra bacillus of pellagra. Di Pietro also tested the toxic properties of penicillum glaucum, and suffered from pyrosis, vomiting, giddiness, weakness in the legs, slight tremor of the arms, frequent miscarition. Finally, having obtained a small quantity of serum from a pellagra patient recently cured, he made an experiment to ascertain whether it had any antitoxic power over the poison of penicillum glaucum. A guinea-pig, previously inoculated with the serum, presented no symptoms of poisoning when inoculated with the spores of penicillin, whilst a control did.

In 1900 Geli stated pellagra to be a true aspergillosis due to two different species of aspergillus—A. fumigatus and A. flavescens. He declared that the seasons in which pellagra symptoms appear in man correspond to “the cycle of annual biological evolution” of the two hyphomycetes. Moreover, he stated that he had been able to observe the growth of culture from the fungus of the lung, pleura, pericardium, and mesengies of cadavers of pellagra patients. He explained that the spores of the fungus pass through the internal organs, and thus reach the alimentary canal in the alimentary tract, and that the germ is formed there. It is evident that the spores pass through the alimentary canal due to the action and influence of the fluid contained in the alimentary canal, and that these spores are able to produce disease in every part of the body, but particularly in the alimentary canal, where the germ is formed and the disease arises. In a later work, Geli, together with Besia, and apparently forgetting his previous theory, ascribed pellagra to certain toxic substances elaborated by aspergilus fumigatus and A. flavescens.

More recently Geli and Besia describe two varieties of penicillum glaucum, the true causative fungus of the disease. The symptoms of one variety are excitative and therefore cause the acute forms of the disease, those of the other variety are depressive and consequently give rise to the chronic forms.

Lastly, in 1904 Possuti declared having reproduced pellagra by feeding or inoculating guinea-pigs with maize damaged either by aspergilus or penicillum. In the case of pellagra, Penicillium gossypii and aspergillus are the causative agents of pellagra it is necessary.
to review all the information we have concerning the patho-
genic rôle of these fungi and more especially of those belon-
ing to the genera aspergillus and penicillium. We know that a nodule, or pustule, that affects the skin, and is seen as the mucous membrane exposed to the air, causing a rule very slight lesions. Some, however, under special circumstances, may reach the internal organs and cause a general infection which may bring about death. For this reason the special care of these fungi is to be known te be caused by fungi; such as, thrush, ringworm, pinta, tokeau, mycetoma, actinomycosis, not one in any way re-
sent.

Several investigators have inculcated the aspergilll and have positively declared to have reproduced pellagra experiment-
ally by the intravenous injections of these hyphomycetes and have even gone so far as to state that these fungi were incriminated independently of any preconceived idea. The first were Grohe and Block. These were followed by Grawitz, Koechlin and Gaffky. 

Lichtheim pointed out that the differences were due to the fact that all species of aspergillus are not pathogenic, and that his predecessors either did not use the fungi or did not determine their species correctly. Some species (A. glaucus, A. niger, A. repens) are harmless; others (A. fumigatus, A. subcubus, A. niger) are very noxious. The virulence of the pathogenic forms is due to the fact that they are able to vegetate at a high tempera-
ture: 37° to 38° C., whilst the harmless species are unable to 
be cultivated above 20° C. Aspergillus is very frequent amongst birds, and affects chiefly their respiratory organs. In mammals it also affects the lungs and bronchial tubes, but it may attack the spleen, the kidneys, and the muscular tissue. The site of their infection is usually the lungs, the bronchial tubes, or the other organs. It is present in the sputum, the pus, the blood, and the urine. In experimental aspergillus is very frequent amongst birds, and attacks chiefly their respiratory organs. In mammals it also affects the lungs and bronchial tubes, but it may attack the spleen, the kidneys, and the muscular tissue. The site of their infection is usually the lungs, the bronchial tubes, or the other organs. It is present in the sputum, the pus, the blood, and the urine. In experimental aspergillus is very frequent amongst birds, and attacks chiefly their respiratory organs. In mammals it also affects the lungs and bronchial tubes, but it may attack the spleen, the kidneys, and the muscular tissue. The site of their infection is usually the lungs, the bronchial tubes, or the other organs. It is present in the sputum, the pus, the blood, and the urine. The infections are most frequent in the upper respiratory tract, and the symptoms are often those of a respiratory infection. They are characterized by a dry cough, a hoarseness, a sore throat, and a general feeling of weakness. The disease may last from three to eight years, and seldom terminates fatally unless complicated by tuberculosis.

The general opinion is that pellagra appeared soon after the introduction of maize into Europe, and that it advanced pari passu with the extension of maize cultivation, and with the adoption of maize as a food. The original hypothesis of Parkison, in his Theor. Botan. (London, 1640) says: "The United States sees that maize is an animal which, as soon as it is introduced into the soil, makes mention of to grow in Bacteria, which saith it was reported to be as big as olive stones."

Although we have no positive information as to the date of the introduction of maize into Italy, it is certain that the cereal was used as an article of food about the middle of the sixteenth century, that is, about 120 years before the date assigned to its cultivation in America. We are therefore confronted with a very serious dilemma. If we allow that pellagra existed and was known by other names previous to its recognition by Frappolli in 1771, then we can no longer assign a date for its introduction into Italy, and the most potent argument in favour of its association with maize is lost. If, on the other hand, we maintain that it did exist before that date, then it is difficult to explain why it did not manifest itself sooner after the introduction of the dangerous cereal.

I think there can be no doubt as to the antiquity of pellagra. In the majority of the authors, especially the "Alpin" sculls" described by Odoardi in 1776 and mentioned by Pujil in 1740, there was something more or less than pellagra. Then again the mool du pelligrino, very briefly sketched by Bibb in his famous "Portrait of the Jew," is surely in 1700, could hardly be referred to anything else. Frappolli
himself believed in the great antiquity of pellagra, and considered it to be the same as pellarea, a disease mentioned as early as 1828 in the region of the Monastero di Monza and the Hospital-Majore of Milan. Indeed, he affirmed that "pellagra is as old as the sun."

Amongst the reasons given by a recent author against the criminately theories. The fact that it would be very difficult to find the term pellagra before the end of the eighteenth century. This I heartily agree, because the term pellagra was first used by Frappeoli in 1771, but the term pellagra does not necessitate the absence of the disease pellagra. Indeed, pellagra has a nomenclature list too long to be mentioned.

I pointed out again and again by numerous observers the areas of pellagra endemicity and those of maize culture by no means overlap, and, indeed, there are vast regions in which maize is extensively cultivated and consumed, but where pellagra is rare. The most convincing example is that of the United States of America. On the other hand, pellagra has been observed very frequently in places in which maize is not cultivated and in people who have never used maize as an article of food. To overcome these embarrassing obstacles to the maize theory, the so-called "pseudo-pellagra" was invented. Now, therefore, the disease is pellagra only when it can be in any way connected with a maize diet; in every other case it is nothing more or less than "pseudo-pellagra." In 1903 Garbin described several cases which he had the opportunity of studying in Lucca, where maize was not eaten for any reason, and where villagers had not eaten maize for several years prior to the appearance of their eruptions. The case of pellagra is, therefore, different from that of enteric fever in India, for when it was almost an offence to call it by its own name, because the medical officers highest in military rank had made up their minds that it was not to be found in India. The topographical distribution of pellagra within its endemic areas is very unequal. The differences are often very marked between contiguous districts, although there may be no difference whatever in the alimentation of the respective populations. Fortunately, however, it has been adopted in the storage or preparation of maize. On the other hand, maize is hardly ever used as an article of food in the Province of Madrid. No less striking are the facts afforded by the epidemiology of the disease in Italy. Pellagra is still very prevalent in the North of Italy where maize forms the staple food of the peasants, but in recent years the disease has become far more prevalent in Umbria and in the Marches, and it has invaded the provinces of Siena and Grosseto in Tuscany, those of Campania, Teramo and Aquila in the Abruzzi and Molise where maize does not represent a principal food. It has invaded the Provinces of Rome, the Campagna, the Puglia, Sicily, and Sardinia, thus proving that it has a tendency to spread slowly, but widely and independently of maize cultivation, and maize theories.

When once established in a region, pellagra is very permanent, but its prevalence varies considerably from year to year, according to the climatic and topographical conditions of the area, as has been erroneously asserted, but in connexion with other oecological conditions as yet underrated.

A very important fact is that pellagra does not attack indiscriminately all those who live chiefly on maize, but only the field labourer. The inhabitants of towns, however poor, and although fed exactly in the same way as the field labourer, are not attacked. Towns in the very heart of intensely pellagrous areas show that some remarkable immunity towards pellagra is manifest in their populations, even in the midst of malaria. Probably, therefore, it is in the maize field that the peasant comes in touch with the specific agent of pellagra, and possibly through the agency of some biting fly. In the case of the disease, the distribution of pellagra is chiefly that of middle age. The majority of cases occur between 20 and 50 years of age. If maize, or bad maize, is the cause of the disease there are doubtless the principal sufferers, as is the case in ergotism.

With regard to sex, in some countries the disease is more prevalent among men, in others it is more prevalent amongst women. This depends entirely on the share the women take in field work. Pellagra exhibits a marked seasonal incidence; it makes its full appearance in late autumn and winter, and is at a maximum during the winter months. The characteristic eruptions reappear constantly every year in pellagrous latitudes, no matter how long they have been secluded in towns in which maize is not an article of diet.

Until quite recently the majority of physicians believed in the hereditary transmission of pellagra, although it is difficult to understand how it could be. Contagion was not admitted, but cases of conjugal pellagra have been reported. All these facts seem incompatible with the maize theory. I consider, therefore, that the general unquestioning acceptance of this theory is to be regretted, notwithstanding that it is supported by the authority of great names.

The Italian Government is spending considerable sums of money for the very praiseworthy object of elucidating and eradicating pellagra. But so unanimous is the belief that the disease is caused by damaged maize that the researches have been directed to the flora of stored maize. The Prefects of the various provinces attacked by the disease are directed to collect specimens of diseased maize, and then to examine them by the laboratories of the Board of Health in Rome, where they await official examination.

I regret that the disease is not more fully investigated in the field by competent men, and independently of any scientific research.

If I were asked to suggest a new theory of pellagra, merely as a working hypothesis, I should feel inclined to draw attention to the many analogies between pellagra and some of the protozoal diseases which have been observed in recent years. But my reason for bringing the subject of pellagra before this meeting is not to propound a new theory, but to urge the necessity of a more thorough investigation of this grave and widespread disease.

NOTES ON FRAMOESIA TROPICA (YAWS).

By J. Campbell Graham, M.D., Delhi, India.

The occurrence of this uncommon affection among Europeans is so rare that this must be my excuse for bringing before you notice two cases, a little Dutch girl of 4 and a little German girl of 6 years of age. In the first case the diagnosis was not so easy, as there was never anything more than a light papule on the thumb of the left hand to go by. This I at first took to be merely a small wart irritated by some cause or other, but the more as a brachial gland threatened suppuration, and it did not diminish, I was led to believe that there was a distinct history of specific infection from a wound on the finger that I was still in doubt as to its true nature. However, the little papule did not persist, for it cleared up the diagnosis; this was strengthened by the discovery of an exuberant yaw on the left breast of the child's Japanese nurse. This is the only case of glandular inanimation I have observed in Framoescia tropica. There was no rise of temperature, but the child became cachectic and irritable. In spite of tonics—quinine, iron, cod-liver oil, haematoxylon—careful diet, and later attempts with potassium iodide and minute doses of...
SECTION OF TROPICAL DISEASES.

[Nov. 11, 1905.]

hydro. o. creta, there was no improvement. So after six weeks I sent her across to Penang, and asked Dr. W. G. Brown to see her. He concurred in the diagnosis, and sent her up Penang Hill. Here she rapidly improved, and returned to Deli three weeks later. The papule was still there and the gland enlarged, but much less inflamed and reduced in size. The child’s general health had been so far improved, however, that we thought it wiser to leave the case to run its course, and it gradually disappeared. In spite of the greatest care, however, and the most generous diet, tonics, etc., there was not much improvement in a year and a half later, the child went to Europe. There all symptoms disappeared within a month.

The second case was obviously yaws from the beginning, though unaccompanied by any prodromal symptoms; there was no fever, no increase of pain, no enlargement of glands. The general health remained good throughout, although locomotion was greatly impeded by a large yaw at the root of the big toe of her right foot. The chief seat of the eruption was the instep of both feet, and other than some menstrual disturbances and the aches of the right foot. Here, also, tonics, internal and local treatment, were only palliative; fresh crops appeared from time to time, apparently yielding to one or another, but the disease was never completely eradicated. A complete cure was only established on her return to Europe about a year later. In this case also a Javanese nurse carried the contagion.

My experience with Javanese, among whom this affection is as common as their daily rice, is that only in an exceptionally small number of cases is there any fever or constitutional disturbance. In the treated case, and probably in the untreated, there were no cases of yaws. It is stated that the Javanese are impervious to smallpox, and I have no reason to doubt it. It is one of the great curiosities of the world that the same race is so impervious to one disease, and so subject to another.

Europeans, it would seem, so far as the two cases may be taken as typical, do not recover as rapidly as natives, although the severity of the attack and the constitutional disturbances are not much greater. Race must have something to do with it, for the Javanese on the tobacco estates in Deli have been attacked, and it is probable that they would also recover in a short time. The man whose photograph I show, a Javanese, acts as private postman on one of the tobacco estates in Deli. He was not in any way affected by the exuberant crop of yaws which covered the greater part of his head and neck, and continued his usual occupation until I stopped him and sent him to hospital. There he made a good recovery and left, after four weeks, with only one or two small patches of desiccating yaws. In other cases the classic symptoms of fever, etc., are seen, but in most of my cases they were slight.

I have lately had some encouraging results with bicarbonate of soda internally in 4 gram. doses three times daily. The improvement was marked in a few days, and continued for a fortnight, but I admit that I then hurried on the convalescence by the local application of sulphate of copper in substance.

NOTES ON YAWS (YAWS) IN FRENCH INDO-CINA.

By M. E. JEANELME, M.D.,
Professeur agrégé à la Faculté de Médecine de Paris.
[Translated and abridged by F. M. SANDWITH, M.D., F.R.C.P.]

The author saw the first cases of this disease in 1899 in Cambodia and Laos. It is prevalent there, but it is very rare. Of all tropical yaws, most resemble yaws, so that Hutchinson and others have suggested that yaws is perhaps only a form of yaws modified by race and climate. This opinion cannot be accepted, because the natives of the same race the two toxins are found simultaneously, each keeping its distinct characteristics, and partly because Charlot has furnished us with the undeniable proof of the duality of the two diseases by inoculating with syphilis an individual who was already suffering from yaws. However, it is quite true that the differential diagnosis is sometimes impossible.

Among the signs which are common to both diseases are the pains in the bones and joints, which become excruciating at times. Moreover, there are almost always a circular form, the preference for the neighbourhood of the orifices of the body, and the eruptrive power of iodide of potassium and mercury. No yaws is given as a form of syphilis, and vice versa. The chief differences of the two diseases are shown in the following table:

**Syphilis.**
- In countries where syphilis is endemic, the disease is almost certain.
- If a chancre or another syphilitic sign is found on an individual who has suffered from yaws, it is impossible.
- A great variety of eruptions.
- The eruption, especially the tertiary form, is almost certain.
- The incidence of the two diseases is shown in the following table:
- The chief differences of the two diseases are shown in the following table:

**Yaws.**
- In countries where syphilis is endemic, the disease is almost certain.
- If a chancre or another syphilitic sign is found on an individual who has suffered from yaws, it is impossible.
- A great variety of eruptions.
- The eruption, especially the tertiary form, is almost certain.
- The incidence of the two diseases is shown in the following table:

With regard to the curability of the two diseases by the same drugs, it must be remembered that actinomyositis, which of course has nothing to do with syphilis, is also cured by iodide of potassium. The author then gives notes of some cases in which an eruption exists with changes in the bones and joints, while reminding us that in countries infested by yaws syphilis is also met with. Unless some rare complications are present, yaws generally ends favourably, though the prognosis is far from being always mild.

The prognosis is unfavourable in cases of chronic ulceration, of progressive serpiginous invasion of healthy skin, and where the lesions are undermined as far as the ligaments and even the bones. These phagedenic ulcers leave horrible scars, like those of scrofula, or of deep burns.

The disease is more prevalent in children than in adults. When a child in a hut has been attacked, all the other inhabitants contract the disease if, up to that time, they have been free from it. The apparently great preference of the illness for the young is due to the fact that many adults have become immune by a previous attack. Children are, of course, more exposed to contagion than adults. In Cambodia and Laos it is quite common to see an infant being sucked by a woman whose breasts are covered with yaws, and when the child is to be weaned the mother puts into its mouth pellets of rice which she has previously masticated. Up to the age of 5 or 6 the children of both sexes are naked, and the skin is covered with scratches, and the ravages of mosquitoes and scabies; is it not possible that insects are the carriers of the disease? In certain countries the parents anticipate the inevitable attack of yaws by encouraging inoculation in every possible way.

Among adults the disease is propagated by the chopsticks which are used instead of forks, the waterpipe which is never passed from mouth to mouth, and the sleeping mats which are common to all.

The author has often seen cases of yaws transmitted from an infant to the breast of the mother. Europeans are not attacked, because they keep their bodies cleaner. The disease is not hereditary.

After a short histological description of the papules, Dr. Jeanelme tells us that the natives of Cambodia, Laos, and Siam avoid all drugs during the first three months of the disease. The patient is then subjected to fumigations of
mercury, while wrapped in a mosquito net; now and then he washes out his mouth with water or tea, which does not prevent his getting stomatitis and loose teeth. This treatment is remarkably successful for a time, though relapses usually occur.

Iodide of potassium in antisyphilitic doses has a good effect on yaws in two or three months.

**OBSERVATIONS ON “DOHIE ITCH” AND OTHER TROPICAL TRICHO PHYtic DISEASES.**

By Aldo Castellani, M.D.,
Colombo, Ceylon.

The classical investigation of Sabouraud has clearly proved the plurality of species of ringworm fungus affecting man. Sabouraud has come to the conclusion that each species has a particular sphere of influence, and that the results arrived at in the investigation of a fungus can only be considered as applying to the particular country in which the investigation has been carried out. In view of these facts it may be desirable to publish some observations I have had the opportunity to make in Ceylon, especially as according to an old tradition ringworm was introduced to England from India and Ceylon.

**Technique.**—In my investigations I have used the technique ordinarily employed for such researches: Microscopical examination of scales and hair in **lignum potassa**; preparations stained with various methods, the best of which is certainly Morris’s method modified by Walker; of cultures the medium most frequently employed being Sabouraud’s maltose agar.

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![Fig. 1.—Dohie Itch. Scraping stained with fuchsin.](image1)

**Dohie Itch.**—As is well known, this term is used very loosely in the tropics by the lay public to denote practically any pruriginous skin affection. The term, however, is especially used to denote a form of severe pruriginous intertrigo, which may affect the inner surface of the thighs, the axillae, and in stout women the region under the breasts. It is in this stricter meaning that I use the word in the following cases, which I have chosen as the most typical among the good number I have seen:

**Case I.**—European. Has been in Ceylon twelve months. A few weeks ago he noticed some roundish elevated red spots on the inner part of the thighs, associated with almost unbearable pruritus. When I saw him the skin of the inner surface of both thighs presented very large fescenoned patches with an elevated abrupt margin, which showed many reddish papules. The central portions of the patches were fawn-coloured and slightly scaly. The itching was very marked.

**Microscopical Examination.**—Scrapings were taken and examined in **lignum potassa**. A very few mycelial tubes and spores were found. The segments of the mycelium were almost completely straight and had a double contour. The mycelium showed some branching. The spores were oval or roundish and not of very large dimensions. Several maltose agar were inoculated. Greyish colonies, slightly crateriform in shape, were obtained. The fungus grew very slowly.

**Case II.**—European. Has been in Ceylon two years. The inner surface of each thigh presented a very large fescenoned patch with an elevated red margin. On the scrotum smaller roundish patches are present. On the skin of the penis from the base to the prepuce was seen a popular eruption arranged in circles, and also in two or three long parallel lines; the eruption was very abundant and itchy. On the skin of the lower part of the abdomen and the external side of the thighs several very large rings with a thin slightly red elevated margin composed of very small papules were observed. The skin inside the rings was apparently normal. The eruption on the penis and abdomen disappeared quickly, practically without any treatment, the patient using simply washing with carbolic soap, the parasitical power of which is slight. The large pruriginous patches on the inner surface of the thighs and scrotum were most obstinate. After having tried all possible remedies for three months, a cure was ultimately obtained when I applied pure oil of turpentine, followed by a borsicic ointment, to allay the pain produced by the turpentine. From the patches found on the scrotum and inner surface of the thighs as well as from the rings on the abdomen, etc., the same species of fungus was isolated identical microscopically and culturally to that found in Case I.

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![Fig. 2. Tinea circinata (Case I).](image2)

This case is interesting as it shows that the trichophytons of dohie itch may attack other parts of the body outside the usual regions (crutch, etc.), giving rise to a tinea circinata-like eruption. This, in my experience, is not a frequent occurrence. In such abnormal localizations the fungus did not apparently find the proper conditions of growth, as it quickly died out without any treatment.

**Peculiar Type of Dohie Itch.**

**Case III.**—European woman. The patient had resided a long time in the East—Japan, China, and Saigon. She has resided in Colombo, Ceylon, during the past eighteen months. On the inside of the thighs, extending also to the labia majora, a polytrichnote eruption was present. The skin inside the rings was very dark, almost black, thickened, and hard. The edge of the patches showed polymorphic lesions—the pityriasic squamae, minute vesicles, and papules. The pruritus was
very marked, and excoriations due to the scratching were present. The affection had lasted, according to the patient, two months. The microscopical examination showed the presence of a fungus with a short banana-shaped mycelium presenting no double contour. The segments of the mycelium were all separated. Several roundish spores of various size (3 to 4 μ) could be seen. The spores were not arranged in regular groups, but scattered here and there irregularly. This fungus could not be cultivated on Sabourand's medium or any other of the media employed, though I repeated the trials several times and kept the patient without any treatment which might interfere with the growth of the fungus. This case corresponds, I believe, to a peculiar form of dermatomyositis observed by Sabourand in patients coming from Indo-China. The seat and appearance of the eruption was identical, and also in Sabourand's cases the fungus, though easily detected microscopically, could not be grown on any medium.

I must add that, also, in typical cases of the common dhobie itch I have not been able in several cases to grow the fungus. In these cases the fungus was generally very scanty, though I failed also sometimes when the fungus was fairly abundant.

Fig. 3.—Tinea circinata (Case IV). Preparation from a maltose agar culture.

Tinea Circinata.

Cases of typical tinea circinata are not rare in the tropics. The disease, in my opinion, is quite different from dhobie itch; it does not generally affect the axilla or the other regions in which dhobie itch is principally found. I believe that fungi present in tinea circinata are quite different from those found in the typical dhobie itch, though all belonging to the trichophytons. The fungi found in tinea circinata in the tropics are also different species, possibly, from those found in Europe.

Case IV.—European. Has been in Colombo twelve weeks. The eruption began ten days ago. When I saw him, the whole trunk and arms was covered with a circinate eruption. The circles were very small, and of a rosy tint. There was much itching. The patient did not show any trichophytic lesions of the hair or beard. The axilla and the region between the thighs and the scrotum were free from any eruption.

Microscopical Examination.—Fungus scanty; mycelial tubes extremely rare, whole sheet are more easily detected. This is the reverse of what one generally finds in tinea circinata in Europe, where the microscopical examination shows the mycelium to be more abundant than the spores, though the fungus is on the whole always very scanty. The spores were roundish and of large dimensions (4 to 5 μ). The fungus grew very well on maltose agar, producing large scambinated snow-white non-powdered colonies. In subcultures the growth of the fungus was much quicker, and the colonies coalesced, giving rise to an elliptical elevated snow-white mass. The fungus grew very badly on agar and gelatine agar, but fairly well in broth; it did not grow in peptone water. The fungus found belongs certainly to the megalosporon type.

Mixed Infection of Dhobie Itch and Tinea Circinata.

Case V.—European. Has been in the island for several years, and is now going home (Germany). The inner surface of the thighs and nates presented a festooned eruption—very itching—practically identical to that found in Case I (dhobie itch). Besides this, on the upper part of the chest and on the left forearm an eruption arranged in rather large circles, with a slightly elevated rosy margin, was seen. The patient stated that this last eruption had appeared quite recently, while he had suffered from the affection of the thighs for several months. From the circinate eruption of the chest and arm a trichophyton was isolated perfectly identical, as far as I could be made out, to the trichophyton isolated in the case of pure tinea circinata (Case IV). The fungus produced snow-white acuminated colonies in maltose agar. In the eruption of the thighs a trichophyton was found identical apparently to that found in Cases I and II of dhobie itch.

Fig. 4.—Tinea circinata (Case IV). Maltose agar culture.

Tinea Imbricata of Mannon (Tobiala).

This skin disease, as it is well known, is due to the trichophyton Mansoni. As far as I know, no cases of such disease have ever been described in Ceylon. In December, 1904, a Tamil coolie presented himself to me stating that he had been bitten by a mad dog. I could not detect, however, any wound. Examining him—rather poorly built man of about 40 years of age—I noticed that he was covered with a diffuse scaly eruption, which at first sight looked very much like a form of lichenoses. On closer investigation it was easy to see that it was not a case of this affection. The dry large scales ran in concentric lines. The eruption involved nearly the whole of the body excepting the face, it was most marked on the trunk and on the back of the hands; the palms were not affected. I examined some scales microscopically with the usual technique; a trichophyt-like fungus was present in great abundance; the mycelial threads, the numerous spores could be easily seen; the mycelial tubes were long, longer than usually found in the common species of trichophytons; they were not curved, and did not show swellings. The spores appeared to be of a large size (about 5 μ) and of irregular shape; some were roundish, some oval, some nearly rectangular.

The microscopical characters of the fungus as well as the appearance of the eruption make me believe that the disease was really Manson's tinea imbricata. The case could not be investigated further, as he never came back. According to his statements, he had never been out of Ceylon.

Ringworms of the Scalp.—I have examined three native children with this affection. All of them presented on the scalp several roundish patches showing hairs broken off. The stumps were extremely short and did not show any whitish sheath. The patches were very slightly scaly. The epidermis was in fact practically not involved; no folliculitis or kerion was observed in any case. Hairs examined microscopically with the usual technique showed the presence of a trichophyton with large spores. These were disposed in regular chains within the hair. Cultivations succeeded in all the patients. The fungus grew on maltose agar, producing large white...
acminate colonies, apparently identical in all the cases. The fungus was certainly a trichophyton of the megalosporon endothenix type. So far I have never observed the microsponor soudini (trichophyton microsponor) in Ceylon.

CONCLUSIONS.

In Ceylon there are many forms of trichophytic affections:
1. Dihobie itch. This is a trichophytic infestro which generally presents the clinical signs of "vulgo-marginum" of Hebra. The fungi found belong mostly to the trichophytions, but, in my opinion, they are different from those found in tineas circinata. It is probably that there are several species of diholie trichophytions.
2. A rare, peculiar type of diholie itch may be observed identical, probably, to the trichophytic affection described by Sabourin in India-China patients. The fungus cannot be grown on the usual media.
3. Tinea circinata is not rarely met with in Ceylon. In the cases examined the fungus was of the megalosporon type.
4. Mixed infections of diholie itch and tinea circinata may occur, presenting the two fungi growing on the same patient, though on different regions of the body. 5. Tinea imbricata, or tokelai, due to the trichophyton megalosporon, occurs in Ceylon.
6. Ringworm of the scalp is observed. In the cases I examined the fungus was a trichophyton megalosporon endothenix. So far I did not see cases due to the microsponor soudini.

A COMMUNICATION ON A TROPICAL SKIN DISEASE.

By JOHN BEIL, M.R.C.S., L.R.C.P.,
Superintendent, Government Civil Hospital, Hong Kong.

As an Indian policeman was admitted to the Government Civil Hospital, Hong Kong, with a sore on his feet. He had been nine years in the colony, and knew of no other case of a similar kind amongst his comrades in the same station. He was accustomed to walking about with bare feet when off duty.

On the sole of the right foot was a superficial sore with ragged edges, about the size of a sixpence, which he thought was caused by treading on a nail. The sore slowly spread, and in about ten days the appearance was that shown in the photograph. There was a large circular patch about the size of the palm of the hand, red, and irritable in the centre, with circular edge undermined and burrowed in places, more especially above and below, the shallow burrow being here well marked and bleeding. Some of the skin was cut off, and after maceration showed several parasites. At no time was there any complaint of itching. Dr. William Hunter, Government Bacteriologist, at once identified the parasite as rhizoglyphus parasiticus, figured in Mr. Max Braun's "Die Tierkrankheiten der Menschen," by Dr. W. Pearse. As our photomicrographic apparatus was not working, I have taken the liberty of reproducing Mr. Max Braun's plate to complete the case, the parasite there figured being identical with the one found in this case.

In discussing these papers, Dr. Richard Crocker said the pathology of elephantiasis arabum required extended observations, and he was not of opinion that the blocking of the lymphatics by the miniature ova of filaria bancrofti, the cause of elephantiasis arabum, heretofore accepted, could be maintained. We have cases of elephantiasis occurring in this country which closely resembled that met with in countries where filaria prevailed, but the change seemed to be associated with the superficial rather than with the deep lymphatics, as in the case in E. arabum. Dr. Crocker stated that the cases of ringworm in all parts of the world seemed in almost complete resemblance, but it was quite possible the type of parasitic infection varied. Dr. Crocker had seen a case of granuloma in the groin of a negro who lived in London and had not been in a tropical country for some seven years.

Professor RUBERT W. BOYCE stated that the presence of filaria in the form of parent worms, as embryos, or as immature ova did not seem to explain the fibrous changes which were so prominent a feature in elephantiasis arabum. The cases of elephantiasis native to this country seemed to be due to a streptococcal infection, and the parasite played a part in elephantiasis arabum, the filarial infection being merely an accidental occurrence.

Dr. F. M. Sandwer believed that weevils were not the direct cause of pellagra in man, though it was certainly true that weevils and mouth infected side by side the maize. As the female weevil pierced the grain with her snout in order to make a convenient hole to lay her eggs within, it was obvious that she could easily introduce inside the grain (which may afterwards be eaten by man) any spores of fungus which happened to be outside the grain.

Major G. H. Fink, L.M.S. (ret.), mentioned with regard to Oriental sore that it was known by a variety of names in India according to the locality it was found in, namely, Delhi sore or boli, Sind sore, Lahore sore, and so forth. If was usually known as "scurvy" because, as he was told by a native, an Eastern Emperor Aurungzebe was supposed to have suffered from it, and tradition stated that it protected him from attacks of malarial fever. Whether Major Fink was not in a position definitely to confirm; but the idea prevailed in India that it did. With regard to his own experience, he had seen it occur in European officials in India on their arrival from the parts of the body while the person was asleep in his pyjama suit—namely, on the ankles, face, and hands, where the sandfly (Simulium) of the district infested bites. He had examined the sandflies, and out of a group perhaps of two (of various kinds) had found one of a particular type (three photomicrographs sent round for inspection) in that group, of which there might be only three or four teeming with parasites,
which were flagellated organisms, and could be seen on one of the photomicrographs distinctly. Sandflies bit and raised a papule, which later developed into a small ulcer with a slight area of circumscribed inflammation around. The person bitten scratched it unconsciously, and the excoriated surface turned to an ulcer with little, if any, discharge; circular, and spreading along the edges; single, sometimes, multiple at others; and in some instances likely to be mistaken for “specific sores” by novices in tropical ulcers, since they left an unholy scarring around the inner ankle, which was a favourite site for bites, and there was a copper stain on healing.

Mr. James Cantlie said that amongst the skin affections referred to by the several speakers he had heard no reference made to a patchy pigmentation of the skin of the face, neck, and hands, frequently met with in South China, and no doubt elsewhere, especially amongst women. Mr. Cantlie had given the affection the name of the “tropical mask,” which was a simple pigmentation not due to any parasitic infection, but resembled closely the pigmentation seen in the skin of various parts of the body in pregnant women. Mr. Cantlie mentioned a case in which the “tropical mask” had caused a medical man practising in Britain to regard a patient with this affection to be the subject of Addison’s disease. Mr. Cantlie stated that he had seen small sores in different parts of the body resembling Oriental sores in an officer recently returned from Tibet; in these sores, however, no Leishman bodies were found.

LEPROSY IN ICELAND.

Dr. Karl Grossmann (Liverpool) gave a lantern demonstration of coloured photographs he had taken of lepers in Iceland.

ON THE PRESENCE OF SPIROCHAETES IN TWO CASES OF ULCERATED PARANGI (YAWS).

By Aldo Castellani, M.D.,
Colombo, Ceylon.

In February last, examining some films from the secretion of the ulcers in a case of parangi and stained by the Leishman-Romanowsky method, I noticed several extremely minute and almost invisible spirochaete-like bodies. I did not attach much importance to this discovery at the time, as the spirochaetes were found together with huge numbers of bacteria and were so slightly stained that their proper study could not be carried out.

A month ago Professor Schaudinn very kindly sent me a reprint of his preliminary note1 on the finding of spirochaetes in syphilis.

Rememering the strict relation which, according to some authors, exists between parangi and syphilis, I began at once some investigations to ascertain if in yaws also identical or similar spirochaetes could be found. Unfortunately at the time there were only 2 cases of parangi in the General Hospital of Colombo, but 2 other cases—beggar’s off the streets—I was able to secure and examine. Only these 4 cases have been examined. Of these, 2 had dry lesions—syphilis the well-known yaws—whilst the other 2 cases, in addition to a few dry lesions, presented some red fungoid, slightly secreting, ulcerated excrescences, such as are generally seen antecedent to the dry-yaws stage. The investigation of the two cases of dry yaws was completely negative as regards spirochaetes. In the other two cases spirochaetes were found, though mixed with various bacteria. Of these two cases, one I examined only once, as he returned to come again. The other is a patient in the General Hospital under the care of Dr. Paul, and I have examined him many times. He is a Singhalese lad, 10 years old; he has never been out of Ceylon, and has had no other disease. There are no signs of acquired or congenital syphilis. He is in the second stage of the disease. The skin of his hands and arms presents several patches of fungoid desquamation; the soles of the feet show several fungoid, roundish, elevated, slightly secreting formations. The patient is undergoing mercurial treatment, and is improving. Practically all the experiments have been carried out on this case; the other, as already stated, refused to come again.

The Spirochaetes.—The spirochaetes found were generally mixed with numbers of bacteria. In the preparations taken from the surface of the lesions the number of bacteria was prodigious, but by scraping away the superficial layers till blood exuded and then making preparations the bacteria were fewer, whilst the numbers of spirochaetes were not sensibly diminished. The spirochaetes are extremely thin; they stain very faintly, and in fact are sometimes scarcely visible. If the Leishman alcoholic solution is, however, allowed to act for five minutes and after the admixture with distilled water for fifteen minutes, the spirochaetes are fairly well stained, generally taking on a purplish, occasionally a bluish, tint. The organisms are generally long, 14 to 20 μ, though short forms, 7 to 10 μ, are also met with. They are often pointed at both ends and present a variable number of waves. Two types might, perhaps, be distinguished—one much more delicate, with very small uniform waves, the other with fewer, larger and more graceful wavy forms and there, in the stained preparations, a few shorter, thicker and more deeply-

stained spirillum-like bodies, presenting one or two chromat points in their substance, are seen together with bacilli of various shapes and cocci. In the few lesions of the same case which had not yet ulcerated spirochaetes of the more delicate type found.

In fresh preparations (hanging-drop), the spirochaetes, though extremely thin, could be distinguished from the many motile bacteria by their peculiar movements so well described by Schaudinn.

The usual laboratory culture-media were inoculated with the secretion taken from the lesions; colonies of various bacilli and cocci developed, but, as was to be expected, no spirochaetes.

In conclusion, I wish to state that in publishing these observations, I desire not to commit myself in any way as regards the etiology of parangi.

REFERENCE.
1 Vervolglicher Bericht über das Vorkommen von Spirochaetem in syphilistischen Krankheitsprodukte und bei Pappillomen.

INFECTION OF THE SCALP IN LEPROSY.

By George Fennett, M.B.C.S., L.R.C.P.,
Assistant, Skin Department, University College Hospital; late Pathologist, Hospital for Diseases of the Skin, Blackfriars, London.

It is generally stated that the hairy scalp escapes in leprosy. As I have now seen two exceptions to this rule in this country the following short details may be of some interest:

The first case was in a male native of Bombay, aged 30, who was seen by me in August, 1896, whilst in charge of patients at University College Hospital. The patient had consulted an opthalmic surgeon about his eyes, the condition of which had led to the idea of syphilitic keratitis, but the general condition of the man being mentioned to me I came to the conclusion that the case was very probably one of leprosy. When I had an opportunity of seeing the patient for myself,
there could be no doubt as to the leprous nature of the complaint. It was a typical case of advanced nodular leprosy. In this place it is unnecessary to give a full description. As to the scalp, this is the note I made at the time: A fair amount of hair in front, but otherwise diffuse irregular alopecia. On the vertex, a finger-tip sized depression which could be both seen and felt. To the left of the this the scalp was slightly infiltrated, and there was anaesthesia to touch over an area the size of a stinging, but pressure with a pin was felt, but not with sharp end.

This patient was under observation for some little time and then disappeared. Inquiries made by me in 1903 at an address he having given me were fruitless. Considering how bad he was when I first saw him, death has probably closed the scene.

The second case is that of a white woman, who first came under the observation of Dr. Radcliffe Crocker in 1895; he described her then condition in The Lancet.1 This was also a case of nodular leprosy. At first the condition improved, but the patient became neglectful of treatment, refusing to carry it out, and was not seen for some time. She returned, however, with a Z-ray burn of the centre of face, as a result of treatment at another hospital, from which she slowly recovered with local improvement. Since then she has been seen twice and, but she has refused to take any treatment.

In March last, hearing the patient was very ill, I visited her at her home and found her in a very bad condition indeed, the disease had further struck her scalp, and if found, to me, it seems that the same was involved. About the vertex and to the right of it and behind there were two areas of raised infiltration, reddened in parts, with loss of hair. In the other parts of the scalp the hair was scanty.

Although the diagnosis of these infiltrations rested on clinical grounds alone, and lacked bacteriological confirmation, yet I have no doubt that the scalp condition was of the same nature as the general disease. In the case of the male patient, there was, it is true, a history of a sore and rash, the former of which might have been a soft chancre and the latter a leprous exanthem. Howbeit any basis for this diagnosis and treatment was absent.

These two exceptions, which appear to me to be well-founded, do not invalidate the general rule that leprosy in the great majority of cases does not involve the hairy scalp.

The same may be said of lupus vulgaris. In that disease it is rare for the scalp to be invaded secondarily by extension from the adjacent parts, and rarer still for it to develop on the scalp separately. I have had an opportunity of seeing three instances of the latter mode of invasion. One of these is in a patient attending the Skin Department, University College Hospital, and a couple of his hair-beds were absent.

The dilated vessels coursing over the lupus infiltrations have appeared to me to differentiate them from syphilomata and represent the same situation.

In conclusion, I have to thank Dr. Radcliffe Crocker for kindly allowing me to make use of the above leprosy cases for the purpose of this note.

REFERENCE
1 Lancet, 1895, ii, p. 136.

A DISCUSSION ON SPRUE AND HILL DIARRHOEA.

I.—JAMES CANTIL, M.B., F.R.C.S.,
Lecturer, London School of Tropical Medicine.

Sprue

Since the discussion on sprue at the meeting of the British Medical Association held five years ago, there has been practically nothing of pathological interest added to our knowledge of the disease. One effect of that discussion was to make sprue a name of more widespread recognition, and to make sprue practising generally in this country, so that patients returning from warm climates, when they stated that they were suffering from sprue, found that their doctors in even remote parts, had some idea of what they were talking about.

The chief features of sprue are not my intention to give a textbook account of sprue, for there are now several books in which the classical symptoms are set forth. Sprue is characterized by a bare and usually painful tongue; by frequent attacks of stomatitis with ulcers on the lips and cheeks, and on the gums; by dyspepsia; by intestinal flux consisting of bulky fermenting, soft and frequent stools of pale colour, by loss of weight, depression of spirits, anemia, and loss of strength with sharp end.

The geographical distribution of sprue is fairly wide, but its special home seems to be the Malay Peninsula and the adjacent countries. From the Far East, Africa, and some of the West Indian Islands the disease is also reported.

The commencement of the illness, which culminates in the manifestations which establish the diagnosis of sprue is quite unmistakable. The prodromal symptoms, if they may be so called, are of long duration. The patients give histories of extended periods of dyspepsia, of irregular attacks of morning diarrhoea, and usually of tender mouth. These signs and symptoms are at first intermittent, and it is only when the morning stools are constantly soft, bulky, and fermenting that the ailment can be called sprue. Sprue is a disease especially consequent upon fairly long-continued residence in the tropics, and is a most common cause of death in Europeans who have become wholly wedded to life in a warm or tropical climate, and who have reached advanced middle age.

In sprue the attack old residents in the tropics or subtropicals long after they have permanently taken up residence in Britain.

The Effect of Life in a Warm Climate on the Organs of Digestion.

To understand sprue and the organic changes it engenders it is necessary to review the effect of climate on a European, especially of Northern Europe, from this time he or she takes up residence in a warm country. At first the heat serves as a stimulant; energy is at its maximum; appetite is good, and in all aspects of life the individual is as well as or better than at home. This situation usually lasts about six months.

After six or eight months this initial physiological excess subsides and gives place to a diminished physiological activity, when anorexia, dyspepsia, lassitude, constipation, and frequent loss of sleep supervene. The physically-unfit person, after a persistence of this physiological minimum of vitality may, and often does, succumb to illness, may be more or less of a chronic invalid with gastro-intestinal symptoms as the chief note of complaint. In the case of persons of better stamina the physiological minimum which follows is not so severe, and is broken off by knowing how to regulate their lives and how to adapt themselves to the exigencies of life in a hot country. The fatigue of a man or woman in time notices that the morning stools are frequently quite soft, pale, and copious, and that the desire to go to stool develops suddenly—as soon, in fact, as one gets out of bed. Accompanying these noted derangements of the tongue is usual, quite intermittently so, however, at first.

Recurrent Stomatitis.

It is not uncommon to meet with Europeans, women especially, in warm, Eastern, Asiatic countries who are subject to irregular attacks of stomatitis. The tongue, gums, and palate are at first tender; during the second week painful spots of irritation and patches of ulceration develop in patches of the mucous surface of the oral cavity; during the third week the tenderness ceases and the ulcers heal. A recurrence of this condition may on some six to eight days, and another three weeks' oral disturbance occurs. These intermittent attacks of stomatitis may recur for a year or two or more, although attended by some degree of intestinal flux especially, they may remain undetected, unless they are necessarily followed by sprue, more especially should the patient leave the tropics for change to a temperate climate. Sprue on the other hand may develop without attacks of stomatitis, or may occur at once with but little (or none) of the above symptoms of the tongue. The tongue and the mucous membrane of the mouth generally look bare and rather glazed, but may not be pronouncedly tender.

Reduced Size of the Liver.

During this period of derangement of the digestive tract the organs of the abdomen concerned show evidence of serious
The only organ, however, in which the change can be fairly accurately gauged during life is the liver, by its change in size and by the nature of its secretions, as far as can be judged by the appearance of the stools. At first, whilst yet the new arrival is enjoying the stimulus of a warm climate, the liver is apt to be somewhat increased in size. But as soon as secretion ensues, as above described, the liver will be found somewhat smaller than normal. Then comes the parting of the ways, when the man is to maintain his health in his new surroundings, or is to break down under the effects of climate. In the former case the liver assumes normal dimensions, which in the healthiest Europeans in the tropics is a matter of over one and a half times that in dwellers in temperate climates. On the other hand, the liver may increase in bulk, or may, as in sprue patients, atrophy so markedly that it is difficult to elicit its presence either by palpation or percussion. Sprue is associated with a diminution in size—an atrophy and atony—of the liver, which would appear more physiological than pathological in character. Tumourous palpation is rare, pathological changes have not been met with in post mortem examination, and the way the liver responds when appealed to by dietary treatment, shows that the tissues and functions of the liver are in a state of depression, and clinical cases of sprue are due to fermentative changes in the contents of the bowel owing to parasitic infection rather than to chemical action.

Changes in the Bowel.

Many careful observers have searched diligently for evidence of parasitic infection of the bowel, but no definite organism has been found that is adapted to the alimentary canal. Dr. Van der Scherre, in a paper translated for me by Dr. T. C. Graham of Sumatra, is inclined to associate sprue with associated appendicitis, and advises the operation of the removal of the appendix as a means of over. The original work of Van der Scherre has been re-examined by another, and the result conflicts with the original observations of Van der Scherre. It is possible that appendicitis may be associated with sprue, but this is not likely to be the cause of the sprue, as sprue is found in the intestines of patients with attacks of appendicitis.

Post-mortem evidence of the changes accompanying sprue is only obtainable in patients that have died after a long illness from exhaustion. The early changes in the organs of the digestive tract are not seen in the post-mortem appearances of the abdominal contents indicate anaemia and atrophy of the viscera generally. The stomach and small intestines are usually empty, and the muscular coat is almost un NOTICE. The post-mortem examination of the human race is to be found in the second, third, and fourth decades of life. The disease is rare in children and adults.

Age of Sprue Patients.

Sprue attacks people between the ages of 30 and 60 as a rule. The youngest case I have treated was a man aged 25, and the oldest a man of 62. When the patient's age is over 55, the changes in the alimentary tract are small, and the disease has recently developed. In cases of long standing, an evening rise in temperature to 101° F, and over is well-nigh invariably an indication that a fatal issue may be expected within a month or two. This is the time to put the patient on a diet (solid administration), if the mouth is a fatal sign; I have never seen a patient recover from sprue after thrush appeared, and in only one case of illness of any kind have I ever seen an adult recover after thrush developed.

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Treatment.

3-4 years ago I advocated the "meat treatment" of sprue, and subsequent experience has convinced me of its efficacy. I have, therefore, condemned the milk treatment of sprue, and I do so still.

Treatment by Milk.

I only mention treatment by milk to condemn it. This plan of treatment has been advocated by many of those who deny its use, and who give details of how it is to be administered. I refrain from giving these details in case any one who reads them might be tempted to try milk owing to want of courage to break away from traditional custom.

Milk is the usual diet in illnesses of every form, although why it is difficult to account for, except that it is handy, requires little or no preparation, and was the food of our infancy. Milk, however, is not used as a meal food by many adults of the human race, and a large proportion of the human race (amongst them the Chinese) never use cow's milk at all, either for children or adults in health or disease.

Without generalizing further, let us see what happens when milk is given in sprue. The stools may immediately or after a time become solid, a fact which is considered so satisfactory that the disease is said to be arrested or curable. The patient is, therefore, encouraged in the treatment, and it is not until additional food is taken that the cure is seen to be unreal. Return to the milk treatment alone is again rigidly enforced, and the same solid stool is produced, with satisfaction to the doctor and the patient. Months pass, and still milk has to be taken or reverted to when other food is attempted. A probability will, still cling to milk as the sheet anchor whereby to check the diarrhoea, and procure a solid stool. What is this solid stool? It has neither the appearance nor the odour of a faecal deposit; it is a cheese manufactured in the intestine, to which the term faecal is inappropriate. There is no appearance of bile in the evacuation, and if the liver is examined during the continuance of the "milk treatment," it will be found to diminish in bulk.

The patient cannot digest the milk—few adults can even in health, let the alimentary canal be normal. He cannot precipitate the curd of the milk and separate it from the whey. The patient absorbs, in fact lives on, the whey and passes the curd as a "solid stool."

The only rational treatment for sprue is to attempt to restore the faulty organs—and chiefly the liver—so that they may attain normal proportions and physiological activity. Milk does not call upon the liver to further its digestion, and the continued exhibition of milk allows the functional inactivity of the liver, which has been in abeyance for years it may be owing to "milk treatment," to continue, and a "care" is impossible. The gradual addition of farinaceous foods to the treatment is the proper remedy, and when the patient is left without work and even if the diarrhoea does not return when the additional elements are added to the diet, yet is the disease curtailed and will return on the least provocation.

Treatment by "Meat."

I was first driven to try "meat" in place of milk in cases of sprue, in circumstances which left an impression impossible to forget. A lady had suffered from sprue for some fifteen years. She had tried milk, change to Europe, and every remedy known to the allopath, homeopath, native practitioner, and quack. When I saw her she had been on milk off and on for many years; solid stools frequently followed its use, but the disease recurred again and again. I gave her milk, even skimmed milk, in small and measured quantities, to no purpose. As a last resort the patient went to a village from Hong Kong to Japan to undergo treatment there, but, when she came back in a state which seemed to indicate that she had used a few days to live and the patient was about to get married, her husband to try "meat treatment" as a forlorn hope, I was rewarded by seeing the patient cured of the disease. This is now twelve years ago, and the patient continues in good health. The régime in which the disease was cured was this one, which was necessary as a rule—necessary, in fact, unless the patient is apparently dying. The treatment was a mixture of freshly made raw meat juice (slightly warmed) every five minutes. After four hours the interval between feeding was lengthened to fifteen minutes, and beef jelly and calves-foot jelly were given alternately with the meat juice. On the second day the same food was given in intervals of half an hour. On the third day 1 oz. pound beef, lightly cooked, was given midday and evening along with the above. On the fourth day the quantity was increased to 2 oz. three daily, and the jellies continued, the patient being now fed every 15 hours. On the
SPRUE AND HILL DIARRHOEA.

The treatment of sprue by meat is quite small, rapidly attains normal dimensions and to all appearances becomes restored in function. The fact that fresh milk, fresh fruit, and fresh milk is employed is not a feature to put in the last analysis of sprue as an entity, but rather is a feature in the course and progress of the disease. In the case of sprue, the beneficial effects of fresh milk are noted, and not the occasional occurrence of sprue in general,
There are various factors that contribute to the transmission of sprue. One is the exposure to the parasite, which is transmitted through contaminated food or water. Another factor is the host's immune system, which can vary in its ability to recognize and destroy the parasite. The length of time it takes for symptoms to appear after infection can also vary, depending on the host's immunological response. In some cases, the parasite may remain dormant for long periods before causing symptoms.

In conclusion, sprue is a complex disease with multiple factors contributing to its development. Identifying and understanding these factors is crucial for effective prevention and treatment strategies. Further research is needed to comprehensively understand the disease and improve diagnostic and therapeutic approaches.
DIARRHOEA FROM FLAGELLATES.

VII.—A. T. Chalmers, M.D.,
Ceylon.

Dr. Chalmers said: I think that cases of sprue are rarer in Ceylon than they used to be. Very careful diagnosis is required, as ordinary cases of diarrhoea are apt to be so labelled. My cases have all been of the better class, and in my only fatal case no post-mortem examination was available. I do not think it useful to theorize as to the cause, but consider that the liver should be more carefully inspected. The treatment I have found most useful has been liver soup both with or without intestinal disinfectant. This liver treatment is well known in Ceylon, and is in fact an old native remedy.

VIII.—Major G. H. Fink, I.M.S. (retired).

Major G. H. Fink stated that with regard to Dr. Sambon’s remarks as to the useful purpose which would be served in having gentlemen bring forward native treatment of diseases by drugs, he would like to point out that a useful addition to our treatment of tropical diseases had been contributed by Dr. Ebele, who had written a book on the subject. As to the drugs, Sir George (Dr.) Watis, the Author of Economic Products in India, had mentioned some of them extensively in his work As to the Treatment of Sprue in India. Sterilized milk treatment had been found to be successful. As to hill diarrhoea, the cause assigned had been minute particles of mica which were in the soil and got into the drinking water supply, and thus brought about irritation, etc., which became most obstinately fixed to treatment. As long as such water was drunk so the case continued.

IX.—Andrew Duncan, M.D., M.R.C.P.,
London.

Dr. Duncan did not agree with Mr. Cantlie that milk would not cure sprue, as he had had cases under his care showing that sprue could be so cured; but he thoroughly agreed with him in his estimate of the much greater benefit exercised by the meat treatment over that of milk. Patients could get cured much more quickly, whilst the liver dullness reappeared much more speedily.

X.—C. F. Harford, M.D.,
Livingstone College, Leyton.

Dr. Harford alluded to the great economic importance of a more exact knowledge of various forms of tropical diarrhoea. At the result of experience of about 30 missionaries returning home from various tropical or sub-tropical climates during the past two years, he was of opinion that diarrhoea was the cause of the various casualties of invaliding and involved the largest expenditure of men and money. Perhaps the most unsatisfactory point was the want of exact knowledge as to the pathology of the various forms of intestinal trouble associated with diarrhoea, and he ventured to hope that the schools of tropical medicine would give more serious attention to this subject. As one who was called upon to advise missionaries going to the tropics in matters of health, he found that very few had any idea as to the seriousness of diarrhoea when occurring in the tropics, and he considered that every effort should be made to impress upon such individuals the importance of treating even slight forms of diarrhoea.

XI.—John Hadden, M.D.,
Berwick.

Dr. Hadden advocated vegetarian diet in the treatment of intestinal ailments, and was of opinion that a rigid vegetarian diet was the best form of food for Europeans in the tropics.

XII.—J. H. Ebele, L.R.C.P.,
Colombo, Ceylon.

Dr. Ebele said he was well acquainted with the liver-soup treatment of sprue in Ceylon, and he could testify to its efficacy. “Toddy,” made from the fermented juice of the coconut-nut palm, was an old routine remedy much used in Ceylon in the treatment of sprue.

XIII.—John Hutson, M.B.,
Barbados.

Dr. Hutson stated that he was of opinion that the disease termed thrush in the Barbados was really sprue. Pellagra seemed to be associated with the disease in the West Indies. The difficulty in employing “meat” as a treatment for intes-
flagellates; in the second, a very few could be detected after a long search.

Recently a case very similar to that just related has come under my notice. The patient, a prisoner in the Borrelia Convict Hospital, under the care of Dr. Johnson, had complained for several days of diarrhea. There was no fever, no griping pains. The stools were liquid and of a brownish colour; they did not contain blood or pus.

The microscopical examination showed the presence of huge numbers of flagellates. The faeces were investigated bacte-
iologically—the bacillus coli and a coccus only were grown.
The patient recovered in a few days, the number of flagellates decreasing enormously, though a very few remained after the attack was over.

The Intestinal Flagellates.
The parasites were studied in fresh preparations and in preparations stained with various methods. The stained preparations, according to my experience, are seldom very successful; if many slides, however, are examined it is possible to find a few parasites fairly well stained. The flagellates present could be divided into three groups:
1. Large, pear-shaped bodies 18 to 25 μ in diameter, possessing an undulating membrane along the body, two or three flagella at one pole, and a short thick one (if it is a flagellum at all) at the other pole. An indistinct nucleus was present, and several apparently non-pulsating vacuoles could be seen. In the very rarely well-stained preparations (Romanowsky's method) the nucleus is more distinct—at least, in some specimens; it is roundish and appears purplish stained. The rest of the body is bluish. These parasites are trichomonas—in the hominis (Davaine).
2. Small roundish bodies 8 to 12 μ in diameter, without any undulating membrane. These bodies had one flagellum only. The protoplasm was homogeneous. No vacuole present. In preparations stained by a modified Romanowsky's method a small roundish, rather indistinct nucleus stained purplish could be seen close to the pole from where the flagellum takes origin. These parasites are, in my opinion, cercomonata. I am aware that at the present time the general tendency is to consider as trichomonas the flagellates known to the older authors as cercomonata. In my two cases, however, the differences between the two groups of parasites were too well marked; size, number of flagella, general shape of the body, were quite different in the two types.

3. Roundish non-motile bodies 15 to 20 μ in diameter, somewhat by Romanowsky appeared blue, with sometimes a few vacuoles. The chromatin was collected generally at one point. These bodies may perhaps be considered as encysted forms. These forms are very much rarer than the motile parasites.

In case No. 2, besides the three groups of parasites just described, two more forms of protozoa were observed.
1. Several actively motile organisms, 12 to 16 μ in length, with eight flagellae, two of them taking origin at the inferior pole of the body and the others from various parts of the body. In fresh preparations these flagellates showed in the posterior portion of the body two very refractive roundish points situated close together. This parasite is Lambi intestinealis (Lambli).
2. A few individuals of another form of protozoa were present which I have met with for the third time. This protozoon may be a developmental stage of the trichomonas. Provisionally, however, I described it under the name of entamoeba undulans.1 I observed the parasite for the first time in a case of chronic dysentery. It is an organism of variable size, the maximum diameter reaching from 8 to 30 μ. Smaller individuals may be occasionally met with—though very rarely. There is an absolute absence of flagella. The organism presents a continuous rapid undulat-
ing movement from one to the other extremity of the body, this being due to the presence of an undulating membrane. Now and then, at an interval of fifteen to twenty seconds, a very narrow, long straight pseudopodium is shot from the body. The pseudopodium is emitted very quickly, and as quickly retracted. Only one is emitted at a time. The para-

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Fig. 1.—A, Encysted form: trichomonas hominis (Davaine); B, cer-
comonata.

Fig. 2.—A, Lambia intestinealis (Lambli); B, entamoeba undulans
(Cat.).
[The text is a discussion on Beri-Beri, with a focus on its causes, symptoms, and prevention. The author discusses various theories about the origin of Beri-Beri, including arsenical theory, rice theory, and place theory. The text mentions the importance of proper hygiene and disinfection in preventing the spread of the disease. The author also refers to previous studies and works by other researchers, such as Ross, Prager, and others.]

A DISCUSSION ON BERI-BERI.

I.—J. TENTUS CLARKE, M.R.O.S., L.R.C.P.,

Or the present theories of the cause of beri-beri only the following need, I think, be considered:

1. The arsenical theory of Ross.

2. The “rice” theory of Hamilton Wright.

3. The “place” theory.

4. The “acute” or “subacute” infectious disease of Hamilton Wright.

The “place” theory, there is in support the similarity of most of the symptoms and the finding of arsenic in the hair and toenails of some cases; but the herpes, the pigmentation, and the painful feet of arsenical neuritis are not seen in beri-beri. To argue that pigmentation might be missed on a dark skin is beside the point, as on the skin of a Chinaman a change of colour is readily seen, and it is impossible to suppose that these symptoms can have been missed by men who have seen some thousands of cases of the disease. In addition to this, the neuritis of beri-beri is chiefly pachyphymatous, whereas that of arsenic is chiefly inflammatory.

The “rice” theory has its main support in the almost complete immunity of Tamils from the disease in a country where beri-beri is one of the three chief causes of death. These Tamils, for example, boil their rice twice; while the other races husk their rice raw, and so it may become contaminated by the poison which lies in the husk. It is the only difference between the Chinese and the Tamils, for the former rarely take hot things with their rice, whereas the Tamils always take a quantity of pungent things containing essential oils, which it would be as reasonable to regard as prevents as it is to regard another article of diet as a cause of the disease.

The “place” theory supposes that the virus exists in certain places or grains, some considering that the organism, others that only the toxin produced by the organism, gains entrance to the system. Only conjectures can be made as to how the organism or toxin enters the system.

The theory of Hamilton Wright is that beri-beri is "an acute infectious disease," having a definite primary lesion in the stomach and small intestine, and that the apparent "organism may be ingested in any food and drink eaten," and may multiply itself in the tissues by a toxin both in the contents and walls of the affected stomach and small intestine. The chief support for this theory is the finding of gastro-duodenitis in all cases which died early stages of the disease. The organism has been isolated, but it is not uncommon to find bacteria in the stools of beri-beri patients. If this theory is correct, it involves the idea that the stomach and small intestine may be infected by a toxin, and it is possible that the disease may be transmitted by contaminated food or water.

Of his definition, the words "acute" or "subacute" may be accepted. I do not understand in what sense the word "infections" is used.

A theory on the "rice" theory is a small experience of my own. I was in charge of a hospital and of a ward for decipient vagrants in Lower Perak, Federated Malay States. The hospital had been in use for several years; the decipient vagrant ward, though an old building, had for many years been empty, except that it was occasionally used for the medical inspection of vagrants. At the time of its opening in September, 1903, as a decipient vagrant ward it had not been used for any purpose for over a year. The hospital and the decipient vagrant ward were about 300 yards apart, the cold, air, and ventilation were the same. The hospital had cement floors and very well ventilated wards, the decipient vagrant ward had raised wooden floors and very badly ventilated wards; the patients and the hospital staff were the same.

The conditions at the hospital were on the whole far better than they were at the decipient vagrant ward, except for the fact that the clothing, bedding, and utensils at the hospital had been in use for some time, whereas all these things at the decipient vagrant ward were absolutely new.

Beri-beri cases were admitted into both institutions, but of 30 cases in the hospital between the middle of November, 1903, and the end of the year, 6 cases arose in the hospital in men admitted for other diseases, and 29 of the 30 cases died, showed an extraordinarily low virulence; during the same period at a decipient ward no beri-beri cases died, and no new cases arose amongst the inmates. After very thorough disinfection of the hospital, including boiling all clothes, linens, beds, floors, and walls, and washing the patients' surroundings, both the hospital and the Chinese ward, the same number of cases were found, and no change was made in the diet.

The Tamils (who were admitted in number to the Chinese acquired the disease, though their rice was the same and actually cooked in the same vessel with that for the Chinese. It may be noted, however, that they had bathed and washed in water containing arsenic, but the same arsenicals and were not regarded as a place, gang, or institutional disease, and the questions are: What kind of organism is the cause? Does the organism or its toxin gain entrance to the body, and, in either case, how? The disease may infect a place, may disappear from that place, and disinfection may apparently prevent further cases. Although it is possible that the infection is in the patient, something more is wanted, as it is quite the exception for the hospital to be admitted, and which are not changed for those of the ward, while with the sick these things are interchangeable.

If the infection lies in a hospital ward, it is most probable that it lies in the bed-boards, clothing, or bedding. It is not easy to see how it could lie in cement floors (which are not washed and limed once a week), in lime-washed brick pillars, or in walls made of "bertam," which allow the air to percolate freely through.

The bed-bug is a most difficult thing to keep out of hospital bed-boards, and though the infrequency of the disease in towns, as compared with the mines, and the rarity of the disease in Tamils might be considered that this is not the case, yet it seems to me that the so-called "place" will probably come down to the bed-board, clothing, or bedding, and that a not uncommon inhabitant of these things might have something to do with it.

With this idea I tried to make bed-bugs bite monkeys. I kept beri-beri patients on beds boards all day, and at night removed the patients and tied a monkey to my bed-board. I find that bed-bugs frequently do this, but I have not had any result. I then shelled a monkey and fixed up bed-bugs (taken from a beri-beri patient's bed-board) under a watch glass, which was bandaged on and wrapped over the monkey. On the days those bugs which had not got out were dead. Nothing happened to the monkey; in fact, I do not think these bugs will bite monkeys. I then crushed up about
twenty bugs in water and injected this under a monkey's skin, but the time of paralysis (three weeks afterwards) was still quite well. On several occasions I injected 4 or 5 grains of cerebro-spinal fluid into monkeys and one monkey was ill after it for some days; did not take its food and was unable to climb a post, after it had got about 6 ft., having apparently great weakness of its hind legs. Although these experiments all had negative results, I attribute the epidemic hospital epidemic described was only small, I think that they may be put forward, as if one insect is eliminated as a cause, another may be tried.

III.—HAMILTON WRIGHT, M.D. McGill, Late Director, Institute Medical Research, Federated Malay States.

DR. HAMILTON WRIGHT having referred to former papers on the subject of beri-beri in which he had endeavored to show that the incubation period of the disease is short—from seven to fourteen days—said: It is an acute or subacute infectious disease, due to a specific organism not yet certainly determined: the organism is not one whose special habitat is any food such as fish or rice, but one that may nevertheless be ingested in any food and drink accidentally contaminated; that after an entrance to the alimentary canal, that organism multiplies in the contents of the stomach and small gut, but chiefly in the contents of the duodenum; that it there elaborates a toxin which, being absorbed, poisons certain different and effect different nerve points in different extents and in different degrees, and thus gives rise to a group of symptoms which can be classified as acute pernicious, acute and subacute beri-beri. I point out that the probable duration of the active stage of the organism varies from two to six weeks, and that after its active stage has ceased and its virus has been eliminated, the paresis or paralysis it has engendered persists. For this persistent paralysis I have proposed the term beri-beri residual paralysis, and I maintain that the great mass of our knowledge of beri-beri is founded on the study and statistics of this residual effect of disease—beri-beri residual paralysis—and not truly upon the disease itself, acute and subacute beri-beri. It then seems evident to me that we should be just as clear in our conceptions of diphtheria if we had based our calculations of it on post-diphtheritic paralysis, as we are in our notions of beri-beri through basing our theoretical and hypotheses upon the residues of the disease.

In my papers I have not only outlined my clinical and post-mortem observations on man, but gave a fuller description of the disease seen in monkeys which had contracted the disease a number of years ago, and which had been found to exist in the stomachs of many adult male Chinese and other Orientals who had succumbed. There is not only clinical evidence, but also indisputable post-mortem evidence, that beri-beri must be regarded as an acute infectious disease, having a definite primary lesion.

IV.—LOUIS W. SAMBON, M.D. Naples, Lecturer, London School of Tropical Medicine.

DR. SAMBON read with great interest Dr. Wright's reports on beri-beri, and considers that the latter's clinical and post-mortem observations in the Malay Peninsular had elicited many important facts concerning the epidemiology of the disease, but he could not follow him when he stated that what we had hitherto called beri-beri was only a sequela, a "residual paralysis," and that the true disease was a duodenitis of short duration, an acute disease which he ascribed to a bacillus that he had neither cultivated nor inoculated but only found post mortem. To Dr. Sambon the neuritis was the essential feature of beri-beri, and considering that in each case, and at different periods during the course of the disease, different sets of symptoms were implicated, that some were implicated and whilst others degenerated, he was inclined to believe that the causative agent of the disease was to be found in the diseased nerves themselves. In his opinion, the epidemic organism was to be found in the spinal ganglia, and in various forms of the disease in the cortex of the brain and in the horn of Ammon. In leprosy, also, the bacillus was frequently found in the diseased nerves. As to the possible agent of transmission, he thought that notwithstanding Dr. Clarke's negative experiments the bed-bug was to be regarded as the most probable, because it was the only one which seemed to fully agree with the peculiar epidemiology of beri-beri. It also explained the difficulty of eradicating the disease from infected collective dwellings. Dr. Sambon wished to draw attention again to the latency and persistence of the disease in those who had once contracted the infection. The relapses of the disease in the second or third section of the year, and under certain conditions, were analogous to that of malaria, and explained many obscure points in the epidemiology and natural history of the disease.

V.—D. J. GALLOWAY, M.D.,

Singapore.

DR. GALLOWAY said that probably no disease had been so lost sight of under the mass of its symptoms, or more obscure in its occurrence, than beri-beri. A study of the literature showed that whatever the phase which the different writers started to elucidate, they all gravitated to the question of epidemiology. Alimentation had two factors—something eaten and the alimentary tract, and a close study of the numberless experiments which had been tried, and the numberless food materials which could only point to the futility of attempting any explanation from the diet. There remained the alimentary tract, and he described the symptoms he had seen in very early beri-beri—epigastric pain, anorexia, diarrhoea, dullness and boarding of the epigastrium, pain over the upper abdomen, and a peculiar condition of the mouth. He found, in conversation with Dr. Lim Born Keng, that these symptoms had been observed also by him, preceding the appearance of localized oedema or neuritic symptoms by ten or fifteen days. He sketched a possible process by which the infection might be spread based upon the habits of expectation of the Chinese, and he drew a rough analogy between the waves of malaria which used to puzzle us before we knew the role the mosquito played, and he suggested that possibly the ordinary fly might play the part of carrier.

VI.—F. O. STEDMAN, M.D.,

Hong Kong.

DR. STEDMAN gave details of an outbreak of beri-beri in a sailing ship during an eighty-day voyage from New York to Hong Kong, no port being visited, beri-beri having been declared in the New York beri-beri developed. The passengers attacked occupied a saloon in the centre of the ship. None of the crew in the forecastle of the ship were attacked. The only explanation of the outbreak was attributed to the fact that a Chinese steward on board the ship, who occupied a berth near the saloon in which the beri-beri sufferers were quartered, was said to have had beri-beri previously. These cases would
VII.—Andrew Duncan, M.D., M.R.C.P. Lond.

Dr. Duncan drew attention to a case of beri-beri under his care, in which, during the week before he was in hospital, there was an increased knee-jerk instead of a loss of the same. He also mentioned the fact that in 1874, when he was House Physician at the Seamen's Hospital, he never saw a case of beri-beri; and, in fact, this disease did not appear in the annual hospital return till several years after this date. Is it a more or less new disease since this date?

VIII.—W. L. Strain, M.B., C.M.,
San Paulo, Brazil.

Dr. Strain said he had seen three sporadic cases of beri-beri occurring post partum, the symptoms appearing within a period of 48 days after confinement. He had not seen any epidemics of beri-beri, but many sporadic cases. On one occasion he saw a small epidemic in the state gaol of Minas Geraes, and as a point of interest mentioned that the native surgeon to the gaol who accompanied him was treating the cedematous cases by administering one litre of cow’s urine, stating that it was the best thing he knew, probably anticipating the modern treatment of cedema by urea. Dr. Strain’s belief as to the etiology was quite in accordance with the views expressed by Dr. Hamilton Wright.

NOTE ON A PECULIAR SCHISTOSOMUM EGG.


Lecturer, Liverpool School of Tropical Medicine.

The eggs were found in the urine of a Madras native suffering from haematuria. Their appearance at once struck one as different from the ordinary schistosomum haematobium egg, as seen, for instance, in the urine of soldiers who had returned from South Africa. Their striking peculiarity is their long spindle-like appearance. The thick end of the egg, instead of being blunt, as regarded as in S. haematobium, is prolonged into a long, snout-like process, giving the egg its spindle shape. The actual dimensions are: Length, 205,5 μ; width, 53,2 μ. So that in length the egg exceds that given by any observer for S. haematobium: 135-160 μ by 53,66 μ (Railliet); 120-190 μ by 50,73 μ (Braun). They somewhat resemble the figures given by Railliet of the egg of S. mansoni in aspect. It must be stated that the eggs were the urine of the specimen of this shape. Some are indistinguishable from S. haematobium. This fact makes it difficult to come to any decision about the natures of these eggs, and we must content ourselves with placing on record their occurrence without expressing any opinion as to whether they are a new species or not. The eggs were submitted by us to Professor Loos, of Cairo, who very kindly examined them for us. He states that he had not seen similar eggs in Egypt, but would hesitate to ascribe them to a new species solely on the appearance of the eggs. In this opinion we concur, and must await further evidence on the question, but we think that having drawn the attention of observers to the possibility of new species of schistosomum, further data may be soon obtainable.

NOTE ON THE METHOD OF TAKING QUININE IN THE PROPHYLAXIS OF MALARIA.

By St. George Gray, M.B., B.Ch., Dub., Sierra Leone.

Quinine is one of the oldest and probably the most generally used of all the agents now employed in the prophylaxis of malaria, but it is not unusuall for this purpose by those who rely on it for the keeping of their health in the Colonies. One man takes his 3 or 5 gr. a day, another takes the same quantity every other day, while still another takes 10 gr. once a week or fortnight.

Each man thinks his own way to be the best, and recommends it to his newly-arrived friends. If the newcomer should happen to be advised by two or more persons whose methods of taking quinine differ, he perhaps consults the medical officer, and, as the medical officers themselves differ in their opinions as to the proper method of taking quinine, he is often puzzled to know which of his many advisers to follow, and takes his quinine or not as he thinks best.

It is certain that on the West Coast of Africa, I have made inquiries on this subject of my European patients, friends, medical officers, and fellow passengers on board ship, and find that hardly any two of them take their prophylactic dose of quinine at the same time, and not one of those that I have asked could tell me why he has adopted his own particular method, except that it was recommended to him by a friend or medical officer.

To those who ask me I invariably give the same advice.
First of all it is necessary to understand something of the action of quinine on the parasite of malaria. It destroys the parasites in the blood, but, so far as we know, it destroys that parasite more slowly when the drug is given by mouth, than when it is given by injection. It is probable, however, that the method of taking quinine which I recommend is based (a) on the fact that the life cycle of the parasites is nearly always forty-eight hours; (b) that some eight or ten days must elapse after infection before they become sufficiently numerous in the blood to cause an attack of fever.

Hence it follows that if a full dose of quinine (10 or 15 gr.) be taken on two successive days, with an interval of eight or nine days before the next dose, it will be found that the parasites will always be destroyed before they can cause fever.

Nearly every European on the West Coast of Africa keeps a calendar on which he ticks off the days every morning until the time for leave. On my calendar I mark certain days—namely, the 1st and 2nd, the 11th and 12th, and the 21st and 22nd of each month. When I see the date marked in this way I know that it is my day for taking quinine, and after taking it I make an additional mark to indicate that this has been done.

This additional mark is necessary as a reminder for those who have short memories, because they sometimes forget whether they have taken their quinine or not.

This is practically the method of Koch. It is quite as efficacious as the 5 gr. a day plan with the advantage that there is a week when one is not taking any quinine at all (which is fully appreciated by those who are susceptible to the unpleasant effects of the drug) and less than half the quantity is used.

I have taken my prophylactic quinine in this way for more than two years, and have not yet been off duty for a single day through illness, although I have been in all sorts of "瘴地" localities, and lived in many different places, on more than one occasion to sleep in an open boat and in other places where it was impossible to avoid being bitten by mosquitoes.

The advantages which I claim for this method of taking quinine as a prophylactic are as follows:
1. It is at least as efficacious as any other.
2. Less quinine is necessary.
3. There is an interval of at least a week when the subject is not taking any quinine.
4. Last, but not least, patients and others are more willing to follow instructions if the reason why this plan should be adopted in preference to any other is explained to them.

MALARIAL FEVER IN BRITISH CENTRAL AFRICA.

By H. HEARSEY, M.D.,
Principal Medical Officer, British Central Africa.

The forms of malarial infection met with in this country are mainly two, namely, those due to the parasites of "tertian" and "aestivo-autumnal" fevers.

1. Tertian.

This is by far the commonest type of intermittent fever prevalent, but owing to the frequent double infection by parasites and their reaching maturity on alternate days, the clinical manifestations are often those of quinine fever. Blood films hardened in equal parts of alcohol and ether, and stained with methylene blue, show the parasites as small blue rings, in the peripheral portions of which are pigment granules. These two types, the "single" and "double tertian," are amenable to treatment with quinine. When there is "multiple" infection by tertian parasites, one of the so-called "irregular" fevers is the result. These forms are generally resistant to treatment with quinine.

2. Aestivo-autumnal.

This is the type of fever which, there is reason to believe, is mainly responsible for the "pernicious" attacks. Specimens of blood prepared in the above manner often show no parasites, but when seen they are found to closely resemble those of the tertian form, except that they are smaller, and contain but scanty pigment. It is probably owing to the presence of these parasites in several groups, with varying cycles of development, that we have associated with this infection the "remittent" and "continued" temperatures. These types are generally resistant to treatment with quinine.

Although one may in some cases fail to detect parasites in the investigation, the red parasites after the first attack has continued for some days show remarkable changes of a necrotic kind. They are wrinkled and crenated and of a marked pallor, giving them the well-known "brassy" appearance.

This leads me to the consideration of Haemoglobinuric Fever.

After repeated examinations of the blood in this type of fever I have failed to find any parasites. But the degenerative changes in the red cells just described have always been found. In only three cases have I had an opportunity of examining the blood before the onset of haemoglobinuria; in two of these the results were negative; in the third a few small ring parasites were observed, which, however, subsequent experience has shown not to have been haemoglobinuric developed. I am therefore inclined to regard haemoglobinuric fever as a "pernicious" form of the aestivo-autumnal, the absence of parasites being probably due to the poisonous fluid having left the peripheral circulation for the spleen and bone marrow.

The occurrence of haemoglobinuria may be attributed to the following causes as favouring its development:

1. An anemic condition of the blood due to previous attacks of malaria rendering the haemoglobin more unstable, and permitting of its liberation on slight provocation.
2. A sluggish condition of the liver, whether induced by malaria, indiscipline and irregular habits of life, food, or drink, or from climatic causes, preventing this organ from disposing of the unusually large quantity of haemoglobinset free by converting it into bile pigment. There is reason to believe that haemoglobin is liberated in every severe malarial attack, but it is ordinarily effectually disposed of by the liver, so that none appears in the urine.
3. The presence of nephritis, post-malarial or other, increasing the permeability of the renal epithelium sufficiently to allow haemoglobin to escape. A large number of post-mortem examinations made in British Guiana in fatal cases of malaria showed the presence of nephritis in considerably over half the cases.

In view of the repeated failures on the part of many observers to discover a specifically different parasite to account for this type of fever, I see no reason for regarding it as other than one of the many forms of pernicious malarial fever induced by one or more parasites. The therapeutic test of quinine failing to alleviate the symptoms of this disease no more proves it to be non-malarial than does the similar test in the case of iodide and mercury prove syphilis as yeaws to be identical.

Treatment.

I now come to the question of treatment. Bastianelli has mentioned certain conditions under which quinine may be given in haemoglobinuric fever, and this has been quoted in almost every work on the subject. So far as this Protectorate is concerned, I think I may safely say that after an extended trial of this drug quinine will not again be given to a patient suffering from haemoglobinuric fever. A much higher percentage of recoveries has been obtained by completely discarding its employment, and patients have recovered satisfactorily and without any complications or sequelae.

Six years ago I pointed out that quinine not only did not appear to exert any beneficial effects, but on the other hand it that it aggravated vomiting and added further to the distress of the patient. Its toxic action rendering the haemoglobin more unstable had also to be considered.

I then advocated treatment with bicarbonate of soda and perchloride of mercury, after a successful trial of this remedy in a case; and later published a paper showing nineteen consecutive recoveries under this treatment. Since then I have only had the opportunity of treating two others, and both have recovered satisfactorily, making a total of 21 consecutive cases of recovery. Of the 5 cases previously treated with quinine, 4 died, 3 from the action of the poison, 1 from partial suppression and syncope. I have not had a single case of urinary suppression since adopting the present mode of treatment.

An injection of 3 gr. of morphia is the most effective and reliable for controlling vomiting. Brandy is used to the exclusion of other stimulants; acid drinks are prohibited and the patient is allowed barley water for allaying thirst. Benger's food, milk diluted with barley water, chicken soup, Valentine's meat juice, and Brand's essences compose the dietary.

The mixture contains 10 gr. of bicarbonate of soda and 50 minims of the solution of perchloride of mercury in each dose. It is given every two hours during the first day and every three hours subsequently, until the urine clears.

TWO FURTHER CASES OF SNAKE BITE TREATED SUCCESSFULLY BY LOCAL APPLICATIONS OF POTASSIUM PERMANGANATE, WITH SUGGESTIONS FOR EXTENSION OF ITS USE.

By LEONARD ROGERS, M.D., M.I.M.S.,
In the Indian Medical Gazette of February, 1905. I placed on record five recoveries from snake bites after treatment by permanganate of potash locally after incision, through the fang marks, so had been made in accordance with the method lately advocated by Sir Landor Brunton, Sir Joseph Fayrer, and myself. Two of the cases were treated immediately after the receipt of the bites of a cobra and of a Russell's viper respectively in the persons of snake men employed at the Zoological Gardens, Calcutta. In two other cases of bites by full-sized cobras, in the persons of natives living in the environs of Calcutta, ligatures were at once applied by the relatives of the patients and a native medical man summoned, who successfully carried out the treatment after fifteen and twenty minutes respectively from the reception of the bite. One of the patients was a boy of 11, for whom the average amount of venom ejected by a cobra would have been twenty times a fatal dose. In each case the cobra was killed and was identified by the medical man. In all the following cases of undoubted bites by poisonous snakes have been reported to me as successfully treated in the same manner, and they are the direct outcome of the revival and improvement of this method of treatment when first tried. I have in one of them the snake lance devised by Sir Landor Brunton was actually used, and in the other the treatment was carried out by a layman when far from medical aid. The case was worthy of record as evidence of the simplicity and practicability of this method of treatment.

RUSSELL'S VIPER BITE SUCCESSFULLY TREATED BY A LAYMAN.

This case has been reported to me by Mr. Elves from a...
remote part of the north of Siam. While in camp there his cook was bitten in three places in the left arm by a Russell's viper. Mr. Elvès was at once taken to a tent, and he had a ligature at once applied near the armpit; one of his men then sucked out the wounds, after which he lanced them freely, and soaked the arm in a very strong solution of permanganate of potash for half an hour. The ligature was tied off after five hours, and the arm swelled very much and became discoloured (as is always the case in viperine bites), but there was no sign of the effects of poisons. The man was killed and was identified with the aid of Nicholson's book on snakes. Although it is not possible to be certain that this man received a fatal dose of venom, yet there is a considerable probability that he did. And I am told that he did not die, as was saved by the treatment so promptly carried out, although Mr. Elvès only knowledge of the treatment was from reading a letter of mine in the Times advocating it in very general terms.

CASE OF COBRA BITE TREATED SUCCESSFULLY WITH THE SNAKE LANCET AFTER HALF AN HOUR HAD ELAPSED.

This case has already been recorded by Dr. Protap Narain Singh in the Indian Medical Reporter, and the following abstract is taken from an account of the case he kindly sent me:—A Hinda, aged 30, when going to bed, trod on a cobra, which bit him on the foot, both fangs entering. He immediately tied two ligatures, one just above the foot and one above the knee. Within half an hour of receiving the bite he was brought to the nearest dispensary, where it was found that he were found to be efficient, and the marks of the fangs very distinct, with a little bloody serum exuding from them. On pressing, a few drops of similar fluid exuded. An H-shaped incision was made with the lancet through and about an inch square, and the wound was washed with a solution of permanganate of potash. A black mark was observed on the foot, and the wound packed with a few crystals of permanganate of potash. He was then sent to hospital. He had been bitten at 9.30 p.m.; the higher ligature was removed immediately after the treatment and the lower one three hours later. The next morning a deeply discoloured wound remained, with much bruising which disappeared on the third day. He remained afebrile, and the man made a good recovery. Two snakes of the Gokura variety of cobra were killed in his room the next day, the smaller of which had a bruise on its back as if it had been trodden on.

SUGGESTIONS FOR THE POSSIBLE VALUE OF PERMANGANATE LOCALY APPLIED IN OTHER CONDITIONS.

The success has already been recorded by Dr. Protap Narain Singh in the Indian Medical Reporter, of its usefulness as a local antiseptic, especially by persons in isolated positions, as when out on shooting expeditions.

TREATMENT OF SCORPION STINGS.—It has recently been shown by a worker in Egypt that the poison of scorpions is of the nature of an albumose—that is, it belongs to the same class of albuminous bodies as do snake venoms. It appears probable, therefore, that permanganate will act as well against the former as it does against the latter, and one case has been reported to me in which it has been applied in a strong solution to the seat of a sting by a scorpion, with the result of at once relieving the pain.

PREVENTION OF INFECTION OF DUST-LADEN WOUNDS BY TETANUS.—It is well known that wounds into which the dust of streets or earthen has entered are particularly liable to tetanus infection, and nowhere is this more common than in Calcutta, where the wards of Medical College Hospital are never long free from this terrible disease. Recently prophylactic doses of tetanus serum have been given in cases of wounds which are likely to be followed by tetanus, and the results of this treatment have been very favourable. I have long been of the opinion that this is the proper method of counteracting the effects of this disease as well as the only available one to the hospitals of large towns as a rule. The difficulty of preventing tetanus with certainty by even careful antiseptic cleansing of deep wounds in the neck region, though slight, has been somewhat removed in such a case taken to be a superficial abscess in the abdominal wall. On the other hand, in Europeans (and occasionally in natives) coming for treatment on the first appearance of symptoms of tetanus, and the absence of any evident signs of a hepatitis, nothing is more difficult than to decide if suppuration demanding early surgical treatment has already taken place, or only acute hepatitis, which will undergo resolution under appropriate

BLOOD COUNTS IN ACUTE HEPATITIS AND AMOEBA ABSCESS OF THE LIVER, WITH FURTHER EXPERIENCE OF THE RELATIONSHIP OF THE AMOEBA DYSENTERICA TO TROPICAL LIVER ABSCESES.

By Leonard Rogers, M.D., I.M.S.

Nothing is easier than the clinical diagnosis of a liver abscess in a large proportion of cases, both in hospitals in India and elsewhere, but the subject is difficult because in this disease, as they so frequently only, present themselves when the physical signs are very evident, and by no means rarely when a soft, bulging abdomen is apparent. In India it is generally not possible to make a superficial abscess in the abdominal wall. On the other hand, in Europeans (and occasionally in natives) coming for treatment on the first appearance of symptoms, and the absence of any evident signs of a hepatitis, nothing is more difficult than to decide if suppuration demanding early surgical treatment has already taken place, or only acute hepatitis, which will undergo resolution under appropriate.


**SECTION OF TROPICAL DISEASES.**

**Nov. 11, 1905.**

**Table I.—Cases of Acute Hepatitis Without Abscess Formation.**

<table>
<thead>
<tr>
<th>No.</th>
<th>Nationality</th>
<th>Sex</th>
<th>Age</th>
<th>Result</th>
<th>Red Corpuscles</th>
<th>White Corpuscles</th>
<th>Ratio of White to Red</th>
<th>Polym.</th>
<th>Lymph.</th>
<th>Large Mono.</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>European</td>
<td>M.</td>
<td></td>
<td>Recovered</td>
<td>5,390,000</td>
<td>11,100</td>
<td>1 - 4.58</td>
<td>73</td>
<td>23</td>
<td>4</td>
<td>Acute hepatitis present—liver abscess suspected.</td>
</tr>
<tr>
<td>2</td>
<td>M.</td>
<td></td>
<td></td>
<td></td>
<td>3,750,000</td>
<td>10,350</td>
<td>1 - 2.67</td>
<td>72</td>
<td>20</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M.</td>
<td></td>
<td>37</td>
<td></td>
<td>3,875,000</td>
<td>7,957</td>
<td>1 - 2.67</td>
<td>73</td>
<td>26</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>M.</td>
<td></td>
<td>24</td>
<td></td>
<td>5,380,000</td>
<td>9,500</td>
<td>1 - 1.78</td>
<td>79</td>
<td>14</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>M.</td>
<td></td>
<td>32</td>
<td></td>
<td>4,400,000</td>
<td>3,750</td>
<td>1 - 1.25</td>
<td>68</td>
<td>20</td>
<td>20</td>
<td></td>
</tr>
</tbody>
</table>

**Cases of Liver Abscess.**

<table>
<thead>
<tr>
<th>No.</th>
<th>Nationality</th>
<th>Sex</th>
<th>Age</th>
<th>Result</th>
<th>Red Corpuscles</th>
<th>White Corpuscles</th>
<th>Ratio of White to Red</th>
<th>Polym.</th>
<th>Lymph.</th>
<th>Large Mono.</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>Native</td>
<td>M.</td>
<td>40</td>
<td></td>
<td>2,670,000</td>
<td>13,850</td>
<td>1 - 5.25</td>
<td>114</td>
<td>50</td>
<td>12</td>
<td>Admitted for fever.</td>
</tr>
<tr>
<td>7</td>
<td>M.</td>
<td></td>
<td>45</td>
<td></td>
<td>4,900,000</td>
<td>11,750</td>
<td>1 - 2.67</td>
<td>58</td>
<td>20</td>
<td>12</td>
<td>Admitted for perihepatitis.</td>
</tr>
<tr>
<td>8</td>
<td>European</td>
<td>M.</td>
<td>45</td>
<td>Died</td>
<td>4,750,000</td>
<td>14,500</td>
<td>1 - 3.25</td>
<td>114</td>
<td>50</td>
<td>12</td>
<td>Admitted for fever.</td>
</tr>
<tr>
<td>9</td>
<td>M.</td>
<td></td>
<td>38</td>
<td>Recovered</td>
<td>3,550,000</td>
<td>8,925</td>
<td>1 - 2.67</td>
<td>58</td>
<td>20</td>
<td>12</td>
<td>Admitted for fever.</td>
</tr>
<tr>
<td>10</td>
<td>M.</td>
<td></td>
<td>50</td>
<td></td>
<td>3,465,000</td>
<td>16,350</td>
<td>1 - 4.62</td>
<td>85</td>
<td>20</td>
<td>12</td>
<td>Admitted for fever.</td>
</tr>
<tr>
<td>11</td>
<td>M.</td>
<td></td>
<td>48</td>
<td></td>
<td>5,420,000</td>
<td>12,750</td>
<td>1 - 2.36</td>
<td>58</td>
<td>20</td>
<td>12</td>
<td>Admitted for fever.</td>
</tr>
<tr>
<td>12</td>
<td>M.</td>
<td></td>
<td>45</td>
<td>Recovered</td>
<td>4,830,000</td>
<td>20,750</td>
<td>1 - 4.30</td>
<td>85</td>
<td>20</td>
<td>12</td>
<td>Admitted for fever.</td>
</tr>
<tr>
<td>13</td>
<td>M.</td>
<td></td>
<td>40</td>
<td></td>
<td>3,460,000</td>
<td>13,000</td>
<td>1 - 3.80</td>
<td>85</td>
<td>20</td>
<td>12</td>
<td>Treated for malaria.</td>
</tr>
<tr>
<td>14</td>
<td>M.</td>
<td></td>
<td>36</td>
<td></td>
<td>3,865,000</td>
<td>16,250</td>
<td>1 - 4.20</td>
<td>85</td>
<td>20</td>
<td>12</td>
<td>Treated for fever.</td>
</tr>
<tr>
<td>15</td>
<td>M.</td>
<td></td>
<td>30</td>
<td></td>
<td>3,050,000</td>
<td>15,250</td>
<td>1 - 5.00</td>
<td>85</td>
<td>20</td>
<td>12</td>
<td>Treated for fever.</td>
</tr>
<tr>
<td>16</td>
<td>M.</td>
<td></td>
<td>20</td>
<td></td>
<td>4,800,000</td>
<td>33,050</td>
<td>1 - 6.87</td>
<td>85</td>
<td>20</td>
<td>12</td>
<td>Treated for fever.</td>
</tr>
</tbody>
</table>

Treatment is present. During the last few years I have made blood examinations in a number of such doubtful cases, either in the Calcutta hospitals or in consulting practice, and as I have found blood counts, especially with regard to the presence or absence of leucocytosis, of great diagnostic value, I propose, in the present paper, to give the results of my experience, with some illustrative cases.

In the table I have placed first a few cases in which very evident symptoms of acute hepatitis were present and elevation was suspected, but in which resolution took place without any evident abscess formation. In the second part of the table cases in which abscess of this organ was found are entered.

A general review of the table shows a striking difference between the two classes of cases as regards the number of leucocytes. Thus, in the first part, comprised of cases of simple acute hepatitis, leucocytosis is either absent or limited to a very slight degree of that condition. In Case 1, 11,500 leucocytes were found, but here the red were also above the normal owing to diarrhoea, so that the ratio of white to red corpuscles was very nearly 1 to 50. On the other hand, Cabot found marked leucocytosis in the large bowel in the first case which he reported.

In several other cases, where the signs of liver abscess were clear, leucocytosis was also present, except in one case in which the abscess was pointing through the abdominal wall as a soft,’hemispherical swelling, and tension was thus to a great extent relieved, and no leucocytosis was found. These cases are not included in the table as there was no difficulty in the clinical diagnosis of abscess of the liver in them.

The Degree of Leucocytosis and the Acuteness of the Case.—The most striking point in the second part of the table is the great variability in the degree of leucocytosis present, between 30,000 and 40,000 having been present in two of the cases, while in others the increase is slight or even absent altogether. It is worthy of note that in the first two cases which gave the highest count the breaking down of the liver substance proved to be very rapid, while in each there was also copious haemorrhage from the bowel and high fever. In one of them a post-mortem examination was obtained and very extensive amoebic dysentery, in addition to extensive liver abscess formation, was found. I have also seen over 40,000 leucocytes in very acute amoebic dysentery, with extensive sloughing of the large bowel. In the second case the acute abscess was not due to amoebic dysentery, but was the result of an abscess of the rectum. On the other hand, in cases of the opposite character in which the formation of abscesses in the liver takes place very slowly and with no obvious symptoms pointing to the local condition for some time, only low, intermitent fever is present, and the degree of leucocytosis is slight. Case x, with only 8,625 leucocytes, and Cases xi and xv, with 12,750 and 15,450 belonging to this type, the last two having had no leucocytosis at all when first admitted to hospital for "fever," but the above counts were found at a later date when some enlargement and tenderness of the liver had begun to appear and the case xii was brought up liver pus. In these cases leucocytosis should be carefully watched for, and a comparatively slight degree of it will warrant exploratory operation either with an aspirator or the knife.

Case viiii, in which no absolute increase of the leucocytes was present, is of special interest, for I was led to give the opinion that no abscess was present. The very experienced physician in charge of the case concurred in this opinion, and no operation was undertaken, but the patient gradually sank and died, and at the post-mortem examination I found a very large single abscess with a thick, fibrous wall in the right lobe of the liver and a main amoebic abscess of the stomach. The higher count might have saved me from this error, for it will be seen that the number of the red corpuscles is only about half the normal, so that although only 8,625 white corpuscles were found yet the proportion of white to red works out at 1 to 332, or nearly double the normal proportion, this figure being about the same as in most of the cases of liver abscesses in the table, while the proportion is considerably greater than in any of the cases of acute hepatitis without abscess formation. In fact, a relative leucocytosis was present which should have put me on my guard, especially in the case of a low and insidious onset such as this was. In this connexion it is interesting to observe that Cabot found marked leucocytosis in 6 out of 7 cases of liver abscess, and he remarks on the exception that "I have never been able to account for its absence in that case." Yet I observe that in his case also the red corpuscles were much below the normal, so that the ratio of white to red works out at 1 to 338, a relative leucocytosis being again present. It is clear, then, that before the presence of liver abscess is excluded on account of an absence of an absolute degree of leucocytosis the red corpuscles should be counted and a relative degree of this condition also excluded. On looking at the column in the table which shows the proportion of white to red corpuscles, the ratio of white corpuscles was markedly above the normal in all except one, and in that case the examination was made twelve days before he began to cough up pus in an insidious case, and unfortunately no later examination was made. Further, in the cases in the first part of the table, in which the signs of acute hepatitis subsided without evidence of suppuration, there was no marked increase of the ratio of white to red corpuscles. This increased ratio, then, appears to be a safer guide to the presence of
amoebic abscess of the liver than is the absolute number of leucocytes.

Remarks on some of the more Instructive Cases.

I have already mentioned that only cases in which the diagnosis of liver abscess was clinically difficult have been included in the table, but it will be seen from this point by relating the histories of a few of the most interesting cases which have not been referred to above.

Case x.—The illness began with hepatitis, and, liver abscesses being suspected, exploratory incision was performed, with a negative result. A week later profuse haemorrhage from the bowel occurred, and I was asked to test the blood for typhoid. As there had been symptoms of hepatitis for two days before the abscess was opened, and found 38,500 leucocytes, while the Widal test was negative. A day or two later the physician in charge of the case asked me to examine the blood again, this time for malarial parasites, as he thought the temperature chart showed some trace of a tertian type of fever. This examination was also negative, and a few days later a large abscess in the liver was found by aspiration and opened; but the case terminated fatally, as cases with such a high degree of leucocytosis usually do.

Case xi. Temporary Recovery with Subsequent Relapse.—This case of exceptional interest, for, in spite of my having found 20,400 leucocytes and suspected liver abscesses as an account, the temperature fell to normal, and ten days later he left hospital apparently well. He proceeded to a hill station, where he quickly recovered and, on marking the abdomen of the liver was successfully opened. In connexion with this history a case which occurred at the General Hospital, Calcutta, some years ago, is of great interest. Symptoms pointing to liver abscess were so marked that the diagnosis of suppurative peritonitis was made, but on the morning it was to have taken place the patient's temperature fell to normal, so it was postponed. He made a good recovery from that day and left the hospital. Some three years later he returned with a totally different disease, which proved fatal. A post-mortem examination was made, and an old encysted abscess of the liver was found. Another explanation of Calcutta x is that the patient was suffering from an amoeba dysentery, and the abscess encysted owing to the amoebic infection having died out, but that another abscess developed, and was subsequently opened, as in a case to be mentioned presently.

Case xii. Case of a Patient with Malaria and sent on a Sea Voyage.—This was another difficult case admitted for fever without any very evident signs of liver disease. The medical man in charge thought he had found malarial parasites in the blood, and, getting no good effect from quinine pushed up to 40 gr. a day, the patient became worse, and I was asked to examine the case, and find, if possible, something of obvious increase. As an obvious increase of the leucocytes, I made a total count and found 13,000 white corpuscles, and a ratio of 1 white to 200 red, the latter being considered as high as is usually found in cases of malarial malady. I had already made arrangements to send his patient on a sea voyage to try and "break the fever," and four days later he started for Colombo against my advice. At Colombo he went straight into hospital, where an abscess of the liver was opened without delay, and fortunately he made a good recovery.

Case of Cocisae Aneurysm with Leucocytosis Simulating Liver Abscess.—This case illustrates the importance of seeking for other possible causes of leucocytosis before concluding an abscess of the liver is present when the local symptoms point to that organ as the seat of the disease. A native patient was admitted to hospital suffering from pain in the epigastric region and some enlargement of the liver. There were also signs of the right lung being affected, and on aspiration some blood-stained fluid was withdrawn from the least part of the right pleura. On examining the blood I found 17,000 leucocytes and diagnosed liver abscess spreading to the base of the right lung. The abdominal pain was more marked than in any other case of liver abscess I have seen, and occurring in the epigastrum which showed neither fluctuation nor pulsation. An exploratory abdominal incision was made, and a hard tumour found projecting from beneath the left lobe of the liver, which was considered to be a sarcoma, and consequently left untouched. A few days later the patient died, and at necropsy I found the tumour to be an aneurysm, nearly the entire degree of leucocytosis was seen in the lminated blood clot, in front, but which had leaked up along the right side of the vertebral column into the right pleural cavity, which contained a large quantity of blood.

Cases of Other Conditions Operated on for Liver Abscesses.—The following cases may be mentioned as examples of errors of diagnosis in difficult cases which might have been avoided by a blood count: In the Indian Medical Gazette of August, 1904, I. S. Major Barry, I. M. S., records one case in detail, and refers briefly to a second, in which a "mass" in the liver was supposed to be a sarcoma, but what he had been asked to operate on was some blood counts, and he has since written to me to say that he has had another case in which he suspected liver abscess, and would have operated but that he had suspected leucocytosis abscess of the liver, and his patient recovered without any operation. Another very difficult case was that of a man admitted in a very bad condition with enlarged liver and oedema over the ribs. Liver was diagnosed liver abscess, but, on the differential count, he made, and his patient improved and recovered without any operation. These cases also showed that the blood count was no abscess could be found. The case rapidly proved fatal, and granular kidneys and oedema of the lungs and other tissues was found. I cut sections of the kidney and lung, and the latter was quite free from inflammatory changes, so leucocytosis was most probably absent. In this case a blood count was only omitted on account of the urgency of the case; but in connexion I would point out that a brief examination of a well-spread blood film rapidly stained will suffice, with a little practice, to enable a correct opinion being formed as to the amount of leucocytes and, from this, and an examination of the film, and, if necessary, a blood film count given in the table above show that this polynuclear increase is nearly so marked as a feature in liver abscesses as in acute malarial attacks, and it is very desirable that a count given in the table above show that this polynuclear increase is nearly so marked as a feature in liver abscesses as in acute malarial attacks, and it is very desirable that a

Conditions 1. Absolute leucocytosis is nearly always found in amoebic abscess of the liver, but in chronic cases with marked anaemia only a relative leucocytosis may be found.

2. The leucocytes are very variable, being highest in the most acute cases, while a low degree is commonly met with in cases with an insidious onset, in which repeated examinations may be necessary.

CONCLUSIONS.
In acute hepatitis without suppuration leucocytosis, both absolute and relative, is nearly always absent. A slight degree may sometimes be met with in the more acute cases, but is usually only of such slight amount as to be equal to or smaller than in cases of ipecacuanha if no suppuration is present.

**FURTHER EXPERIENCE OF THE RELATIONSHIP OF THE AMOeba DYSENTERY TO TROPICAL ABECCS OF THE LIVER.**

In a paper read before this Section three years ago I dealt very fully with this question, and recorded that by examining the walls of the abscesses of patients who had died of amoebic dysentery, I had been able to find living amoebae in 35 consecutive cases. In two-thirds of which were otherwise sterile, either in the cavity of the abscess or in the suppuration, I have found them present. In conclusion, I stated that the amoeba is the sole cause of the large "tropical abscess" of the liver, as opposed to the multiple pyaemic abscesses which I have described. Those nearly as many cases have now been recorded, but the results are so similar to those I have already reported that it seems unnecessary to record them in detail. They may be summarized by saying that in nearly every case which I have described of the large abscess of the liver, there has been obtained living amoebae from the walls of the abscess which have been found, while the considerable majority of these abscesses were opened at the time of death, and examined post mortem.

In the course of the abscess examination, I had been able to find amoebae in a large majority of cases, and it was stated by many observers that the abscesses of the liver seen in European countries as I have found in Calcutta, for a large proportion of patients who have lived to reach Europe, will present the more chronic forms of the disease. I have also observed that there is a greater chance of the amoeba becoming scantly or dying out before the abscesses are opened. Moreover, in the cold climate of England the amoeba will usually have lost their characteristic movement before the necropsy is held, and hence be very difficult to identify with certainty. Again, in many recorded cases only the pus from the abscess has been examined, while in my experience, the examination of the abscesses has been found to be much more conclusive. In the case of the abscess, cause and effect have been found, but in the cases of the abscess, cause and effect have not been definitely established.

**Case of Single Tropical Abscess of the Liver in which no Amoebae were Found Post Mortem but with Amoebic Dysentery.**—A Hindu male, aged 30, was admitted to the Medical College Hospital for liver abscess. He gave a history of diarrhoea but not of dysentery. Two days after admission a large abscess was opened through the eighth rib on the right side. The abscess cavity was of smooth, fibrous wall of an evidently chronic abscess. A careful examination of scrapings from the wall failed to show a single amoeba. A large half of the large intestine showed typical and extensive amoebic abscesses. In cases terminating fatally so soon after operation as in this case I have always found the find amoebae post mortem hitherto, but it will be observed that in this case the abscess was evidently an old-standing one, but the amoeba had died out, and the wall cicatrized to form a thick layer of fibrous tissue. In view of the absence of amoebic dysentery there can be little doubt that this was what had happened. Further, it is evident that abscess of the liver may be completely encysted, as in the case already mentioned, and such a process is much easier to understand in the case of suppuration caused by an organism like the amoeba than when produced by virulent staphylococci, etc. The strongest possible confirmation of the view that an amoebic infection of an abscess of the liver may die out is afforded by the following case, which has been met with during the last week.

**Triple Tropical Abscesses of the Liver, with Amoebae in only two of them and Chronic Amoebic Dysentery.**—The patient, a native, was admitted for abscess of the liver, but with no history of dysentery. Two days after a large abscess was opened in the right lobe of the liver, but he gradually sank and died in two weeks later. At the necropsy I found two additional abscesses besides the one which had been drained. One presented similar features, as it produced the under and anterior portion of the longitudinal fissure between the right and left lobes of the liver in the shape of a very small abscess from half an inch to an inch in diameter. The other was an abscess, which had died out, and the wall was formed of a dense layer of fibrous tissue which was also visible on the upper surface of the organ. It contained thin granular material, which showed and the microscopic examination of the abscesses for acicular crystals, a few necrotic pus cells, while a careful and repeated search failed to show any amoebae in scrapings from the nearly smooth fibrous wall of the abscess. In the opened abscess living amoebae were easily seen in the third day, and at the recent abscess very numerous living amoebae were present, a number being seen in active motion in a single field of the microscope. The large intestine showed both recent scars of ulceration and also long transverse slit-like healing ulcers of a chronic amoeboid nature, but the rest of the large gut was free from ulceration, and the alimentary canal was otherwise healthy. The liver, showing a large abscess covered with a thick layer of fibrous tissue, was opened. A careful search for living amoebae in the walls of the abscesses and in the surrounding tissue was made, and I have failed to find any amoebae. The abscesses were opened in the course of the operation, giving rise to liver abscesses without any symptoms of previous dysentery. I think there can be no doubt that in this case the encysted abscess was the earliest, and that the abscess which had caused it had died out, although they still remained in the other two more recent abscesses.

**SEVEN CASES OF LIVER ABCESS OPERATED UPON BETWEEN JULY, 1904, AND JULY, 1905.**

By James Cantlie, M.B., F.R.C.S., Surgeon, Seamen's Hospital; Lecturer on Surgery, London School of Tropical Medicine.

For some years now I have made it a rule to report the cases of liver abscess I have treated during the preceding twelve months at this Section of the British Medical Association. Of the cases treated, 6 were tropical abscesses and the seventh developed in a man who had neither been out of England. In 2 of the tropical cases the liver pus had penetrated the diaphragm and destroyed the lower lobe; these patients died. Of the 4 tropical cases in which the pus was confined to the liver, 1 died of gangrene of the right half of the liver.

CASE 1. Intrahepatic Abscess: Operation: Recovery.—J. S., aged 59. Resided in Lagos since 1901. In good health until April, 1905, when he sustained an injury over right lower ribs. Whilst in hospital he developed fever, pain in right side, and profuse night sweats. On July 5th, 1905, at Seamen's Hospital, an exploratory puncture showed pus close to the surface; a large trocar and cannula was immediately introduced. After the abscess was drained, an abscess of the peritoneum and subphrenic space was opened. The abscess cavity contained 3 ozs. of pus which was drained off, and the abscess healing. The patient was discharged with complete recovery.

CASE 2. Suprahepatic Abscess: Operation: Recovery.—W. G., aged 27. Had lived in Rio de Janeiro for four and a half years. In February, 1903, had hepatitis, and subsequently had occasional attacks of fever.
with diarrhoea. On January 17th, 1905, soon after reaching England, was seized with rigors, fever, and watering. Dr. Hugh Knox suspected liver abscess and called for consultation with me. On January 20th and 21st, I found pus, 4 in. from the surface between the liver and diaphragm. A hepatic trocar and cannula was introduced in the midst of the left lobe, a large aspirating needle between the seventh and eighth ribs, a large cannula, with a tube was introduced and siphonage established. There was some difficulty and I left for France on Holiday, February 2th, which turned out quite well and left for India on March 4th. The pus drawn off at first was sterile. Dysentery was denoted, but attacks of diarrhoea following Hepatica.

Caso III. — Intrarehepatic Abscess: Operation: Recovery. — H. T. J. 42. In May 1904, had acute dysentery on board ship in Eastern waters. The dysentery developed on May 19th and was treated symptomatically with constipation with mild attacks of diarrhoea. In May 1904, he developed a cough with red-brown coloured expectoration. He was admitted to the hospital by Dr. B. S. Morgan, Shanghai Hospital, with all the signs of hepatic abscess and coughing up a bad-smelling trophistic mucus. Amenorrhoea was developed in the aputum. On April 3rd, trocar and cannula were introduced between the seventh and eighth ribs in mid-axillary line and drainage tube inserted. Patient continued to come up, but the temperature kept up at night. On April 6th, seventh rib resected and drainage tube inserted into lower seven inches. ASCENSION fell for some time but fever recurred. Exploration showed a huge cavity in the lower lobe of right lung. A counter-opening was made and the small abscess cavity in the left lobe of the liver, which was opened and drained, packed with gauze and sutured. Patient improved in health and was sent to Osborne House. One month later he died.

Caso IV. — Suprarehepatic Abscess: Destruction of Lung: Operation: Death. — M. H., aged 35, native of Calcutta. Admitted to the Seamen’s hospital for dysentery May 3rd, 1905. On May 7th developed pain in hepatic region and right and left upper quadrants. No elevation of temperature; leucocytes 40,000. May 19th, painless, with croupy and expectoration of trophistic mucus tinged with blood; leucocytes 40,000. May 24th, pus formed, and drainage tube introduced between ninth and tenth ribs, just in front of posterior axillary line; pus ceased to flow after six hours. May 29th, pus formed by needle aspiration between seventh and eighth ribs, anterior to diaphragm, drained-house on 30th; pus flew freely for twenty-four hours. As drainage seemed unsatisfactory and temperature rising, a in. of seventh rib resected, when on exploring the cavity was found the lower lobe of right lung completely broken down; a counter-opening was made behind and a thoracic tube inserted. The patient gradually lost strength and died June 7th.

Caso V. — Abdominal Abscess: Destruction of Kidney: Operation: Recovery. — Death. — S. Y., 35, native of Calcutta. Admitted to the Seamen’s Hospital for dysentery May 3rd, 1905. On May 7th developed pain in hepatic region and right and left upper quadrants. No elevation of temperature; leucocytes 40,000. May 19th, painless, with croupy and expectoration of trophistic mucus tinged with blood; leucocytes 40,000. May 24th, pus formed, and drainage tube introduced between ninth and tenth ribs, just in front of posterior axillary line; pus ceased to flow after six hours. May 29th, pus formed by needle aspiration between seventh and eighth ribs, anterior to diaphragm, drained-house on 30th; pus flew freely for twenty-four hours. As drainage seemed unsatisfactory and temperature rising, a in. of seventh rib resected, when on exploring the cavity was found the lower lobe of right lung completely broken down; a counter-opening was made behind and a thoracic tube inserted. The patient gradually lost strength and died June 7th.

Caso VI. — Suprarehepatic Abscess: Operation: Guncrene of Right Side of Liver: Death. — M. Y., 44, resided in India twenty years. Had dysentery some years previously. Had hepatitis in May 1905; invalided and sent to England. Last week in June liver abscess diagnosed by Mr. George Morgan, F.R.C.S., Brighten. On July 3rd pus found 4 in. from surface on dome of liver. The liver bled freely both when aspirating needle introduced into different parts of the right lobe of the liver between the ribs, and when the large trocar and cannula intro-duced. Siphonage established, but no pus flowed. On July 5th liver explored round seat of tube tract, but only sero-sanguineous fluid drawn off. In 24 hours pus formed and was sucking through tube and cannula. On July 30th pus difficult to suck; pus flown for 24 hours, but no pus produced. On following day (July 8th) patient died suddenly. Mr. Morgan was allowed only a partial post mortem examination. He found the right side of the liver distended, and the pelvis was distended and dangerous in part. Small loop of low pus were put in addition to the small abscess cavity in which the drainage tube originally introduced lay. This unique speciment is extremely impossible to preserve.

Caso VII. — Dynamic Abscess of Liver: Operation: Recovery. — D. M., 44. Had lived in England since birth. In May 1904, had all the symptoms of influenza, then temperature remained high for weeks. Dr. Gardner, in consultation with Dr. Mitchell Bruce, concluded that the lesion lay in the right lobe of the liver. On May 20th, after exploring the tract, I opened the abdomen and examined the liver. The liver pectuncles had bled freely into the cavity of the abdomen. The liver reached everywhere, except at the point where the suspensory (talofo) ligament of the liver is attached to the surface. There was no discoloration to indicate pus nor there any fluctuation; the absence of oedema and the unnatural hardness of the liver at that point reinforced the diagnosis. Not finding it possible to bring the part of the liver where pus was suspected to the incision in the abdominal wall, I closed the wound with sutures, and, after aspirating the tube, I left the wound as it was. The patient had a long illness; a wandering suppulsive peripatitis developed, and the wound in the abdominal wall never healed. After fourteen days, a small fistula was noticed on the original wound. Counter openings were made at two separate places, but the evening temperature remained high for eight weeks. The patient never continued to take food well, and he so far recovered that he was able to be moved to Newcastle. Recent reports are that he is doing well.

British Medical Association.

CLINICAL AND SCIENTIFIC PROCEEDINGS.

BIRMINGHAM BRANCH.

PATHOLOGICAL AND CLINICAL SECTION.

Birmingham, Friday, October 27th.

PROFESSOR J. W. TAYLOR, President, in the Chair.

SUCCESSFUL ENTERECTOMY FOR IRREVERSIBLE INTUSSUSCEPTION.—Mr. GEORGE HAYTON showed the path-ist and specimen from a case of irreducible intussception. The patient was admitted to hospital, and an irreducible intussception of the small intestine close to the ileo-caecal valve, and showed the usual appearances of a gangrenous focus. The patient died a few hours after admission. The abdomen was opened, and an enterectomy for intussception had been done, but who had been acutely ill for four days with vomiting, abdominal colic, absolute constipation, and passage of blood and mucus per rectum. The intussception was excised, and the two ends of the divided small intestine tied by 40 sutures. An anastomosis was then made between the lower end of the ileum and the ascending colon by a side-to-side approximation of the gut. The anastomotic opening was simple suture. The patient made an excellent recovery. For some time she suffered from occasional attacks of violent abdominal colic, but was now, some eight months after the operation, in perfect health. Mr. Heaton referred to the various operations in vogue when an intussception was found to be irreducible, and advised that whenever possible enterectomy should be performed on children over the age of 6 years. Under that age enterectomy seldom succeeded.

MALIGNANT ADENOMA OF UTERINE APPENDAGES.—Dr. THOMAS WILSON showed a malignant adenoma of the right uterine appendage the size of the fist. The tumour was lobulated on the surface, and was situated at the outer part of the broad ligament, and was joined by the right ovary, a considerable portion of which was distinct on the surface. The Fallopian tube gradually became fixed to the surface of the swelling. On microscopic examination the tubal lumen was distended, and at one limited part of the surface a mass of the growth was seen to be continuous with the epithelial lining. On the left side the meso-vascular and cysatic, and the tube closed and distended with semen. The body of the uterus was enlarged by several interstitial fibroids. The specimen was removed by total abdominal hysterectomy from a woman aged 50, whose one child was born eight years ago. There had been menorrhagia for one year, and slight pelvic discomfort for two or three months.

SARCOMA OF OVARY.—Dr. Wilson also showed a spindle-celled sarcoma of the left ovary removed from a patient aged 24, who had been married eight months and never pregnant. There was tense ascites and fluid in the peritoneal cavity. The tumour had accumulated very rapidly after repeated tappings. The tumour was removed under local anaesthesia, and the patient made a good recovery. The chest three weeks after operation remained dull, but there was a rapid accumulation of fluid the wound, and the left chest was not distended, and the patient had markedly improved.

SOUTH WARKS AND MONMOUTHSHIRE BRANCH: CARDIFF DIVISION.—At a meeting held at Cardiff on October 19th, Dr. C. VACHELL, the Chairman, who presided, delivered an address on the operation of enterectomy three years ago. Mr. WILLIAM SHIEN described the case of stone in the right ureter of a male adult. The stone was removed by incision through the wall of the ureter. The