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THE

Journal of Tropical Medicine

A MONTHLY JOURNAL DEVOTED TO MEDICAL, SURGICAL AND GYNÆCOLOGICAL WORK IN THE TROPICS

EDITED BY

JAMES CANTLIE, M.B., F.R.C.S., AND W. J. SIMPSON, M.D., F.R.C.P.

VOLUME I.

AUGUST 1898 TO JULY 1899

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AUGUST, 1898, to JULY, 1899.

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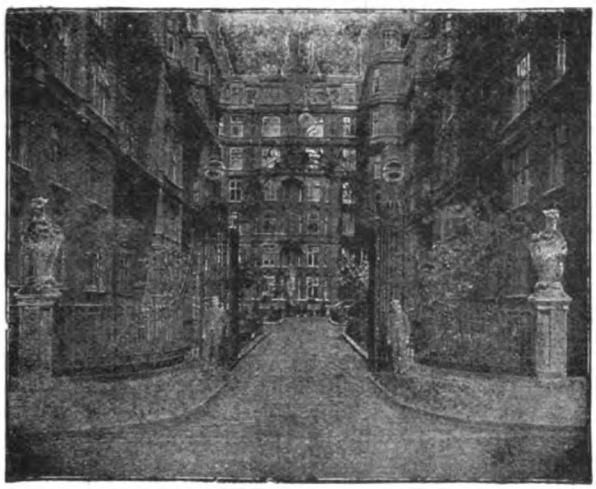

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Introductory Remarks.

By Sir Joseph Fayrer, Bart., K.C.S.I., LL.D., M.D., F.R.S.

The Journal of which this is the initial number is intended to, and it is hoped will, supply a want which has long existed in the medical journalism of this country. It is indeed a matter of surprise that so large and open a field of observation and experience should hitherto have remained unoccupied.

The object of the Journal is the consideration and discussion of tropical diseases and of questions of etiology, hygiene and preventive medicine, or any cognate scientific subjects affecting so large a part of our empire. It is not intended in any way to supplant or interfere with the numerous Medical Journals published in India or the Colonies, the merits of which are fully recognised; but by collecting in one focus the knowledge that has been acquired upon the above subjects, under such varying conditions of existtence and climate, will give wider circulation to the information they contain, and afford a broader basis upon which these subjects may be considered.

It has appeared to the originators of this Journal that it must necessarily be of service not only to the 6,000 or more qualified men who are practising in the various widely separated dependencies of the British Empire, but also to others who look forward to spending their professional life in tropical or sub-tropical regions. Those who are interested in the foundation of this Journal firmly believe that, if conducted upon broad principles, by men of experience in the subject upon which they are writing, and having personal knowledge of the climate and diseases of these countries, it must be of great practical utility not only to the profession, but to administrators and to the populations whose interests are so deeply concerned. The writer of these remarks, who himself has had considerable experience of tropical climates and tropical diseases, and who has always felt how valuable such a Journal would be, hails its appearance most cordially, and commends it to the consideration and support of the various Governments of our tropical dependencies and of his professional brethren, to whom are entrusted the health and well-being of such vast and scattered populations, for without such support it would be impossible that its aspirations, however well founded, could be realised. He is well assured that it has been committed to very competent hands, and that every confidence may be felt in the scientific and professional acumen with which it will be conducted.

The want of such a publication has been fully recognised; the materials spread over widely separated parts of the world are abundantly available, and need only to be gathered together and collated. The men who are competent and willing to do this work are forthcoming. It requires, therefore, only the sympathy and support of the medical profession generally, to give effect to a scheme which should commend itself upon so many grounds.

Announcement.

We have much pleasure in issuing the first number of the Journal of Tropical Medicine. The Journal will be devoted to the publication of papers on tropical disease, and to the discussion of subjects scientific and practical, affecting the interests of medical men in tropical and sub-tropical countries.

That no publication of the kind exists is a matter of surprise, when the vastness of the population, the variety of climate, and the scattered nature of the countries composing the British Empire are taken into consideration.

When the young medical practitioner first essays the diagnosis and treatment of disease in a tropical climate, he finds that his previous experience, gained in more temperate regions, is of but little use in fitting him to deal with the unfamiliar ailments he is sure to meet with in tropical practice. He begins to appreciate the fact that medical journals devoted to meet the general interests of the profession do not afford him sufficient guidance, and he will long for an opportunity of interchanging his newly-acquired experiences with others similarly situated to himself.

It is felt that many of the conclusions arrived at on the subject of tropical disease have been framed upon experience too local in its extent to be accepted as universally applicable. The opportunities for intercommunication, which would be afforded by a journal with the special aims herein indicated, would do away with this defect by giving a wider basis upon which to found conclusions and generalisations.

For a Journal with such aims a wide circulation is necessary, not only to cover expenses, but in order to secure men of high standing as contributors.

Being convinced of the necessity for such a Journal. and, as there are well-nigh 6,000 medical men holding British diplomas practising their profession outside the British Isles, a large proportion of whom, it was believed, would take a deep interest in the success of a Journal devoted to their more immediate professional wants, we issued a circular during the month of May, 1898, announcing the establishment of such a Journal. Already we have received most gratifying support, not only from subscribers, but from eminent men willing to contribute articles and information.

The Journal will appeal very specially to the medical officers of the naval, military, and colonial services, the medical missionaries and, most numerous of all, the general practitioners who, though following general practice, are really specialists in tropical diseases, can, from the wealth of their experience, supply material at once authoritative and interesting. Hitherto, from the want of such a Journal, too little has found its way into medical literature from the capable men in the public services, and the loss to medicine in all its branches is, in consequence, very great.

The editors have received cordial support from many influential men interested in tropical work, amongst others :—

Sir Joseph Fayer, Bart., K.C.S.I., LL.D., M.D., F.R.S.
Sir Henry F. Norbury, K.C.B., R.N., Director-General Naval Medical Department.
Sir James N. Dick, K.C.B., R.N. (late), Director-General Naval Medical Department.
Surgeon-Major-General J. Jameson, C.B., Director-General Army Medical Department.
Surgeon-Major-General W. Roe-Hooper, M.D., C.S.I., President Medical Board Indian Department.
Patrick Manson, M.D., LL.D., F.R.C.P., Medical Adviser to the Colonial Office, and to the Crown Agents for the Colonies.
Alexander Turnbull, M.D., R.N. (late), Inspector-General Royal Naval Hospital, Haslar.
Surgeon-Major-General Taylor, Army Medical Department.
Surg.-Col. Kenneth McLeod, M.D., LL.D., Professor of Military Medicine, Netley.
Surgeon-Major Andrew Duncan, M.D., F.R.C.S., Indian Medical Service.
Andrew Davidson, M.D., F.R.C.P.Edin., Colonial Medical Service (retired).

The late Sir William Mackinnon, K.C.B., formerly Director-General Army Medical Department, a few days before his death, wrote as follows :—"I should think it a very good and useful move, for the interests of the public and that of the medical profession, that a Journal of Tropical Medicine should be brought out in England; and, if it be well supported by medical men, now so numerously employed in all our tropical and semi-tropical colonies and stations throughout the world, it would add greatly to our knowledge of diseases and climates."

This communication is but a type of many received, and the Editors are aware that the same feeling pervades the general body of medical men resident in tropical and sub-tropical countries.

In addition to medical officers in the public services, and those interested in tropical work in Great Britain, the Journal is being subscribed to by medical men dwelling in :—

Europe.—Paris: Huelva (Spain): Gibraltar: Constantinople and Malta.

Asia.—India: Sidapur, Coorg: Bombay: Aligarh: Saugor, C.P.: Burkola, Cachar: Oorgaum, Mysore State: Calcutta: Travancore: Peshawur: Madras: Ranaghat: Midnapore.—*Assam:* Lakim Pur: Jorhat: South Sylhet.—*Burmah:* Mandalay.—*Ceylon:* Talawakele: Udugama and Colombo.—*Asia Minor:* Smyrna, Hebron, Marmarice.—*China:* Swatow: Shanghai.—*Straits Settlements:* Perak: Penang: Singapore.—Sumatra.

Africa.—Egypt: Cairo: Alexandria.—*Cape Colony:* Port Elizabeth: Graff Reinet: Barkley: West Namaqualand. — Kimberley. — *Orange Free State:* Vride.—*Transvaal:* Vryheid and Germiston.—Sierra Leone.—Accra, Gold Coast.—Grand Canary Islands.

America and the West Indies.—U.S.A.: Sullivan County, New York, Michigan. — *Jamaica:* Port Antonio, Kingston, Cross Keys, Claremont, Chapelton, May Pen: St. Lucia: Nevis.—*British Guiana:* Coriva, Demerara, Georgetown, Mahaica, Berbice, Trinidad: Belize, British Honduras.

A glance at this list will show how widespread is the interest evoked in, even the prospect of, a Journal devoted to the study of tropical disease. The Editors hope to be able to avail themselves of the valuable information which it will be possible to glean, and to conduct the Journal to the satisfaction of the subscribers. Already in Germany, France and Holland journals of a similar character are published, and for the honour of British science it is desirable that this country should not fall behind in a public duty, which is peculiarly its own.

We trust that our numerous subscribers will assist the interests of tropical medicine generally, by not

only contributing original communications and notes of cases, but also by forwarding reports from time to time of all important matters, whether of medical, surgical, gynæcological, therapeutic, epidemiological, epizootic, hygienic, meteorological, or pathological interest as met with in the tropics.

The Journal will be issued monthly with a letter-press of not less than 26 pages to commence with.

Communications to be addressed to

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Original Communications.

TROPICAL HEART.

By Surgeon-Colonel K. MACLEOD, M.D., LL.D.

Professor of Clinical and Military Medicine, Army Medical School, Netley.

DISEASES of the circulatory system constitute a considerable factor of sickness, mortality and invaliding in the British Army. They account for 1 per cent. of admissions, 5 per cent. of deaths and 10 per cent. of permanent invaliding. The proportion of cases per 1,000 of strength varies from 3 to 16 in different stations, of deaths from ·21 to 1·21, and of invaliding from ·75 to 3·56. Tropical and subtropical stations give higher figures than temperate. These numbers do not, however, represent strikingly the proportion of cases of heart failure causing incapacity, which occur in hot countries. This is better indicated by a study of particular cardiac affections. Thus the Report of the Sanitary Commissioner with the Government of India for the year 1895, shows that in a strength of 71,031 men, 336 were admitted into hospital for palpitation, 154 for valvular disease and 13 for hypertrophy. The deaths were 0, 11, 1, and the invalidings 42, 64, 4. Among 179 medical invalids recently received at Netley from India, no fewer than 35 were heart cases, or about 20 per cent. Of these 18 cases had been diagnosed as valvular disease of the heart, and 15 as disordered action of the heart, 1 as hypertrophy and 1 as endocarditis. In a large proportion of these cases considerable improvement of general health had taken place during the voyage home, and the murmurs, which had been taken as indicating diseased valves, had disappeared. These data bring out very clearly the fact which has long been known, that functional heart failure, without discoverable organic disease, is, among the adolescents and young adults who now constitute the bulk of the British Army, a common and serious cause of break-down, more especially in the tropics. I have carefully examined these 35 cases and find that none of them present aggravated symptoms of heart disease, a few of them give evidence of dilatation with or without hypertrophy, and many of them present modifications of the heart sounds of varying kinds and degrees, from simple accentuation to unmistakable bruit. One thing they all manifest without exception, and that is great excitability of action with or without exertion, the excited action being regular (in most cases) or irregular. These results agree with the careful observations of Brigade-Surgeon H. Veale, M.D., recorded in the Report of the Army Medical Department for the year 1880. He made a systematic examination of 100 cardiac invalids in the Netley Hospital and found that there was mitral abnormality without marked hypertrophy or dilatation in 3 instances; with ventricular dilatation, with or without hypertrophy, in 6 cases; dilatation without hypertrophy in 48 cases, in 2 of which the arch of the aorta was probably dilated; dilatation with hypertrophy, in 19 cases; and no evidence of either dilatation or hypertrophy in 24 cases. In 38 cases the heart sounds were normal and in a large proportion of the remainder mere alterations in tone existed. Murmurs were heard in 12 cases only. The evidence points to lameness of heart rather than damage; and as a matter of fact, with rest and proper treatment most of these lame hearts recover, though the tendency to give way under exciting circumstances, especially heat and strain, remains. The great majority of such cases are found to be unfit for military service and are permanently invalided. The history of them is that they "fall out" frequently on the march, or at drill or gymnasium, with palpitation, breathlessness, pallor or lividity, and sometimes actual fainting. They are sent to hospital, recover under rest, return to duty, fall out again more easily and frequently; oscillate for a time between hospital and duty, and finally have to be invalided for change or discharge, according to the severity of the case and the evidence of structural lesion.

On account of age, selection and elimination, heart disease ought to be rarer in the army than in civil life, but it is far otherwise. Maclean, Myer, Veale, Davy and Aitken have clearly demonstrated the large amount of heart failure in the army. At first improper clothing and heavy accoutrements were considered to impose, by pressure on the large vessels and fixation of the chest, an undue stress on immature hearts; but both clothing and accoutrements have been reformed, and still the bulk of heart cases has not been reduced. The fact is, that the strain of training and service, which is undergoing increase in these days of gymnasia, running, drill and manœuvres, acting upon a growing and unstable heart, constitutes the chief cause of disturbance. Sir William Aitken has shown that the heart does not attain maturity and stability till about the age of 25; and 60 per cent. of the soldiers of the British army are below that age. To this chief cause must be added excessive smoking and drinking, and perhaps debauchery. These are influences which operate in all climates. Residence and work in the tropics introduce a new and additional element of strain which acts perpetually and powerfully. There can be no doubt that a comparatively high temperature imposes exceptional work on the heart and circulation. The diminished proportion of oxygen in the air and the slower and shallower respiration necessitates for efficient oxygenation of the blood more active transmission through the lungs. Acclimatisation involves an altered blood distribution—a more

copious distribution to the skin and mucous surfaces for purposes of refrigeration. The blood heat is more easily raised above standard by exertion and chemical action, and more active arrangements, which are mainly vascular for dissipation, become necessary. In ordinary tropical life and in fevers more especially, violent alterations in blood distribution arise, and these impose new embarrassments and fresh labour on the central organ. These then are factors which make for weakening heart power. The heart is highly sensitive to thermic forces. Heat, exhaustion and syncope are the most acute expression of this; but a more chronic and abiding functional depression is also exercised by heat. High internal temperatures are apt to cause cloudy swelling and other degenerative changes, which further impair heart power. Under these circumstances, is it strange that the heart muscle loses tone and that dilatation of cavities, excitability and irregularity of action and positive failure under moderate strain arises?

Tropical heart is by no means limited to the army. It prevails largely among all Europeans residing in the tropics—witness the palpitation and dyspnœa which occur when they go to the hills, or undertake any unusual exertion.

The "Madras heart" is a well-known affection in the southern presidency, and it is by no means confined to the latitude of uninterrupted heat. It is found in acute and chronic forms through India. The lesion is precisely the same as the "disordered action," for which so many soldiers are sent home.

The moral to be drawn from these facts and considerations is, that in examining persons, especially adolescents, for tropical employment, very particular care should be exercised in taking stock of the condition of the heart and circulation. None but sound hearts are fit to encounter and withstand the strain which the new and strange environment imposes on both the central organ and vessels. A heart which is quite equal to the ordinary demands of civil life in a temperate climate, is apt, if at all faulty or lame, to succumb to the stress of circumstances met with in the tropics. Very specially and stringently does this caution apply to the selection of subjects for military service. This of itself, and even in temperate climates, causes disability, and may result in damage in hearts which present on examination every token of soundness. As regards structural unsoundness—positively damaged hearts—there can be no question. In justice to the service and humanity to the individual, these ought to be unhesitatingly rejected. The real difficulty arises in respect of these disturbed hearts—lame but not actually damaged—which are so often met with at, and after puberty. Excitability, irregularity, and even dilatation are met with, induced, perhaps, by excessive exercise, study, or smoking, which even give rise in some cases to modification of valvular accent—so called accentuation and functional murmurs. These hearts, under proper regimen, recover their tone and volume and stability when they are not subjected to a continuance or increase of the conditions which originally gave rise to the disorder. The history of the case and enquiry regarding the performance and "records" of the youth may suggest that the disturbance is temporary and insignificant. The heart has, however, acquired a bad habit, and if rest and regimen do not remove the evil habit within a reasonable time, the case is one for rejection—even if no diagnosis of structural damage has been made. The wreck of sound hearts which takes place in the army, especially in the tropics, is a serious matter, which demands not only care in selection, but indicates also continued solicitude in matters of training and drill.

AN EXPOSITION OF THE MOSQUITO-MALARIA THEORY AND ITS RECENT DEVELOPMENTS.[1]

By Dr. Patrick Manson, LL.D.

President of the Section of Tropical Diseases.

It was only last week that I received intimation that I might have to say something at this meeting of the Association about the more recent advances in our knowledge of the malaria parasite. Unfortunately I was ill, so that what between bad health and want of time I have had but little opportunity for preparation. This is my apology for what you will doubtless find is but a meagre and halting exposition and demonstration of perhaps one of the most important problems in the ætiology of tropical diseases—the life history of the malaria parasite outside the human body.

In order that those of my hearers who have not made a special study of malaria may understand what is novel in my remarks, it may be well that I first describe briefly certain well-known and thoroughly established facts about the life history of this parasite inside the human body.

If we examine the blood of a man just before the rigor stage of an ague attack, we shall find within a proportion of the blood corpuscles a number of pigmented discs of a pale protoplasmic material. As the stage of rigor approaches, the particles of pigment in these discs become concentrated, and the surrounding protoplasm arranges itself as a number of spherules. Presently the remains of the blood corpuscle which had hitherto enclosed this pigmented body, which is really the parasite of malaria, fall away, and the spherules disperse themselves in the liquor sanguinis. Many of them are absorbed by the leucocytes; a few of them, however, escape, and contrive to enter healthy blood corpuscles. Inside these the little body now becomes amœboid and active, and rapidly grows at the expense of the hæmoglobin, ultimately becoming the large pigmented disc I have already referred to.

Such, briefly, is the history in the human blood of all the varieties or species of malaria parasite. I should say rather of one phase of the parasite within the body, namely, that phase which subserves the reproduction of the parasite in the already invaded host.

In this, however, there is no arrangement obvious by which the parasite can escape from the human

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[1] A lecture delivered by request at Edinburgh during the British Medical Association Meeting.

body, a thing which analogy surely indicates as an essential element in the economy of all parasites.

If we examine malarial blood, we occasionally encounter a form of the parasite which does not seem to have any direct reference to the well-being of the organism while it is inside the human body. As a matter of fact, the phase of the parasite to which I allude does not come into existence until the blood has been removed for a considerable time from the body. If it has any purpose, therefore, that purpose must lie also outside the human body. This form of the malaria organism is known as the "flagellated body." It consists of a central pigmented sphere and a number of long arms which are in a state of continual and very vigorous movement. Occasionally some of these arms break away and swim free in the liquor sanguinis. Fixing my attention on the fact that these singular flagellated bodies come into existence only when the blood has left the human body, I came to the conclusion that in some way they subserve the interest of the parasite outside the human body. Also, fixing my attention on the fact that the malarial parasite exhibits no provision for spontaneously escaping from the human body, I came to the conclusion that it must get its chance of escape through some extraneous agency; and, for many reasons which it is not necessary for me to particularise on the present occasion, I came to the conclusion that this agency was the mosquito. Doubtless I was influenced towards this conclusion by my former experience of the mosquito as liberating agent of filaria sanguinis. Some have doubted my statements on this subject. Lest there be any doubters here, I have placed two slides under the microscope, showing the filaria at two different stages in the thoracic muscles of the mosquito, as a sort of object lesson of the capabilities of this insect as a transmitter of disease agencies.

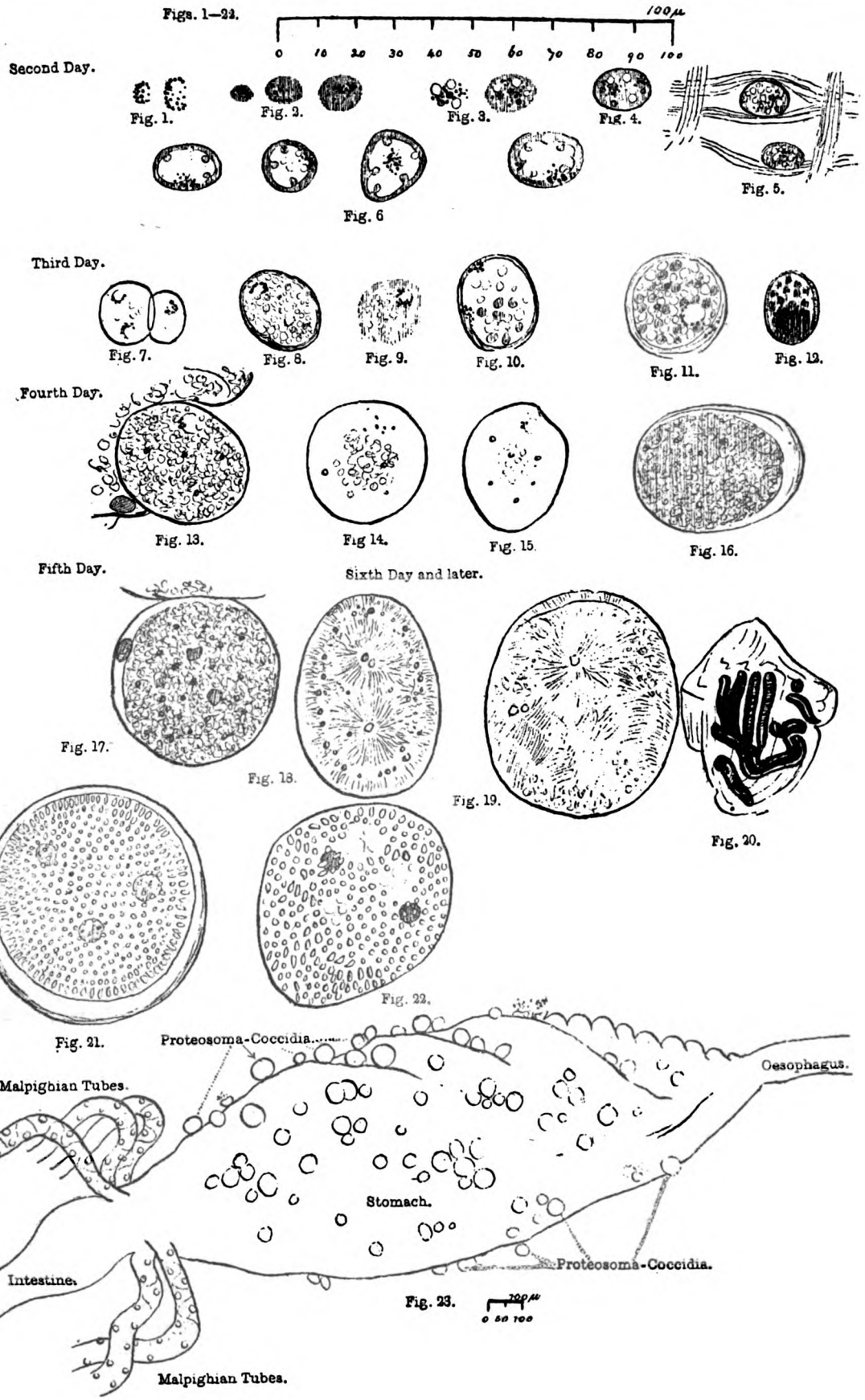
Well, I concluded that the mosquito sucked up, in its latent but potential state, with the malarial blood that form of the parasite destined for extra-corporeal life; that it became transformed in the stomach of the mosquito into the flagellated body alluded to; that the flagella broke away, and in virtue of their power of locomotion penetrated some tissue of the mosquito, and in this started the extra-corporeal phase of this important parasite. Such briefly, was my theory; we shall see now how recently observed facts have borne it out.

Circumstances made it impossible for me personally to work on the subject; but Surgeon-Major Ronald-Ross, who strongly believed in my doctrine, commenced some three years ago to test it by actual experiments and observations. He quickly found that at least one part of the theory conformed to facts. He found, and confirmed the finding by hundreds of observations, that the flagellated body was rapidly evolved in the mosquito's stomach, and that the flagella rapidly broke away from the central sphere, but what next became of the flagella, for the time being he completely failed to ascertain. Last year, however, he had an opportunity of experimenting with a species of mosquito which he had not hitherto worked with, what he calls the "dapple-winged" mosquito. Four of these insects he fed on a patient whose blood contained many parasites of the species associated with that form of fever which has been called summer-autumn fever. On examining their stomachs in from two to four days after feeding, he came across bodies incorporated in the walls of the stomach of a character which he had never before encountered in any of the hundreds of mosquitos' dissections which he had previously made. He saw a number of oval bodies dotted over with black pigment—pigment which was optically indistinguishable from that which is so distinctive of the malaria parasite as seen in the blood. Ross at once recognised the importance of this find. Here, however, fact and theory seemed to diverge. It was difficult to reconcile the presence of pigment in these bodies with the absence of pigment in the flagella, which, according to my hypothesis, are the infective agencies. The pigment always remains in the central sphere, or what may be regarded as the residual portion of the flagellated body; it does not enter the flagella. How then account for it in Ross's bodies? Fortunately at this juncture help came from America which apparently reconciled theory and fact. MacCallum, of Johns Hopkins University, observed that in halteridium, an intra-corpuscular parasite of birds closely allied to the malaria parasite of man, the purpose of the free flagellum is to impregnate certain halteridia, and, as it were, prepare them to enter on a new phase of existence. In watching slides of blood containing halteridium, he observed parasites escape from the blood corpuscles and assume a spherical form. Certain of these spheres emitted flagella which, breaking away, accumulated about other spheres which did not emit flagella, and finally entered them. Whereupon, after a time, the impregnated spheres changed shape and acquired locomotive powers; became, in fact, travelling vermicules, containing the entire substance of the original halteridium sphere, including its pigment. They exhibited great powers of penetration, passing indifferently through red blood corpuscles and white blood corpuscles, and moving with freedom and activity about the field. Depend upon it, this locomotive penetrating power exhibited by the pigmented halteridium vermicule has a purpose. Observe that it comes into existence only after the blood containing it has left the body of the bird. What more likely, then, than that this purpose is the attainment and penetration of the walls of the stomach of some special kind of mosquito that has ingested it? It is pigmented. Hence, arguing from analogy, I would suggest the pigment in Ross's pigmented bodies.

This explanation, I think, is admissible; although it cannot be said that absolute proof is as yet forthcoming.

Later Ross had an opportunity of dissecting a mosquito of a different species, a grey mosquito, which he found feeding on a case of ordinary tertian fever. In this insect likewise he found in the stomach wall similar pigmented bodies.

The Government of India now took Ross by the hand, relieved him of his military duties, and placed him in a well-equipped laboratory in Calcutta, with instructions to work at the mosquito-malaria theory and to report quarterly on the progress of his investigations. I hold in my hand the report on his first


Figs. 1—23.
100μ
0 10 20 30 40 50 60 70 80 90 100
Second Day.
Fig. 1.
Fig. 2.
Fig. 3.
Fig. 4.
Fig. 5.
Fig. 6
Third Day.
Fig. 7.
Fig. 8.
Fig. 9.
Fig. 10.
Fig. 11.
Fig. 12.
Fourth Day.
Fig. 13.
Fig 14.
Fig. 15.
Fig. 16.
Fifth Day.
Sixth Day and later.
Fig. 17.
Fig. 18.
Fig. 19.
Fig. 20.
Fig. 21.
Fig. 22.
Proteosoma-Coccidia.....
Malpighian Tubes.
Oesophagus.
Stomach.
Proteosoma-Coccidia.
Intestine.
Malpighian Tubes.
Fig. 23.
200μ
0 50 100

three months' work, and when I tell you the story set forth in its contents and in the private correspondence which I have had with Ross, I am sure you will agree with me that he has opened up a field of enquiry which cannot fail to yield scientific and practical results, sooner or later, of the utmost importance to science and, I would add, to humanity. When Ross arrived in Calcutta it was not the fever season; consequently he had difficulty in procuring patients for experimental purposes. Not to waste time, he took up the study of what may be called the malaria of birds. You are doubtless aware that in warmer climates birds are exceedingly liable to a variety of protozoal blood diseases. They have intra-corpuscular parasites almost identical with those of man. Two forms especially have been described and named. One form Labbé calls halteridium, the other proteosoma. Though closely allied, they are manifestly different species. Halteridium, when mature, extends alongside the nucleus of the blood corpuscle, forming at either end two bunches of spores; proteosoma, on the other hand, is a more condensed parasite, so to speak, obtaining room for development by displacing laterally the nucleus of the corpuscle. Both possess flagellated forms; both consist of a colourless protoplasm containing grains of black pigment; both form rosette bodies just as the human malarial parasite. There can be no question, therefore, of their close affinity to the latter.

Circumstances led Ross to study more particularly the relationship of the mosquito to proteosoma. He found that the common grey mosquito, after it had fed on the blood of birds infected with proteosoma, nearly invariably exhibited pigmented bodies almost identical with those he had seen in dappled-winged mosquitos fed on the blood of summer-autumn malaria. It is unnecessary to enter into detail about the large number of experiments which Ross performed in order to establish incontrovertibly this fact. Suffice it to say that out of an aggregate of 245 grey mosquitos fed on birds with proteosoma, 178, or 72 per cent., contained pigmented cells. Of 249 insects fed on men and birds unaffected with proteosoma, not one contained a single pigmented cell. One set of experiments is particularly convincing. Ross obtained a number of mosquitos from larvæ caught at the same time in the same drain. Ten of these mosquitos he fed on a sparrow with numerous proteosoma in its blood; on subsequent dissection and examination, on an average the stomach of each mosquito was found to contain 101 pigmented cells. Ten of the mosquitos he fed on a sparrow with a milder proteosoma infection; in these the average number of pigmented cells amounted to 29 only. Ten of the insects he fed on a sparrow in whose blood there were no proteosoma; in none of these insects did he find a single pigmented cell. I myself subsequently examined these same preparations, and can confirm the substantial accuracy of Ross's statement.

I could cite additional and equally convincing experiments, all tending to show that it is only from blood containing the proteosoma that the pigmented cell in the grey mosquito can be obtained. Fed on human blood with summer-autumn malarial infection, on bird blood containing halteridium only, or fed on healthy blood, the stomach of the grey mosquito never contains a single pigmented cell. Manifestly, therefore, this cell is derived from proteosoma, and we are driven to the conclusion that it is an evolutionary stage of this parasite.

When he had thoroughly established this fact, Ross, with characteristic energy and skill, set to work to follow up the development of this interesting body. In the first place, he determined its exact location. If the stomach of a mosquito is examined microscopically, on tracing the layers from without inwards, we come first on the branching air vessels which ramify on the external surface of the organ; next there is a layer of longitudinal and transverse muscular fibres crossing each other at right angles and forming a coarse quadrangular pattern; next there is a homogeneous structureless layer; and lastly, and forming the inner coating of the stomach, we have several layers of what may be called epithelial cells.

Now it is not in these epithelial cells that the pigmented body lies; it is located either on the outer surface of the homogeneous membrane referred to, or in the muscular layer. And you will find in some of the preparations under the microscope that in many instances the fibres of the muscular layer are separated by the parasites just as we see the fibres of the muscles of the pig separated by the embryo of trichina spiralis. Ross further found that as the parasite grew to its full size—some 40 to 70μ in diameter—it gradually protruded from the external surface of the stomach, so that by the fifth or sixth day after feeding the infected viscus, seen through a low power of the microscope, appeared to be covered with so many warts. Of this you will see an excellent illustration under the microscope.

The very early stage of the parasite in the stomach wall—that is to say, during the first twenty-four hours—Ross has not studied; but about the second day it appears as an oval body, 7 by 6μ, with sharply defined outline and clear contents, through which some twenty granules of black pigment are scattered.

Later on, in certain of the parasites, vacuoles and granules appear. Others preserve a more hyaline structure. Later still the outline of the proteosoma coccidium, as Ross calls it, becomes more circular, the black pigment completely or almost completely disappears, the contents become more granular, and indefinite appearances, which may be segmentation, are in some instances dimly distinguishable.

Ross seems to think that there are two types of these bodies: one characterised by granularity, one by a hyaline structure; and in yet others he finds peculiar black sausage-shaped bodies whose significance he cannot interpret. In a few of the larger bodies he made out a distinct concentric arrangement of the granules giving an appearance of pattern, recalling what we sometimes see in diatoms. For a time, Ross could get no further with the study of these bodies. He observed, however, that about the sixth or seventh day many of the coccidia-like bodies had collapsed—an empty capsule, doubtless representing the external wall of the parasite, alone remaining.

When the report which I hold in my hand was sent in, and for permission to use which I am indebted to the Secretary of State for India, this was virtually the sum of his investigations.

But from private letters since received I gather

that a further and most important advance has been made, and that Ross has triumphantly concluded at least one part of the problem he had set himself to solve.

He had already observed in many of the insects which he dissected, scattered throughout the tissues and in the body cavity, a number of extremely minute, somewhat flattened, spindle-shaped, trypanosoma-like bodies. He now made the significant discovery that these bodies had escaped from mature proteosoma coccidia.

By mounting the stomach of the insect in salt solution, and compressing it under the cover glass, he caused the larger parasites to rupture and give forth an innumerable swarm of these spindle-shaped, germinal rods, as he calls them. Undoubtedly, then, what happens in nature is this: when the large coccidia mature and protrude from the surface of the stomach they rupture and discharge their contents, the germinal rods, into the general body cavity. Although these germinal rods are not distinctly motile, somehow they manage to diffuse themselves throughout the entire insect. Ross contends they do so by entering the blood-vessels, for he found that by pricking the dorsum of the thorax of an infected mosquito, and examining with the microscope the white fluid, or blood, which exudes, myriads of these germinal rods could be detected.

And now comes the most curious and interesting of all his observations. While dissecting, in search of these germinal rods, the head of an infected insect, he came across a gland composed of clear cells arranged along a branching duct. Within many of these cells he found, snugly lodged and in prodigious numbers, the spindle-shaped, germinal rods. By careful dissection he found that the duct of this gland or glands—for he found two—communicated with the proboscis of the mosquito. Doubtless they are the glands from which is derived the secretion which the insect instils in making its bite, and which is after a time the cause of the irritation many of us are so familiar with as the unpleasant sequel of a visit from this insect. This discovery suggested that the germinal rod might be extruded in this excretion, and that in this way it might be the—or a—means of conveying infection.

To test this idea Ross fed mosquitos on a proteosoma-infected sparrow. After an interval, and when he considered that the germinal rods had been formed and had arrived at the venemo-salivary gland, he got these mosquitos to bite birds free from proteosoma infection. Later, on examination, he found that the bitten birds had become infected, and that their blood contained proteosoma.

I am sorry I cannot give full details of this important experiment. The information I received by telegram, and naturally one is brief when each word costs shillings to transmit; but I have the utmost confidence in Ross's judgment and veracity, and I now believe that malarial infection may be conveyed by insect bites. I do not say it is the only or the ordinary way, but I feel fairly sure that it is one way.

These observations and experiments of Ross, elaborate and interesting though they be, by no means exhaust the subject. They do not tell us in what way the malaria parasite multiplies outside the human body, and independently of man or other warm-blooded animal.

It is yet to be shown how mosquito infects mosquito. Indeed it may be that in the mosquito the parasite is only on its way to some other medium, in which it can multiply, and from which infection is diffused.

It may be objected that what holds good for proteosoma may not hold good for *plasmodium malariæ*, but the similarity of the parasites is so great that one cannot resist the conclusion that their histories are also similar. Moreover, Ross has distinctly shown, as already mentioned, that certain species of mosquito do elaborate pigmented cells when fed on malarial blood.

I am sure you will agree with me that the medical world, I might even say humanity, is extremely indebted to Surgeon-Major Ross for what he has already done, and I am sure you will agree with me that every encouragement and assistance should be given to so hard-working, so intelligent and so successful an investigator, to continue his work. His observations all tend to the conclusion that the malaria parasite is really a parasite of insects; that it is only an accidental visitor to man; that not all mosquitos are capable of subserving it; that particular species of malaria parasites demand particular species of mosquito; that in this circumstance we have at least a partial explanation of the apparent vagaries of the distribution of the varieties of malaria. It seems to me that when the whole story has been completed, as it surely will be at no distant date, that, in virtue of the new knowledge, we may be enabled to indicate a prophylaxis of a practical character, and which might enable the European to live in climates now rendered deadly by malaria. Other practical issues will occur to anyone who thinks of the importance of an accurate knowledge of the life history of this as of any other parasite.

DESCRIPTION OF THE PLATE.

Drawings of proteosoma-coccidia from the second to the twelfth day.
Figures 1—6, coccidia of the second day.
Figures 7—12, coccidia of the third day.
Figures 13, 14, 15, 16, coccidia of the fourth day.
Figure 17, coccidium of the fifth day.
Figures 18—22, coccidia of the sixth day, and later.
Figure 23, drawing in outline of the stomach of a mosquito studded with proteosoma-coccidia of the sixth day, seen by a low power.

AN Association, having for its aims the prevention of the spread of tuberculosis, has been formed in London. It is supported by Sir Samuel Wilks, Bart., President of the Royal College of Physicians, London; Sir William MacCormac, Bart., President of the Royal College of Surgeons; with Sir William Broadbent, Bart., as Chairman of the Provisional Committee, and Mr. Malcolm Morris as hon. treasurer. Under such able guidance this laudable scheme should have a great future of usefulness, and be of infinite benefit to the health of the Empire.

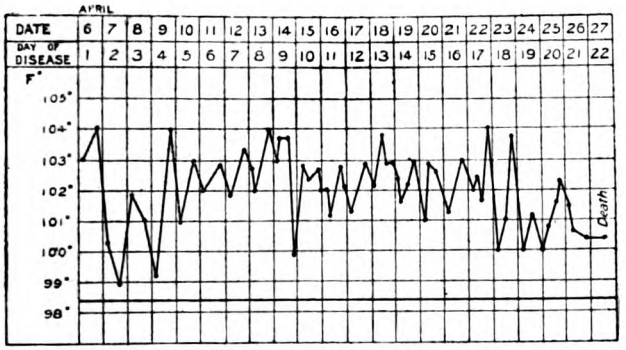
EPIDEMIC CEREBRO-SPINAL FEVER IN INDIA: WITH THREE CASES.[1]

By Surg.-Capt. W. J. Buchanan, B.A., M.B., B.Ch., Dip. St. Med.

Superintendent Central Jail, Midnapur, Bengal.

Though cerebo-spinal fever has no claim to be considered as a strict tropical disease, yet the fact of its existence in tropical countries, *e.g.*, India, is not as well known as it should be.

Having recently had the opportunity of seeing four cases of this disease, and having, as I believe, been able to demonstrate the existence of the *diplococcus intra-cellularis* of Weichselbaum in the pus cells taken from the spinal membranes of one fatal case, I think it worth while to record the same and give a brief account of this disease as it has been met with in



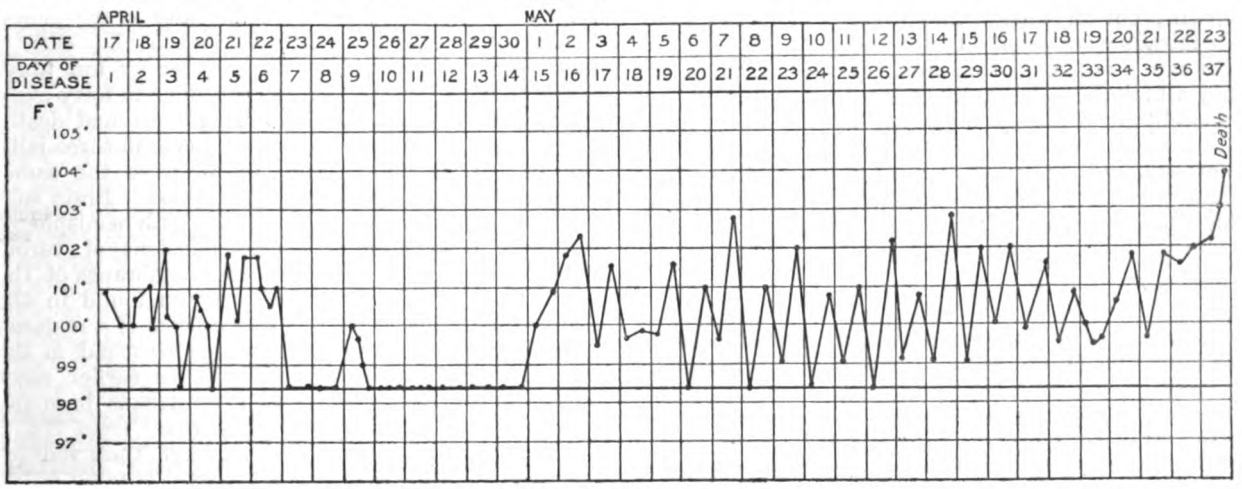
India, the more especially as the publication of Councilman's exhaustive and valuable report on the recent Boston epidemic has again attracted attention to the disease.

I have recently seen three cases in the Central Jail, Buxar, and one case in the Central Jail at Midnapur; the notes of three cases are given, those of the fourth non-fatal case having been mislaid.

Case 1.—Prisoner Kanoo, age 21, admitted to Jail Hospital, Buxar, on April 6, 1898, from ward 19, with high fever 103° F., pain in neck and back, constipation, severe headache and redness of eyes. Pulse on second day was noted as "slow and regular," with a temperature of 102° F. On another occasion pulse 66, with 102·4° F., pupils normal, no paralysis. Later on marked retraction of the head, and slight strabismus of left eye; bowels generally moved once daily. Towards the end diarrhœa. Abdomen noted as rigid. Pulse varied. Temperature, taken mostly four times daily, ranged from 101° to 104° F. Delirium set in fifth day, then coma, alternated with partial or complete consciousness. Died on twenty-second day of illness at 3 a.m. Retention of urine during last three days.

Post-mortem Examination (after five hours). Sinuses full of dark fluid blood. A large amount of yellow purulent exudation over membranes of both brain and spinal cord. Turbid fluid in the lateral ventricles. Old dysenteric ulceration in the lower portion of the large intestine.

Case 2.—Prisoner Mukwa, age 24, admitted to Jail Hospital, Buxar, on April 17, 1898, from ward 20. Fever 100° F., great restlessness. Is quite unconscious. The attack came on very suddenly without any premonitory symptoms, patient having been at work the evening before. Had clonic convulsions of arms and legs shortly after admission to the hospital ward. Constipation. Retention of urine. When conscious, skin was hyperæsthetic. Great pain in head and nape of neck. No hepatic nor splenic enlargement. Eyes and pupils normal. On the sixth night the temperature fell to normal, and patient asked for food, which he ate (sago and milk). He remained certainly free from fever till the ninth day, when the temperature rose to 100·2°, falling again to normal before evening. After this it remained certainly normal for four and a-half days, during which period his condition improved and he was thought to be convalescent, but on fourteenth night temperature rose again, the pain, semi-unconsciousness and delirium returned. The pyrexia continued remittent or intermittent till the thirty-seventh day, when he died, with the thermometer marking 103° F. The pain in the head and neck are noted as "unbearable." Perspirations were noted three or four times, but not in connection with any remission of the fever. Retention of urine throughout the case. Urine 1015. sp. gr. No sugar, no albumen. The



[1] Read at the Annual Meeting of the Brit. Med. Association.

pulse bore no regular relation to the temperature. During the last few days he lay in a curved position on one side, generally the right. He died on the thirty-seventh day of illness and twenty-third of the relapse.

Post-mortem Examination (two hours after).—Yellowish white exudation over the membranes of the brain and spinal cord. A large collection of pus at base of brain. No bone disease, no ear disease. Other organs normal.

For the above notes I am indebted to Assistant-Surgeon Baroda Kanta Ray, who was in charge of both cases throughout.

Case 3.—In Midnapur Central Jail, Prisoner Luchman Paria, age 25. Previous health bad (spleen), but had improved since arrival in jail two months previously. Came to hospital complaining of fever (101·2° F.) and pain in head and neck. Was suddenly attacked while at work under cover in a weaving shed. No cough, no sputum; spleen much enlarged. Had severe convulsions on second day, and died on morning of third day in hospital after having a second (or possibly third) convulsive attack. No eye symptoms. Patient was unconscious nearly whole time in hospital.

Post-mortem (within one hour).—The spinal cord and brain membranes were covered with thin yellow lymph. The brain substance was intensely congested. The lungs were also affected. Right lung full of red blood, but in centre of left lung was a mass of hepatised tissue (size of an orange) distinctly marked off by its deep red colour and solidity from the surrounding tissues. Other organs normal, except the old malarial spleen.

Bacteriological Examination.—I at once prepared slides of the purulent matter from brain and spinal membranes, staining with a watery solution of gentian violet. Six slides were prepared. In all I found a diplococcus within the pus cells answering in all particulars to that of the *diplococcus intra-cellularis* of Weischelbaum. In some slides only a few were present, but in others they were abundantly seen. No tetrads were seen. I also inoculated three agar-agar tubes with some thin fluid from spinal canal, but they all failed to show any growth except one, which, on twelfth day, showed numerous small white colonies all over the surface. These I tried to inoculate on another agar tube, but they failed to grow. Agar-agar I find is inferior to blood serum for the cultivation of this diplococcus, and often fails. On another occasion I will try lumbar puncture.

History of Cerebro-Spinal Fever in India.

This must next be sketched as far as available records permit. The most complete accounts of this disease in India are to be found in Surgeon-Lieutenant-Colonel Moorhead's paper in the "Transactions of the First Indian Medical Congress, 1894,"[1] and in the "Official Report[2] to Government of the Committee appointed in 1885." The disease has chiefly been noted in the large central jails of Bengal, and under the strict conditions of jail life such cases cannot well be overlooked, and their early and previous medical history is usually obtainable; moreover, except in jails *post-mortem* examinations are difficult to obtain in India. Next to the jails we find the disease most commonly recorded in emigrant ships trading with the West Indies and in emigration Coolie depôts in Calcutta. Up to the present year cases are constantly recorded in such ships and depôts. Occasionally we hear of a case in private practice, but a very few have been recorded. (See below.)

The following is a brief account of the disease as it has existed in the prisons of Bengal for the past dozen years.[1] These notes show the progress of the disease and the extremely erratic nature of its distribution and spread.

1885.—The Jail Report for this year notes the first known occurrence of the disease in Bengal jails in some detail. In the large Central Jail at Alipur (Calcutta) there were 39 cases with 16 deaths. Most of these were met with in the cold weather. In October, 1884, there were 15 cases with 6 deaths, 12 cases and 3 deaths in November, 12 cases and 7 deaths in December. The disease then disappeared for two months, but 2 more cases are reported in March, 1885.

Sir A. Lethbridge, M.D., the Inspector-General, quotes the following conclusions as having been arrived at:—(1) that the disease is cerebro-spinal fever; (2) similar cases have been met in the Presidency Jail, Calcutta, and at the General Hospital in the Burdwan Jail (70 miles from Calcutta), and in the Mayo Native Hospital, Calcutta. A few cases are mentioned as having been met with in private practice, and two emigrant ships are reported as having been affected, in one 18 cases were met with during the months of August and September, *i.e.*, immediately before it was recognised in the Alipore Jail. (3) No eruption was noted (all the cases were in Natives) except urticaria in one case. Herpes is not noted. (4) The symptoms were those of "remittent fever," with extreme pain along the spine and in the joints. Temperature was irregular, 102°—105° F., with a daily range of 1 to 1·5° F. In three or four days delirium, which lasted from twenty-four to forty-eight hours, and was usually followed by coma and death. (5) *Post-mortem* examinations were made in three jails, and the appearances found in all were the same. Vessels of *dura mater* full of dark blood. Brain substance much congested. Lymph over both hemispheres and over base. In all cases effusions of turbid puriform lymph was found on the membranes of the spinal cord, and in one case pus was found in the internal ear. (6) There was no evidence of a malarial origin except that the earlier cases occurred in the usual autumnal fever season. (7) The earlier cases came from sleeping wards and worksheds in many different parts of the jail. Later cases from No. 5 and No. 7 worksheds chiefly. Though their ventilation needed improvement, yet these weaving sheds (jute) had been long in use, and no such cases had ever before been noted. (8) It was noted that strong, sturdy men were as much attacked as the sick and weakly.

[1] Thacker Spink and Co. Calcutta, 1894.
[2] Report dated April 22, 1886. Calcutta.

[1] Taken from the "Bengal Jail Administrations Reports," 1885 to 1897.

1886.—Eighteen cases in Alipur Jail from January to March 12, with 4 deaths, 1 in April, 3 in June, and 2 in December—the 6 latter all fatal. The extraordinary rapidity of the onset of death is noted: 1 case died in ten hours, 1 in one day, 2 after two days, 1 after four days, and 1 after five days' illness. Cases came from all parts of the jail. Old and young, long or short term prisoners, all seemed equally liable to attack. In consequence of the Committee's recommendations, improvements in ventilation were carried out.

1887.—The disease continued in a quasi-epidemic form in Alipur in spite of the sanitary improvements: 13 cases, with 8 deaths. None in neighbouring Presidency Jail, and none in the General Hospital, but 10 cases were reported from the emigration depôts. One jail warder (residing outside) was attacked, and 1 case in private practice is noted.

1888.—This year we first meet with recorded cases in jails outside of Calcutta. Alipur, however, continues affected: 8 cases with 5 deaths at intervals throughout the year. At the Rajshaye Central Jail, a long distance from Calcutta, 4 cases were met in one week, December 20—27, with 3 deaths. The 3 fatal cases had only recently (14 days before) arrived on transfer from a neighbouring district jail (Dinajpur), where no such cases were reported, but this same Dinajpur had 3 cerebro-spinal cases recorded in 1890.

Dr. Russell (now Professor of Materia Medica, Medical College, Calcutta) reported carefully on the Rajshaye cases. He also noted the occurrence of rapidly fatal (two to seven days') cases in the town, which were, however, called "remittent fever." One case was also reported from Mymensingh Jail, and 1 "typho-malarial" fever case from Rungpur Jail.

1889.—In this year we have 28 cases and 19 deaths in all jails: Alipur, 8 with 4 deaths; Rajshaye, 4 with 3 deaths; Hazaribagh, 14 cases with 11 deaths; Mymensingh, 1 fatal case; Jalpaiguri, 1 fatal. Except at Hazaribagh, all these cases occurred at irregular intervals. In the latter jail there were two outbreaks; in both instances the disease appeared and disappeared suddenly. The first lasted from May 16 till June 5; the second from October 8 to November 5. There were 6 deaths in the first set and 5 in the second. Dr. Moorhead[1] notes that there was not even an approach to overcrowding at this period, there being only 188 prisoners with accommodation for 1,000, and the affected wards were not filled. The jail is situated on a plateau 2,000 ft. above sea level, and the sanitary condition was the same before, after, and during the epidemic, and was good.

1890.—Only 2 jails reported cases. Alipur again 8 cases and 6 deaths throughout the year. The Dinajpur jail had 3 cases.

1891.—In all jails 23 cases with 18 deaths. Alipur again heads the list with 12 cases, 11 of which were fatal. One jail is freshly affected—Bhagulpur Central—6 cases, 5 deaths. The small jail at Rangpur had 5 cases, 3 deaths.

1892.—A falling-off, only 9 cases, 6 fatal, in all Bengal. Alipur was still affected: 6 cases, 4 fatal. Bhagulpur 1 and Jalpaiguri 1.

[1] *Op. cit.*, p. 144.

1893.—Only 3 cases in all Bengal jails; 2 at Alipur and 1 at Buxar Central Jail, all 3 cases fatal.

1894.—This was a most unhealthy (malarious) year, but there was only 1 case of cerebro-spinal fever, and that fatal, at Alipur.

1895.—Four cases and 1 death. Three (1 fatal) at Arrah Jail and one at Barrisal. None in Alipur, for the first time for ten years.

1896.—No cases reported.

1897.—Report not yet published, but cases heard of in Bhagalpur Central Jail.

1898.—Only 3 cases at Buxar Jail known to the present writer and 1 in Midnapur, above detailed.

We find, therefore, that this disease persisted for close on a dozen years in an irregular way in the jails of Bengal. In Alipur cases occurred for ten successive years. Out of the 49 central and district jails in Bengal only nine were attacked, but only three at all severely.

Cerebro-Spinal Fever in other parts of India.

Apart from Bengal I can find but little record of this disease, Dr. Moorhead[1] quotes from the "Sanitary Commissioner's (India) Report for 1892" that from 1881, when the disease first appears in statistical reports, there had been recorded in all India 333 cases and 259 deaths, or an average of only 27 cases a year. Though Bengal jails have apparently suffered most, yet those in other provinces have not escaped entirely. On the Bombay side Shikarpur Jail has had several outbreaks. So also the Central Jail at Lahore. Cases have also been reported from Madras, Burma, Andaman, and the Nicobar Islands, so that Hirsch's statement (*apud* C. Allbutt's system) that the southern distribution of the disease is limited by the 30° N. latitude is certainly wrong. This line would exclude all India except North Punjab. Latitude 5° or 10° N. is more probably correct.

In an appendix to the Jail Committee's report, Dr. C. H. Joubert, F.R.C.S., gives details of a number of undoubted cerebro-spinal fever cases in emigrant ships trading from Calcutta to Demerara, and to Trinadad. The earliest cases (recognised as this fever) were seen in July, 1884, a few months before the disease was recognised in the Hipur Jail. Could Calcutta have become infected by means of returned emigrants from the West Indies? The coolie depôts in Calcutta were certainly affected during 1884. In May, 1886, Dr. Sanders read a paper with one illustrative case, and stated that the disease had been for two years epidemic in Calcutta and the neighbouring districts, and in the discussion at the Calcutta Medical Society Drs. Harvey, O'Brien, and M'Leod agreed as to the existence of the disease, though it was seldom recognised or at any rate "returned" under this name. I can find no records of the disease in the North-west Provinces, but it was long ago recognised in Lahore (Punjab), certainly in 1881 and following years (before its recognition in Calcutta). At Skikarpar (Scinde) Dr. H. P. Dimmock was apparently the first to recognise the disease in the Bombay Presidency. In 1883 he had 38 cases with 27 deaths. He notes that spasms of the muscles and opisthotonos were frequently seen. Another recorded outbreak was in

[1] *Op. cit.*

the extra-mural jail at Chinawar, on left bank of River Chenab, Punjab. This was in 1885; there were 14 cases, of which 10 died. They occurred in December, January and February (1885-6), during which months "bitterly cold north winds prevail." All prisoners were at work on canal digging, and all exposed. Dr. Shand (in Committee's Report) gives details of symptoms seen. No eruption was observed except herpes, which was often seen. In 1885 at Kohat on the North-west Frontier 5 cases were reported in the 2nd Punjab Infantry.

Dr. H. Vandyke Carter had a case in Bombay in September, 1885, which he records in detail (*Indian Medical Gazette*, December 1886). He also notes that the case described at p. 436 of his "Spirillum Fever" was really cerebro-spinal fever. This case was met in 1878. He notes that many such cases have occurred in Bombay "contemporary with and subsequently to the late famine" (1885). In the somewhat old stained specimens he examined he could find no micrococci, and in this connection we may note that Dr. D. D. Cunningham records that he found no special organism in the cases seen by the Calcutta Jail Committee. Dr. V. Carter also notes the slowness of the pulse.

Symptomatology.

To decide upon the identity of the disease in India with that reported in other (European and American) countries it is necessary also to compare the symptoms, &c. Having been fortunate enough to receive a copy of Dr. Councilman's report (State Board of Health, Massachusetts: Boston, 1898), I shall contrast the symptoms and signs therein recorded with those of cases reported by Dr. Moorhead and others in India, and from my own experience.

Councilman recognises two practical types of the disease: (1) the acute, including "foudrayant" cases; and (2) the chronic. Cases under fifteen days are acute; the chronic cases may run on for two or even three months. Both, or, including fulminant, cases, all these types are well known in India.

The temperature is very irregular, at first continuous and afterwards, if case survives, more "remittent." Moorhead notes that the terminal rise was absent in his 12 fatal cases. Bauer, in 1867, noted that fulminant cases were more common at the beginning of an outbreak. This was the case also in Alipur Jail. Genuine intermission of temperature is not noted in any Indian case except in mine quoted above. The "remittent" type of the pyrexia has often been commented upon, and accounts for the fact that such cases are sometimes overlooked or buried in the *cloaca maxima* of the "remittent." I can only find the above-quoted case of a relapse.

Vomiting, only present in 2 out of Moorhead's 16 cases; I have found it in 2 out of 3 successive cases, not, however, as an initial symptom. It was noted in 60 per cent. of the Boston cases. It is usually noted upon as of central origin, but Moorhead noted it with flatulent symptoms.

Delirium.—This is common, in more than half Moorhead's cases. In one case it began ten hours after onset, in others in first few days. It was never violent. It occurred in more than half the Boston cases. It often alternated with periods of consciousness.

Pain.—Generally early complained of in the Indian cases. Moorhead describes it as "excruciating," the Boston report as "agonising." It is chiefly in head, or in neck, or in back, and in a few cases in joints.

Neck Symptoms.—Retraction of neck and pain are commonly looked upon as pathognomonic, but they are not always present. Distinct retraction was only noted in 3 out of 16 cases (Moorhead). Symptoms referred to the neck were only absent in 28 out of the 111 Boston cases. In most cases simple stiffness will be found. Stiffness and retraction were most marked in the 3 last cases I saw.

Coma.—In 10 out of 12 cases (Moorhead). It lasted sixty-two hours in 1 case, which recovered. May be early; within three hours in 1 case, generally it follows delirium.

Opisthotonos was absent in all Moorhead's cases, but is mentioned by Dimmock. Moorhead noted rigidity of abdominal muscles, but no paralyses except of bladder (2 cases retention of urine). In my experience retention is more common (2 out of 4 cases). "Paralyses," says the Boston Report, "are not uncommon."

Skin eruptions are always rare in India. Urticaria in 1 jail case. No petechiæ noted in dark-skinned Indian cases except by Dr. Sanders. Herpes in many cases, but this is very common also in India after even malarial attacks. It was the most common eruption in the Boston cases—35 out of 111; petechiæ in only 11. Petechiæ were very marked in the Dublin outbreak in 1866.

Special Senses.—No eye complications noted in any Indian cases except by Dr. Sanders, though they are very common in Continental and American reports. I saw squint in only 1 case. Hearing impaired in 1 of 3 cases seen by me, also in 1 case of Moorhead. Pus in internal ear in 1 Jail Committee's case.

Joints.—Noted in many Indian cases "pains in the joints"; no further details. Acute synovitis in 6 of 111 Boston cases.

Blood.—No record in any Indian cases. Leucocytosis always present in the Boston cases. The inequality of temperature and pulse, common in Boston, is specially recorded in several Indian cases. The sudden change from slow to very rapid pulse is also well known in plague cases.

Constipation is most common in the Indian cases. A feebleness of *memory* after recovery is noted by Moorhead in 1 case. Pericarditis,[1] with effusion, in 10 out of 12 fatal cases (Moorhead), but in only 2 of the Boston cases.

The condition of the lungs in cerebro-spinal fever is of the utmost importance, both from a clinical and from an ætiological point of view. In 2 fatal cases I have recently seen the lungs were quite normal. In another affected (*v.* above). Moorhead gives details of the lungs in his 12 fatal cases. In 2 fatal cases (which lasted over twenty days) the lungs were intensely bloodless. In 5 nothing abnormal was detected. In 5 there were distinct morbid changes. In 1 the whole right lung was hepatised, and the left lung intensely congested. In a second, the same condi-

[1] I have noted pericarditis to be decidedly common in fatal cases of croupous pneumonia in India.

tion, with recent pleurisy. In a third case there was recent pleural lymph, and a part of one lung hepatised. In the fifth the left lung was only congested. In the Boston cases congestion was found in 13, in 7 broncho-pneumonia, in only 2 croupous pneumonia with fibrinous pleurisy (pneumococci were found). In 8 cases pneumonia due to the diplococcus was found. It is said that the characteristic diplococcus-pneumonia consists of small foci of consolidation and necrosis in various parts of the lungs, but especially in the lower lobe and under the pleura. In some cases these areas coalesced so as to resemble croupous pneumonia. "Lung complications due to the diplococcus may take place at any period of the disease."

Spleen.—Not often or much affected. In one of Moorhead's cases the enlargement was known to have been of (previous) malarial origin (see Case 3, also *supra*).

Ætiology.

A review of the history of the disease shows many guesses and suppositions, but little definite fact. The attempt to connect the disease with overcrowding and with bad ventilation altogether failed in Calcutta. Hot and cold weather have little influence. Food grains have been suspected by the late Sir W. B. Richardson, but no evidence has ever been produced. The rice used in Alipur Jail was certified as free from any disease by Dr. D. D. Cunningham. One fact stands out clear in the history of the disease in every country, viz., the tendency to attack bodies of men, whether collected in regiments, jails, coolie depôts, or emigrant ships. It is, however, probable that this is partly because of the care and attention bestowed on the diagnosis and registration of disease among such bodies of men.

The disease never becomes epidemic in the sense that cholera or plague are epidemic. A few isolated cases or a series of two or three cases at a time in any particular place is the usual history. Connection can seldom be traced from one case to another. The disease in Bengal jails has always been treated as if contagious, but no evidence pointing to infection is forthcoming. Its persistence in Alipur Jail for ten successive years is very remarkable. In considering the question of contagion, if we admit it to be a specific febrile disease it is of necessity in some way communicable, but with the organism shut up as it were in the closed cavity of the brain and spinal cord, it could not well escape except by the lungs or nose, and as the diplococcus has been found both in the nose and lungs, it is not improbable that it is by means of cases in which these organs are affected that the spread of the disease takes place. This point, at least, is worth paying attention to in future outbreaks.

Two diseases, cerebro-spinal fever and Malta fever, have thus been rescued from the *cloaca maxima* of the remittents as seen in tropical countries. That yet another as yet undifferentiated fever of a continuous type exists is my firm belief, but so far the evidence in favour of this view is too indefinite to do more than allude to the belief here.

That the disease here called cerebro-spinal fever (as in Nomenclature of Disease) is identical with the epidemic cerebro-spinal meningitis of Continental and American writers will be clear from above account. The diagnosis of isolated sporadic cases will always be doubtful without bacteriological examination.

The other forms of meningitis are—(1) Pneumococcic, due to pneumococcus, and met with rarely in acute croupous pneumonia cases; (2) Streptococcic, due to septic ear disease, erysipelas, &c.; (3) Tubercular, so common in children; (4) due to any other infective agency, *e.g.*, anthrax.

Midnapur, Bengal.

FILARIÆ AND FILARIAL DISEASE IN BRITISH GUIANA.[1]

By C. W. Daniels, M.B.
British Guiana Medical Service.

As little English work on these subjects has been published for British Guiana, it may be of interest to compare our experiences with those of workers in other countries, and to record such points as seem to be peculiar to this Colony.

(1) *The Filarial Origin of Elephantiasis.*

To most workers in the tropics the evidence of the dependence of elephantiasis on filarial infection by the Filaria nocturna seems so conclusive that the need for any further confirmation may seem superfluous, but as the subjoined quotations show that doubts are still expressed it may be useful to give such evidence bearing on the point as I have been able to obtain from local sources. Hutchinson (*Archives of Surgery*, January, 1893, p. 279) states that "although in tropical patients the filaria is commonly found in the blood of elephantiasis patients, yet, as it is absent in cases seen in temperate climates which are exactly the same in character, it is impossible to assign to it the rank of a true cause." Legrain (*Archives de Parasitologie*, January, 1898, p. 151) states: "La question de savoir si l'éléphantiasis est sous la dépendance de la Filaire est encore fort controversée; De Brun admet la co-existence fréquente, mais sans relation de cause à effet. Dans plusieurs cas d'éléphantiasis dont j'ai examiné le sang je n'ai pas rencontré de Filaires."

One of the more important arguments for the view that elephantiasis is due to the Filaria nocturna is based on the fact that the two have the same geographical distribution; in countries where the inhabitants commonly harbour this filaria elephantiasis is frequent, and the converse is equally true, viz., where elephantiasis is common the filaria is also frequently met with. As will be shown, the rare exceptions to the converse do not invalidate the argument.

Thornhill in Ceylon has strengthened this argument by showing that the rule also applies to the *local* distribution of the disease in that country, as in districts where elephantiasis is met with the filaria is common, whilst where it is not found neither is the filaria.

Similarly, in British Guiana the races inhabiting the coast lands are the only ones amongst whom elephantiasis is found, and amongst these the filaria is common, whereas the aboriginal inhabitants of the

[1] Paper read at the meeting of the British Medical Association, Edinburgh.

interior are free from the disease, and in no case has the Filaria nocturna been found in their blood.

Secondly, how far does *racial* incidence support or negative the causal relation of the filaria to elephantiasis? Experience in this colony has long shown that even amongst the coast inhabitants some races are more commonly affected than others, and that females are more often attacked than males. Dr. Grieve statistically confirmed this popular belief by an analysis of the cases in the Berbice Lunatic Asylum in 1883. This showed that 6 per cent. of the male East Indians, 4·6 per cent. of the male negroes, and 7·2 per cent. of the female negroes had elephantoid legs. The Portuguese and mixed races were attacked in a still larger proportion, but the numbers of these races were small. In 1895 Dr. Conyers and myself determined the incidence of the disease amongst the inmates of the Public Hospital, Georgetown, and this year I have continued the determination so as to have figures sufficiently large to minimise the probability of error. The following table (Table I.) shows the result obtained.

West Indian negroes are immigrants from the West Indies, the majority from Barbadoes, many of whom have been long residents in British Guiana. East Indians are the Coolie immigrants from India; none resident under five years are included, and the majority have been here much longer. Aboriginals are the aboriginal Indians of South America.

The table shows clearly that whilst some races are rarely attacked, in others elephantiasis is common, and that among the natives the women are more frequently attacked.

This unequal incidence of elephantiasis amongst the races has been used as an argument against the probability of the filaria being the true cause of the disease. It was considered that people living under such similar conditions would all be equally exposed to the attacks of the parasites, and that therefore the racial element *per se* must be an important factor. A determination, however, of the proportion of persons harbouring these parasites, no selection of cases being made, shows that the proportion of infected persons also varies amongst

TABLE I.

Race	British Guiana Negro		West Indian Negro		Coloured		E. Indians		Aboriginals		Portuguese	
Sex	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
No. of cases	343	260	196	56	141	90	463	142	Many	Many	99	51
No. with elephantiasis	43	40	15	3	21	25	5	2	0	0	25	20
Percentage with elephantiasis	12·5	15·3	7·6	5·3	14·8	27·7	1·0	1·4	0	0	25·2	39·2

TABLE II.

Race	British Guiana Negro		West Indian Negro		Coloured		East Indians		Aboriginals		Portuguese	
Sex	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
No. of cases	120	108	63	39	49	42	108	84	Over 50	Over 20	68	50
No. with filaria nocturna	23	34	7	5	10	14	7	5	0	0	16	16
Percentage with filaria nocturna	19·1	31·4	11·1	12·8	20·4	38·6	6·4	5·9	0	0	23·5	32

Tables I. and II. show the incidence of elephantiasis and of infection by the Filaria nocturna respectively in the more important classes of inhabitants of British Guiana.

The proportion of elephantiasis is no doubt above that for the general population, as though cases of elephantiasis are very rarely admitted for that alone, still on account of the liability of these to ulcers, abscesses, pyrexia, &c., it causes indirectly their admission. This, however, does not affect the reliability of these observations as to the relative incidence in the various races.

The division into races requires some explanation. British Guiana negroes are those born in British Guiana. "Coloured" are those in whom the European admixture is very evident. The distinction is arbitrary and is made as it is the popular opinion that the latter are more liable to elephantiasis than those of purer descent. This opinion is supported by my tables. the different races, and moreover that the relative incidence varies in the same manner as the incidence of elephantiasis.

Thus we find that a study of the race incidence of the filaria and of elephantiasis goes only to strengthen the view of the dependence of the one on the other.

The annexed chart and Table III. show more graphically the relation between the proportional incidence of filariasis and elephantiasis in the races here represented. In it the somewhat superficial distinctions of colour and birthplace are omitted, and it will be seen at a glance how close is the correspondence.

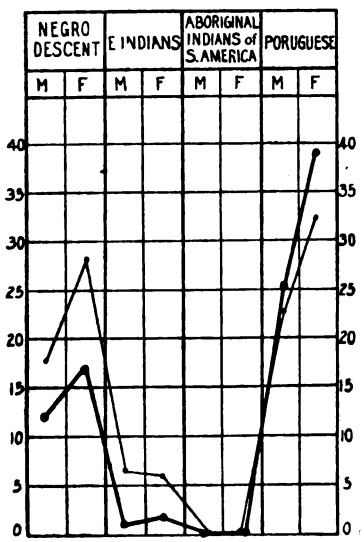
Though not bearing directly on the main argument of the essential cause of elephantiasis, it will be convenient here to consider the meaning of this startling

double incidence among races apparently in most essential respects living under the same conditions. It is not to be contended that race *per se* protects from invasion by these parasites, as it is well known that no race is immune, and that the East Indians can be, under favourable conditions, frequently attacked. We have therefore to inquire what difference in the racial habits is responsible for the different proportion of persons who are attacked living under the same climatic conditions.

TABLE III.

Race	Negro Descent		E. Indians		Aboriginals		Portuguese	
Sex	M.	F.	M.	F.	M.	F.	M.	F.
Percentage with elephantiasis	11·6	16·7	1·	1·4	0	0	25·2	39·2
Percentage with filaria nocturna	17·2	28·0	6·4	5·9	0	0	23·5	32
No. of cases examined for elephantiasis	680	406	463	142	Many	Many	99	51
No. of cases examined for filaria	232	189	108	84	Over 50	Over 20	68	50

The inquiry is rendered simple, as the life-history of the F. Bancrofti has been so fully worked out, and we know that for human infection it is requisite that the person attacked must have drunk water in which has been deposited the body of the intermediate host, a certain mosquito, which in its turn has fed on the embryo-laden blood of the original host, an individual harbouring the parent worm.



—— Percentage with elephantiasis.
—— Percentage with Filaria nocturna.

Now, the Aboriginals are under entirely different conditions to the other races. Amongst them there are no infected hosts, mosquitos are rare, and perhaps not of a species capable of serving that purpose, and they use running water from the creeks and never store it.

The East Indians, who form the great majority of the hospital patients, live sufficiently amongst the other races for the conditions as regards the primary host to be much the same, and the intermediate hosts must be the same. The water they drink is however little likely to be the recipient of the bodies of the mosquitos, as it is either pipe or the slowly running creek water. The East Indian rarely takes the trouble to collect rain water, and does not go to the expense of storing it. The opportunities he has of acquiring the parasites are therefore restricted.

The Negro and Coloured races have a very decided preference for rain water, and dislike the taste and smell of the peaty creek and pipe water. If vats are not available, they catch the rain water and store it in casks close to and sometimes inside their houses. The amount of trouble they will take varies, but whilst some will buy rain water when their own supply falls short, others will use the pipe or creek water, though even then they often store it for days in order to allow the sediment to deposit. Thus the opportunities for infection of their drinking water are numerous.

The Portuguese are, as a rule, in a rather better position, are still more careful and particular in their storage of water, and live a more domesticated life, and therefore have still more special opportunities of imbibing the immature parasite.

Females, as a rule, live more at home than the males, and in a like manner have increased risks.

An analysis of the source of the drinking water used by 54 unselected cases in whose blood filaria had been found, will show how favourable the conditions are for the acquisition of the parasites. Of these 54, in 2 no clear history was obtainable, 2 expressed no preference, as they used either creek or rain water indifferently. The remaining 50 all used rain water when they could possibly obtain it, and all but 13 denied taking pipe or creek water at all or for many years (forty, thirty, twenty-five in three cases). Of the 13 who admitted using pipe or creek water, 6 state that they always leave it to settle in a cask for days before using it.

The method of storage of the water is important. When provided with vats in some cases, if not close to the house, the water is again stored in casks, when not so provided, the rain water is caught from the roof and stored in casks, or in dry weather bought or borrowed from the more fortunate neighbours and similarly stored. These casks are at the most only roughly cleaned and are never allowed to dry, "as it would spoil the cask." They are usually kept close to and sometimes inside the house, and are rarely covered up. Mosquitos, therefore, have ready access,

and in most cases the water is said to contain numerous mosquito larvæ. The people have no objection to them unless very numerous; some state that they "purify the water." The water is never boiled.

In 30 out of the 52 cases the water was stored in these casks, in 11 of them the casks were kept inside the house, and in others close to it.

A similar analysis of the water supply of 10 persons with elephantiasis, in whose blood no filariæ were found, gave the following results, showing that they also had lived under conditions which had rendered them liable to acquire the worms. They all preferred rain water; 2 had at times to use creek water, and 1 of these always allowed it to settle in a cask inside the house; 6 used water stored in casks, in 3 of these the cask was kept inside their own house, and in a fourth, an East Indian, inside a neighbour's house.

From a study, then, of the liability of the races to infection and their conditions of life in relation to the life-history of the parasite, it would appear that this varying incidence of infection among the different races depended on (1) the presence of infected primary hosts, (2) suitable intermediate hosts, (3) a water supply in which *storage in small* receptacles allows of the ready deposit and concentration of the intermediate hosts with their embryonic contents.

To return to the original argument; there are two facts, which are not disputed, urged against the acceptance of the filaria as the true cause of elephantiasis.

(1) That elephantiasis, indisguishable from that met with in countries where the filaria is found, is, though very rarely, seen in England and other temperate climates. It is not requisite to believe that the filaria is the only agent capable of causing lymphatic obstruction—in fact we know that it is not. Hutchinson states that when lupus affects the extremities it causes elephantiasis indistinguishable from tropical elephantiasis. Similarly in this colony the granuloma pudendum, when it involves the penis, scrotum or vulva, produces an elephantoid condition quite irrespective of, and indistinguishable from, that produced by the filaria.

(2) That in many cases of tropical elephantiasis no filariæ are to be found in the blood. This fact is fully admitted; our experience here is in accordance with that of Manson and others elsewhere, that the statement does not go far enough, as it is found that the proportion of cases harbouring filariæ is smaller among elephantiasis patients than among the general population, and that in whatever manner they cause lymphatic obstruction such a result would be expected as if the parent worm did not die after setting up the obstruction and its embryos would not have access to the circulation. In the Portuguese, indeed, a larger proportion have elephantiasis than harbour filariæ.

(2) *The Pathology of the Lymphatic Obstruction.*

Probably the obstruction may be caused in many ways. The first, which is probably only of importance when the larger lymphatic vessels are involved, is by the parent worm causing hæmorrhage into the lymphatic channels. That this can occur is shown by a case here in which the parent worms were found in dilated lymphatics filled with recent blood clot. The history is as follows: On July 1, 1896, the patient, a male negro, aged 35, after a busy day, was seized with a sudden pain in the left groin, and a swelling appeared there which rapidly increased in size and became more painful. He was admitted into hospital the following day and was found to have a movable, tense tumour in the inguinal canal about the size of a small egg. An ice-bag was applied, but the swelling did not diminish, though the pain did. There was no fever. On the tenth day an exploratory incision was made over the tumour. It was found to be an ovoid tumour obviously containing blood, with a broad neck passing up into the abdomen. This was tied as high as possible and the tumour removed below the ligature. On opening it it was seen to consist of a mass of thin-walled channels distended with recent partly organised blood clot, in which a dozen or so adult worms or portions of them were found, living and in active movement. In this case the adult worms had in some way excited hæmorrhage into the lymphatics, forming an organised clot, which would effectually block the lymphatic channels.

In another similar tumour, removed seven weeks after its first appearance, no worms were found and the clot was almost decolorised and organised. In this case there was little pain with the onset.

In several *post-mortem* examinations of patients with dilated lymphatics in the pelvis, the point of obstruction has been found and appeared to be due to an organised coagulum in the thoracic duct.

We have no evidence as to the frequency with which the occurrence of hæmorrhage takes place. It would only be indicated by pain when sufficient in amount to cause tension, or by a tumour when in such a position as to be visible.

A second and as far as clinical evidence goes a commoner method of obstruction is by the parasites setting up inflammation of the lymphatic channels. These attacks are common in patients with filariæ, are accompanied by fever and ague, and are frequently excited by some trivial cause. They usually recur, sometimes frequently, and in many cases pass on to elephantiasis. The majority of cases of elephantiasis give a history of such attacks before the elephantiasis developed, but this method cannot be held accountable for the large minority, as these come on without such antecedent attacks of lymphangitis and without fever.

Neither of the above methods satisfactorily explains the cases in situations where there is free collateral lymph circulation, and particularly those where a small region only is involved.

The theory advanced by Manson gives a much more satisfactory explanation. He supposes that under certain conditions the female aborts, and, instead of discharging embryos, discharges ova into the lymph stream. The smallest diameter of these is three times that of the uncoiled embryo. These ova would, therefore, block channels through whlch the embryo could pass, and the process continuing would in turn block every path of exit for the lymph. Manson, in support of this, records that he has on several occasions found ova in the fluid from chyloceles and elephantoid tissues. The narrow tubular orifice of the genital organs of the parental worm, just sufficient for the embryo to pass, would seem to be designed to prevent such an acci-

dent, but these observations show that that safeguard occasionally fails. I have had one opportunity of repeating Manson's observations under circumstances that render it improbable that the ovum had escaped from a dead or mutilated worm. The patient in whom I had, in 1896, found the parental worms, early this year presented himself for operation on a right inguinal hernia of many years' standing. In the course of the operation a mass of moderately dilated lymphatics, with one small cyst, was found. These tissues were removed. On examination the cyst, which was about half the size of a pigeon's egg, was isolated, and still remained tense. It had clear transparent walls. The fluid contained was quite clear, and there were no remnants of parental worms. Microscopic examination showed, in addition to the ordinary filarial embryos, one coiled embryo in an egg. There had, in this case, been no symptoms of lymphangitis, no pain, and no fever, whilst the fact of its being found in a closed sac renders its escape from a dead worm improbable. No adult worms were found in the surrounding tissues.

There is thus evidence (1) that the parental worm can cause obstruction by exciting hæmorrhage; or (2) by exciting frequent attacks of lymphangitis; or (3) that the embryos may be discharged into the lymph stream still in their egg capsules, in which case from their size they will be stopped at the next lymphatic glands, and thus cause obstruction. In the first two cases the worms would be included in the blockage and would either die, or if they escaped below the obstruction their embryos could not reach the blood circulation. In the third case the parent worms would always be below the points of obstruction. It follows that filariæ will be frequently absent in the blood of persons in whom lymphatic obstruction has resulted from the filaria, even when the adult worms are still living.

The Incidence of the Forms of Filarial Disease.

The regions of the body attacked are much the same as in other countries where elephantiasis is common. Out of 137 cases, in 80 the legs, in 51 the glands alone, and in 6 the genital organs were attacked. This is the order given by Manson for China. The most important difference is, that chyluria and hæmaturia are rather more common, as they are met with quite as often as scrotal elephantiasis, whilst they are much rarer in China. In Fiji, chyluria was certainly very rare, and in a much larger proportion of the cases the genital organs were attacked. One form, consisting of the formation of pendulous elephantoid masses, weighing several pounds, dependent from Scarpa's triangle, is found in Fiji in the proportion of, I estimate, about 1 to 10 cases of scrotal elephantiasis. I have seen four cases of these tumours in British Guiana, but it is very rare. In China it does not seem to have been met with. Much importance cannot be attributed to these small differences in regional distribution, which are probably due to some difference in the habits of the inhabitants.

The Embryonic and Adult Forms of the Filaridæ.

The parental form, the F. Bancrofti, and its embryo, the F. nocturna, have been indentified in India, China, Queensland, and in British Guiana, and, as the filaria was not indigenous in America, but was introduced with the negroes from Africa, we can assert the complete identification of the parasites for all elephantoid regions but Polynesia, where the embryos only have been observed.

Except among the Aborigines and a few other persons who have resided in the interior, I have found no other form but the F. nocturna in the cases I have examined. I have not found the Filaria Demarquayi in any of the West Indians, including six from St. Vincent.

The aboriginals who inhabit the interior of British Guiana, harbour one or two species of filariæ. They were found by Manson and described by him at the Montreal meeting of the Association, and were named by him the Filaria Ozzardi. Dr. Ozzard and myself have in all examined over 250 persons, and find that these parasites are present in 58 per cent. of the cases examined. We have found them in persons from all parts of the interior, including a few living on or near the Dutch, Brazilian, and Venezuelan frontiers, so that they are probably widely distributed through Central South America. There are reasons for believing that the distribution is not uniform. Dr. Ozanne informs me that in some villages or families all the individuals have them, and in others none. In bloods sent me great differences were observed. Out of one lot of 13, in all but 1 I found filariæ, in two lots of 8 and 6 respectively none were found. They seem to be equally common in the two sexes, and are found in children. Dr. Ozanne informs me that he found them in a child 2 years of age, but has not found them in children in arms. Drs. Ozzard and Ozanne have found them in negroes living in the interior, and I have found them in one negro and one Portuguese, who had lived many years in the interior, and had lived with and under similar conditions to the aboriginals. I have not found any cases amongst a large number of gold-diggers who have lived a few months in the interior.

As was pointed out in Manson's paper (*Brit. Med. Jour.*, December 15th, 1897) some of these filariæ were sharped tailed, and others blunt. All are shorter than the F. nocturna, and when fresh measure from ·2 to ·25 mm., and much thinner, rarely exceeding ·0046 mm. as against ·32 mm. by ·007 for the Filaria nocturna. They have no loose sheath, display no periodicity in their habits, exhibit locomotory as well as wriggling movements. The V-spots are smaller and less easy to see. The cephalic movements are more active, and there is an appearance of the protrusion of a retractile spike from the head. A similar appearance in the adult worm is seen with low powers, and in that case is due to an optical delusion from the rapid change of the head from a globular to a more cylindrical form and consequent refractive effects.

The question as to whether or no these two forms are distinct species is not yet definitely settled. The blunt-tailed are much more common, but in the 18 cases under my observation if a sufficient number of slides were examined one or more sharp-tailed specimens were found. On the other hand, the relative proportions of the two forms vary greatly in different cases, whilst not differing greatly in different examina-

tions of the same case. The commonest proportion is one sharp to from six to twelve blunts. The smallest proportion where both were found was one sharp to fifty-six blunts. In the specimens of blood sent to me, all single slides, it was common to find only blunt-tailed embryos, when the number present was small, but not when it was large. In two cases sharp-tailed embryos alone were present; in one only one embryo was found, but in the other there were sixteen. In another case there were nine sharps to one blunt.

Both forms vary much in size according to the mode of preparation, the blunt the more so. The sharps are slightly longer and in their broadest part thicker. When stained the nuclei are found to extend to the top of the tail in the blunts, whilst in the sharps the finely-pointed tail extends for a considerable distance beyond the last nucleus. In the living specimens the sharp tail is more difficult to find or to keep under observation, as its movements are so rapid. Its tail remains sharp and the worm does not appear to elongate and contract in the marked manner that the blunt tail does.

If these two forms represent distinct species the blunt-tailed may be the same as the African F. perstans, as it resembles it in size, shape, structure and movements, as well as in the absence of periodicity. Of this embryo the adult form was found in two cases. It was found in both cases in the upper part of the mesentery, and in the second case also in the subpericardial fat. They appeared to be in the connective tissues, not encysted, and are very difficult to remove entire. They are difficult to see, but are more readily found if a watchmaker's glass be used and the tissues slowly dissected. They are a little shorter, but little more than half the thickness of the F. Bancrofti, which they resemble in the general anatomical features (*vide Brit. Med. Jour.*, April 16th, 1898). The embryo *in situ* can hardly be distinguished, but in all cases where they were allowed to escape they were found to be blunt tailed, and no sharp tails were found, so that this must be considered as the parental form of the blunt tails at least. No other parental forms were found and the examination was sufficiently thorough to render it probable that none were present in any part of the visceral cavities or organs, the cavities of the heart, the larger blood vessels or the thoracic duct.

If the two forms represent one species it is not the African F. perstans, as that is never sharp tailed. We must also assume that in some of the embryos some further development takes place either before or when they reach the peripheral circulation, and that this change occurs more readily in some individuals than in others, as such differences as sixteen sharps without any blunts and fifty-six blunts to one sharp cannot be otherwise explained.

If the sharp-tailed embryos represent another species it has yet to be decided whether it is a new one or the F. Demarquai.

So far we have no evidence of any pathological *rôle* played by these parasites, and it is difficult to predict what form of disease would be likely to be caused by parasites in the position in which these are found. Most of the hosts, even when the parasites are numerous, have been in good health; in the others there was no disease that could be attributed to the parasites. None of the cases had elephantiasis or any signs of lymphatic obstruction. In several cases under observation for months there were no cutaneous eruptions or anything resembling the African "Kra, Kra," nor did the patients show any signs of lethargy. On the other hand our knowledge of the diseases of the Aboriginals is very small.

Nothing is known of the life-history of the parasites. The embryo do not leave their host in any of the natural discharges—at least they have not been found in any of them, though repeated examinations have been made. Dr. Ozanne found them in one case in the pus from an abscess in the arm. It seems probable that they are removed by some blood suctorial insect, but certainly a different one to the carrier of the F. nocturna, and probably not a mosquito, as the drinking water used is not likely to be contaminated by that insect.

ARMY MEDICAL SERVICE.—The designation of the substantive ranks of the Officers on the Retired List liable to be recalled to service to be as follows:—

Brigade Surgeon Lieutenant Colonels to be Lieutenant Colonels.
Surgeon Lieutenant Colonels to be Lieutenant Colonels.
Surgeon Majors to be Majors.
Surgeon Captains to be Captains.
Surgeon Lieutenants to be Lieutenants.

The designation of those retired officers who, though not liable on account of age to be recalled under the provisions of Article 486c. Royal Warrant, April 26, 1897, have been, or shall be, in military employment on or after June 23, 1898, to be as follows:—

Brigade Surgeons to be Lieutenant Colonels.
Brigade Surgeon Lieutenant Colonels to be Lieutenant Colonels.
Surgeon Lieutenant Colonels to be Lieutenant Colonels.
Surgeon Majors (ranking as Lieutenant Colonels) to be Lieutenant Colonels.
Surgeon Majors (ranking as Majors) to be Majors.
Surgeon Majors to be Majors.
Surgeons (ranking as Captains) to be Captains.
Surgeon Captains to be Captains.

THE ROYAL ARMY MEDICAL CORPS.—Captains J. S. Davidson, M.B., J. Will, M.B., J. Moir, M.B., J. Fallon, J. V. Salvage, M.D., A. R. Aldridge, M.B., J. Fayrer, M.D., F.R.C.S.E., C. P. Walker, M.B., C. J. Macdonald, C. J. W. Tatham, T. H. T. Clarkson, H. W. Austin, C. Garner, M.B., B. W. Wright, E. Eckersley, M.B., R. J. C. Cottell, J. R. Burrows, M.D., J. Keatly, F.R.C.S.I., D. Hennessy, D. M. Saunders, M.D., W. Kiddle, M.D., D. M. O'Callaghan, M. J. Whitty, M.D., H. A. Cummins, M.D., J. F. Donegan, J. Donaldson, H. B. Mathias, G. Bent, G. H. Barefoot, F. R. Newland, M.B., R. J. Windle, R. J. D. Hall, E. S. Marder, C. W. Allport, M.D., J. J. Russell, M.B., J. S. Edye, G. Scott, T. Browning, A. Hosie, M.B., G. F. H. Marks, M.D., R. Holyoake, R. N. Buist, M.B., J. J. C. Watson, M.D., T. Du B. Whaite, M.B., H. T. Knaggs, M.B., and F. S. Le Quesne, V.C., to be Majors; Lieuts. L. F. Smith, M.B., R. J. Blackham, S. H. Fairrie, M.B., G. T. K. Maurice, R. Fawssett, J. V. Forrest, M.B., H. W. Grattan, F. E. Gunter, M.B., J. H. Campbell and J. Grech to be Captains; Surgeons on probation W. H. S. Nickerson, M.B., G. S. Nickerson, M.B., G. B. Crisp, R. S. H. Fuhr, W. Jagger, M.B., A. B. MacCarthy, M.B., G. J. S. Archer, M.B., R. Selby, M.B., S. O. Hall, A. E. Weld, J. S. Gallie, F. J. C. Heffernan, A. E. Thorp, C. W. Mainprise, H. Herrick, E. J. Dobbin, J. Cown, M.B., A. R. O'Flaherty, H. B. G. Walton, and E. P. Hewitt to be Lieutenants.

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1.—The address of the JOURNAL OF TROPICAL MEDICINE is Messrs. BALE, SONS & DANIELSSON, 83-89, Great Titchfield Street, London, W.

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3.—All business communications and payments should be sent to P. Falcke, Secretary to the JOURNAL OF TROPICAL MEDICINE. Cheques to be crossed London and South Western Bank, Great Portland Street Branch, London, W.

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THE

Journal of Tropical Medicine

AUGUST, 1898.

A SCHOOL FOR THE STUDY OF TROPICAL MEDICINE.

THE announcement that it is intended to establish a school for the study of tropical diseases in London will be received with satisfaction. Of late years the Imperial idea has laid hold on the minds of, not only the Government of Great Britain, but the press and the public of all political shades of opinion are in unison as to its importance. The one individual who has fostered the idea and cherished it, more keenly perhaps than any other, is the present Minister for the Colonies. Not only has Mr. Chamberlain given his support to the principle of the Imperialistic idea, but in the details necessary to fulfil the national aspirations he has laboured with earnestness and vigour. The most recent proof of this, is the welcome announcement of the establishment of a school of tropical disease. The Colonial Office has initiated the scheme with a donation of £3,550, and an annual subsidy of £1,000. The school is to be attached to the Branch Seamen's Hospital at Greenwich. The Hospital is to be enlarged for the purpose, and Sir Henry Burdett has thrown himself into the scheme with his characteristic energy. There should be no difficulty in raising the small sum of money required to increase the size of the Hospital to meet the local demands. Those who benefit directly by the Hospital are the owners of shipping, and the immense advantages certain to accrue are sure to enlist the sympathy and active support of all concerned in the shipping interest. With adequate accommodation in the Hospital the efficiency of the school—under the able directions of the medical adviser to the Colonial Office, Dr. Manson, the author of the idea, and at whose suggestion the school was founded—is assured. The cases brought into Hospital are natives from the vessels lying in the London Docks. The patients are gathered from representatives of divers races, and the diseases they suffer from are, in consequence, equally varied. Natives of India, Africa, China, the West Indies and the islands of the Pacific, seek the relief the Hospital affords and their diseases are as varied as their nationality. In fact, no hospital in the world can show so great a variety of ailments under one roof. Here, then, we have the material ready for the purposes of instruction, and it is a satisfaction to know that through the wisdom of the Colonial Office it is to be taken advantage of.

The students who come up for instruction will be expected to take out a two months' consecutive course of demonstrations and clinical work. There is not likely to be any lack of pupils; the number of medical men who proceed to the tropics every year, whether for service in the Government of the Crown Colonies or as private practitioners, is considerable; and when to these are added medical officers of the army, navy, and the colonial services who are home on "study-leave," the aggregate of possible students is seen to be large. The details of the scheme of instruction are as yet in embryo, but the broad principles have been already worked out. The syllabus will include the whole range of tropical disease, and the teaching is intended to be chiefly of a tutorial description. Every student will have an opportunity of personally studying and verifying accepted beliefs in regard to malaria more especially. An endeavour is to be made to form a pathological museum, and the medical officers in the several colonies are to be asked to contribute suitable

specimens, by which means, no doubt, a good teaching collection will speedily be got together. A library of works relevant to the subject will also form part of the equipment of the school.

We entertain very little doubt that one of the fruits of this scheme will be a marked advance in the knowledge of tropical disease in the next generation of medical men proceeding to practise their profession in the tropics. They will start with a knowledge of methods of recognising and investigating disease, such as up till now was impossible. We are well aware how difficult it was, when Laveran's announcement of the discovery of the plasmodium malariæ first became known, for those who were untrained in methods of investigation, to not only recognise the parasite, but to interpret it aright even when they found it microscopically. This disadvantage will in the near future be removed, and by a short course of study at the tropical school before leaving home, medical men will be in a position not only to benefit science, but also to secure for their patients the fullest and most recent information as regards the rational treatment of their ailments.

We can easily conceive how a medical practitioner in distant lands rejoices to hear of the establishment of this school. He has longed to be systematically instructed in the work of his life, and he looks back to the day when, all ignorant and unprepared, he landed in the colony of his selection to treat diseases, many of which he had never even heard the names. For at least twelve months after he landed he found himself face to face with diseases of which his patients frequently knew more than he did, and not unfrequently lost a life which in after years he believes he might have saved. It is an uncomfortable reminiscence, but it was a position there was no avoiding, as his knowledge had to be gained by the bitter experience of want of success. By the establishment of a school of tropical medicine no practitioner need go to the tropics unprepared; and in future no practitioner dare present himself in any colony, however distant, without first having qualified himself to treat his patients intelligently.

DR. MANSON'S ADDRESS ON MALARIA, AND THE RESEARCHES OF SURGEON-MAJOR ROSS.

Of the many interesting subjects dealt with in the Section of Tropical Diseases at the Edinburgh Meeting the most attractive, perhaps, was Dr. Manson's demonstration on the malarial parasite and its extra-corporeal life as investigated by Surgeon-Major Ross. The lecture was given in the large Pathological Theatre of the University, and all the benches were crowded with medical men. The personality of the speaker, and the clear and simple language in which the facts of an intricate and difficult subject were expounded, held the large audience from the beginning to the end, and evoked from all who had the privilege to be present expressions of the highest praise.

Dr. Manson gave a short account of the malarial parasite, and its development in the blood of man. He then passed on to a consideration of the flagellated bodies, explained the views he had for long held regarding them, influenced by his former researches into the life-history of the filaria sanguinis hominis.

In Surgeon-Major Ross, a former pupil, Dr. Manson has found an ardent disciple, and it was interesting to note the enthusiasm of the master for his pupil's brilliant researches.

Dr. Manson's address, which we publish, will give our readers these researches in full. Briefly, the result of them is that Surgeon-Major Ross, after discovering flagellation of the crescent bodies in mosquitos fed on malaria-affected patients, added to that discovery, by a series of experiments on different species of mosquitos, the extremely important fact that the human malaria parasite—as well as a corresponding parasite of birds—proteosoma were not only to be found in the walls of the mosquito's stomach, but there underwent well-marked developmental changes. The different stages of development in the mosquito were shown in excellently prepared specimens under the microscope, and could be followed easily by those who saw the specimens. None who heard the lecture and saw the specimens prepared by Surgeon-Major Ross can doubt

the accuracy or importance of Surgeon-Major Ross's observations. To what they will ultimately lead it is premature to prognosticate, but in the meantime there can be no doubt, thanks to Dr. Manson and Surgeon-Major Ross, the evidence that the mosquito is the medium, or one of the media by which malaria is spread is becoming stronger every day.

INSTRUCTION IN TROPICAL DISEASES AT HASLAR.

At the meeting of the Parliamentary Bills' Committee of the British Medical Association, on July 5, Mr. Cantlie proposed the following resolution:—"That the Council of the British Medical Association be requested to represent to the Admiralty the need for instruction in tropical diseases at the Royal Naval Hospital at Haslar, and to ask the first Lord of the Admiralty to receive a deputation upon the subject." Dr. Ward Cousins seconded the resolution, which was unanimously carried.

At the Council on the following day the subject was brought forward by Dr. Saundby, the President of Council, and carried. In the course of his remarks, Mr. Cantlie pointed out that there had been no systematic teaching of tropical diseases given to naval medical officers since 1881. Up to that date the medical officers of the army and navy had conjointly received instruction at Netley, but since the medical teaching of the services were separated the subject had been dropped for the navy. Considering the primary importance to naval surgeons of their becoming thoroughly acquainted with this subject, it is scarcely to be believed that such a state of matters was allowed to continue. Surgeons in the navy are thrown more upon their own resources than any other section of their professional brethren. Gun-boats are often sent on independent cruises for long spells of service, when all communication with civil or other naval medical officers is entirely cut off. The surgeon has then to act on his own responsibility, and it is neither to the benefit of the patient or the service that the surgeon should be called upon to treat ailments with which he is unfamiliar. No doubt, after a few years, the medical officers of the navy are masters of their profession, and become the best informed of our medical experts in tropical disease; but if it is considered necessary to train the officers of the army and colonial services in tropical medicine, it is very much more so in the case of naval surgeons. There is scarcely a seaboard in the world that the vessels of the British navy do not visit, and there is no disease, however local, that a medical officer of the navy may not be called upon suddenly to treat. There are several other branches, appertaining to the instruction at Haslar, that ought to be added to the course before it is placed on an equal footing with the admirable instruction afforded at Netley; but of all imperative requirements, instruction in tropical medicine is the foremost. The hygiene of the ship calls as loudly for expert care as the military camp, and the surgery of the cock-pit is as important as that of the battle-field.

It is satisfactory to learn that the necessary steps are being taken to remedy the defect here referred to, and we hope soon to know that this important branch of the public service is receiving the attention it deserves.

The reply received from the Admiralty to the petition forwarded by the President of the Council of the British Medical Association is as follows:—

ADMIRALTY,
16th July, 1898.

SIR,—Mr. Goschen desires me to acknowledge the receipt of your letter of the 11th instant with regard to the question of providing special instruction in the treatment of tropical diseases to medical officers on joining the Royal Navy. In reply, I have to inform you that this subject is receiving the very careful consideration of the Board of Admiralty, who are quite alive to the importance of giving surgeons of the navy every facility for acquiring, as far as possible, the latest knowledge obtainable on the nature of the diseases in question, and the progress of scientific enquiry into the methods of treatment.

As the Board are in agreement with the views of the Council of the British Medical Association in the matter, it appears to Mr. Goschen to be unnecessary to put the Council to the trouble of sending a deputation to represent the importance of the case to the Admiralty.

The difficulties which may have to be dealt with are

mainly of an administrative character, such as to what extent, and at what periods, officers can be spared from their duties afloat for the purpose of going through special courses of instruction on shore, and in order to examine in what way the object desired can best be obtained it is the intention of the Admiralty to appoint a small departmental committee of enquiry.

I remain, yours faithfully,

W. GRAHAM GREENE.

ROBERT SAUNDBY, Esq., M.D.

THE BRITISH MEDICAL ASSOCIATION.

ANNUAL MEETING AT EDINBURGH, JULY, 1898.

THE sixty-sixth annual meeting of the British Medical Association was held at Edinburgh during July 26, 27, 28, 29, and 30.

The gathering of medical men was a large one, to which several circumstances contributed. In the first place, more men enter the medical profession through Edinburgh than through any other school; it is by far our largest centre of medical education. In the second place, the fame and traditions of past teachers have given Edinburgh, its University and Colleges, a halo of greatness beyond compare; and lastly, but by no means least, Edinburgh itself possesses an attraction to the artist, the historian, and the ordinary traveller such as no other city in the wide domains of the Empire can boast of.

Well nigh 2,000 medical men assembled, and a highly interesting, pleasant and satisfactory meeting was the result. It is not intended to give a detailed account of the social doings or even a general *résumé* of the professional work. These are fully recorded in the journals devoted to the general interest of the profession.

Great interest was taken in the newest sectional addition, that of Tropical Diseases. The President of the Association, Sir Grainger Stewart, took keen personal interest in its development and watched its proceedings carefully.

Who initiated the idea of a Section of Tropical Medicine at the meetings of the Association we cannot say. To Dr. Argyll Robertson we are indebted, perhaps more than to any other person, for the fact that the Section was formed and incorporated amongst the several departments under which the work of the Association is carried on. Although ophthalmology, as all the world knows, is Dr. Argyll Robertson's speciality, we can easily believe that the trip round the world, which he took some four years ago, opened his eyes to the fact that Greater Britain demanded to be considered, and that the professional requirements of some 6,000 of his medical brethren practising abroad were worthy of a place at the representative meetings of the Association.

The Section of Tropical Diseases was marked "N" in the syllabus of the Association, and the place assigned for its meetings was the Practical Pathology Laboratory in the University New Buildings.

The development of the Section was entrusted to the following:—*President:* Patrick Manson, M.D., LL.D. *Vice-Presidents:* Andrew Davidson, M.D., LL.D.; Andrew Smart, M.D., LL.D.; William John Ritchie Simpson, M.D. *Honorary Secretaries:* James Cantlie, M.B., 46, Devonshire Street, Portland Place, W.; Surgeon-Captain M. Louis Hughes, A.M.S., Aldershot.

To gather together, in fair number even, men interested in tropical medicine is no light task, seeing how widely scattered are the potential components. It speaks, therefore, highly in favour of the keen interest men in tropical countries take in their profession, that the Section was largely attended, not only at the commencement of the sittings, but up to the very last moment allowed by the rules of the Association. The commodious room placed at the disposal of the Section was none too large, and at times every seat was occupied.

Amongst the foreign visitors the best known are Professor Blanchard (Paris); Dr. Guiart (Paris); Professor Osler (Baltimore, U.S.A.). The Royal Navy, the Indian Medical Service, the Royal Army Medical Corps and the Colonial Service were well represented at the Section, and almost every part of the Empire was represented by one or more medical men. From the West Indies, Central Africa, the West Coast, Egypt, Ceylon, India, the Straits Settlements, Hong Kong and China, medical men assembled and took part in the discussions or contributed papers.

All this augurs well for the future of such meetings, more especially when it is taken into consideration that the machinery for organising the Section was entirely new. In future years, when the Section is better known, we have no doubt that even larger gatherings will assemble, but in the meantime the promoters of the idea and the executive of the Section have every reason to be satisfied with the complete success of the initial venture.

Wednesday, July 27.

The PRESIDENT (Dr. Patrick Manson) opened the proceedings by an introductory address:—

"It has been the policy of the British Medical Association, a policy especially apparent in recent years, to include in its operations the Colonies and the Dependencies of the Empire, as well as the three Kingdoms themselves. As many of these Colonies and Dependencies are situated in or near the tropics, as in the aggregate they carry an enormous population, and as many of the diseases of these countries are more or less peculiar to the tropics, the institution at the annual meetings of this Association of a section devoted to tropical medicine, was the inevitable outcome of the catholic policy I refer to.

"Furthermore, the remarkable progress which this branch of medicine has made during the last twenty years, more especially since Laveran's discovery of the malaria parasite in 1880, has been so great that the study of tropical medicine has become a life's work in itself, and consequently, like so many departments of our science, is perforce, day by day, becoming more specialised.

"Every year the volume of discovery increases; new fields of enquiry are constantly being opened up, and pathologists are beginning to realise the important fact that just as the ordinary fauna and flora of the tropics are infinitely richer in species than are those

of the temperate zone, so the pathological fauna and flora of the tropics and the diseases to which they give rise, are also infinitely more numerous than their congeners of cooler climates. It must be so. High temperatures and plentiful moisture, in the aggregate, everywhere make for abundant life, whether it be a question of ordinary beasts and plants or whether it be a question of pathogenic organisms.

"From a pathological point of view, therefore, there is abundant justification for the initiation of this section. There are other grounds, however. The profession is becoming alive to the fact that a large proportion of its numbers—a fifth or a sixth—have a direct personal interest in tropical medicine. Moreover, public interest in this matter is manifestly growing, and even the rulers of the empire are at last waking up to a sense of a neglected responsibility. It seems to me, therefore, that the Association has done well to institute this section, and I think that it has been especially happy in its selection of this particular time and place for so doing. It seems to me that it has hit upon the psychological moment, and, considering the leading position which Edinburgh has always held in the field of medicine, I think it has hit upon, if I may be allowed the expression, the psychological place.

"I have no intention of inflicting on you a long address, but I consider it incumbent on me briefly to indicate that, at the special Tropical Section, we have three distinct functions to fulfil. In the first place, we must work for the affiliation and organisation of the five or six thousand British practitioners who are more or less directly interested in tropical medicine. Five or six thousand is an enormous body. Such is its magnitude, however. Hitherto it has been without head or organisation; it has been conscious of its duties only, unconscious of its size and power and absolutely oblivious of its rights.

"Second. We have to see that those who propose to join our ranks in the future are properly educated in our special branch.

"Speaking to an audience most of whom, I presume, have had personal experience of tropical practice, it is hardly necessary to enlarge on this point. We all of us feel acutely the backward state of our educational system in its bearing on tropical medicine. There is not one of us that does not bewail the crass ignorance in which, at the outset of his career, he lightly undertook the care of men's lives in dangerous climates. There is not one of us that does not mourn over innumerable lost opportunities of adding to our common stock of knowledge; opportunities lost, not even recognised, because of the inadequacy and unsuitableness of our initial medical education. There is not one of us that cannot pillory himself with the recollection of lives that perished entirely owing to the lack on our part of an elementary knowledge of tropical medicine. This is a theme very susceptible of illustration. I forbear; but we must have, we must insist upon, improved education in tropical medicine.

"Lastly. We have to discuss the problems of tropical diseases. Though much has been done in recent years, that which has been accomplished has but served to once more show that there is no finality in research. The higher we rise, the wider becomes the horizon. Laveran's discovery has illuminated many things that were dark before, but it has also opened out new problems and shown dark spots of ignorance hitherto unsuspected. It has left untouched the practical aspect of that important subject, the ætiology of malaria. Beyond that which Ross has just told us, we still know absolutely nothing of the malaria parasite as it exists outside the human body. Similarly, we are still in absolute ignorance of the location, and of the physical features of the malaria parasite while latent in the human body, or of the exact physical conditions that cause it after months, or even years, to spring into active pathogenic life. Again, that other great tropical disease, dysentery, is still, as regards many of its forms, their causes and their treatment, a sealed book. The pathology of liver abscess is still, as it has ever been, a *quæstio vexata*. We just begin to know a little about the symptoms, epidemiology and pathology of beri-beri: we do not know its cause. Filariasis, ankylostomiasis, distomiasis, are all new subjects, bristling with obscurities, as well as with practical points in prevention and treatment. There is plenty of work for us to do. These are not a tithe of the problems in tropical pathology calling for our consideration.

"Every one who has tried to work in this field must have felt dimly that the recognised and named diseases of the tropics are far out-numbered by the un-named and un-differentiated diseases. In all pathology there is no such field for fruitful original research and discovery as tropical medicine.

"I conclude these few remarks with the hope that the creation of this Section will receive its justification in the amount and quality of the work we may get through on this occasion; and that the Tropical Section may become a permanent feature in the annual meetings of this Association."

The valuable papers contributed to the Section will be published in the JOURNAL OF TROPICAL MEDICINE, and the remarks made by those taking part in the discussions will be given as fully as possible.

A summary of the proceedings is all that space will allow of for the present issue:—

Brigade-Surgeon-Lt.-Colonel CROMBIE, I.M.S., opened the discussion upon "Unclassified Fevers of the Tropics." The paper he read was an elaborate summary of tropical fevers, compiled with great care and completeness. Brigade-Surgeon-Lt.-Colonel ARNOTT, Surgeon-Major BAKER, Major DAVIDSON, Captain HUGHES and Dr. THORNHILL (Ceylon), SAMBON (Rome), CHARLES (Edinburgh), Dr. COUSLAND (Swatow, China), and CANTLIE (London), took part in the discussion.

Dr. ANDREW DAVIDSON (Edinburgh) read an interesting paper on "The Malarial Problem in its Epidemiological Aspects."

Dr. MANSON (London) described the "Appearance of Pigmentation in Lymphocytes in relation to the Diagnosis of Malaria." The paper was chiefly devoted to a differentiation between lymphocytes and phagocytes, their purposes, functions and appearances.

Dr. SAMBON introduced the subject of "Blackwater Fever" in an able paper, in which he attempted to place this fever in the nomenclature of disease.

Dr. SYMES THOMSON and Dr. PRENTICE joined in the discussion.

As the result of the alarming state of mortality which was shown to exist in Central and West Africa from this fever, the meeting sent a resolution to the Council of the Association, which was brought forward on the following day by Dr. Saundby, as seen by an extract from the proceedings:—

"Dr. Saundby, President of the Council, announced that the Section of Tropical Diseases had asked the Council to bring before the general meeting the desirability of petitioning Government to send an expert pathologist to South Africa to investigate the blackwater fever. He moved that the general meeting should make a representation to Government to that effect.

"Professor Chiene seconded and the motion was agreed to."

Dr. THIN contributed a paper on "The General Characteristics of Pernicious Fevers in British Guiana." A microscopic specimen illustrative of the parasite of these fevers was also shown by Dr. Thin.

Major YARR, R.A.M.C., read a paper of extreme interest on "Malarial Affections of the Eye." The paper will be published *in extenso* in an early issue.

Surgeon-Major J. H. TULL WALSH, I.M.S., contributed a paper on "Clinical Aspects and Treatment of Malarial Fever."

Surgeon-Captain W. J. BUCHANAN, I.M.S., described the clinical, pathological and epidemiological aspects of "Epidemic Cerebro-Spinal Fever in India," and maintained that the cerebro-spinal fever in India is identical with the epidemic form of the disease described by European and American writers.

Thursday, July 28.

The President (Dr. Patrick Manson) in the chair.

Mr. CONOLLY NORMAN (Dublin) introduced the subject of "Beri-beri in Temperate Climates" in a paper of great scientific and historical value. Mr. Norman showed that beri-beri was not confined to the tropics, but that in North Germany, England, Ireland, the United States of America and Newfoundland, recent outbreaks of the disease had taken place. Dr. Osler, Dr. Manson and Mr. Cantlie joined in the discussion.

Dr. SANDWITH (Cairo) read a paper on "Pellagra" in Egypt, and showed that the disease as known in Italy existed in Egypt.

Dr. Sandwith (Cairo) also read a paper on "The Treatment of Acute Dysentery." He urged the advantages of sulphate of magnesia, and of enemata of sulphate of copper, 15 grs.; tincture of opium, 15 minims; starch, 1 oz.; with water, 2 pints. Dr. OSLER (Baltimore), Dr. BENTLEY (Cairo), Dr. OZANNE (British Guiana), Dr. PRENTICE (Central Africa), Major DAVIDSON, R.A.M.C., Brigade-Surgeon-Lt.-Col. CROMBIE and Sir JOSEPH EWART (Brighton, late I.M.S.) joined in the discussion, which was kept up with keen interest for some considerable time. Sir Joseph Ewart warned practitioners against neglecting ipecacuanha. He stated that he had seen the beneficial effects of the drug when it was first introduced in India, and, comparing the enormous lessening of mortality from dysentery by the introduction of the drug in the treatment of dysentery, he could not speak too highly in its favour.

Inspector-General TURNBULL, R.N., read a paper on "Insanitary Environment the Cause of the Spread of Yellow Fever and the Bubonic Plague."

Owing to want of time the following papers were taken as read: "A Leaf from the Early History of Bacteriological Discovery," by Andrew Smart, LL.D. (Edinburgh); "On the Training of Regimental Officers in the Prevention of Disease in the Army in Tropical Climates," by Surgeon-Captain Louis Hughes, R.A., M.C.; on "Surgery in the Tropics," by James Cantlie, F.R.C.S.

Friday, July 29, 1898.

Dr. ANDREW DAVIDSON, LL.D., Vice-President, in the Chair.

Professor W. T. R. SIMPSON (London, late of Calcutta) read a paper on "Plague in India." Dr. Simpson dealt with the past history of epidemics of plague in India, with the spread of the recent epidemic, and urged strongly the necessity for a sanitary service for India.

A paper on "The Testing of the Plague Prophylactic in Plague-Stricken Communities in India," by Professor HAFFKINE and Surgeon-Major BANNERMAN, I.M.S., went to show that by Haffkine's method of serum inoculation the mortality of protected persons, as compared with the unprotected, was lessened by 86 per cent.

An abstract of the report on the Pathology of Plague to the Bombay Plague Research Committee, by Surgeon-Capt. L. F. CHILDE, I.M.S., was read by Brigade-Surgeon-Lieut.-Colonel ARNOTT.

Brig.-Surg.-Lieut.-Col. DIMMOCK, I.M.S., gave a detailed account of the measures adopted in Bombay for dispelling the plague.

Brig.-Surg.-Lieut.-Colonels ARNOTT and DIMMOCK, Surg.-Major BAKER, and Mr. CANTLIE entered into the discussion on points raised by Professor Simpson. The general opinion seemed to be that India was not likely to be free of plague for some years, and that strict sanitary precautions were requisite.

Dr. THORNHILL (Ceylon) read a communication on the "Treatment of Cholera."

As the meeting drew to a close Dr. ANDREW DAVIDSON, the chairman, read the following resolutions, which were seconded by Sir JOSEPH EWART and carried unanimously:—

(1) That this Section, recognising the great importance of the researches of Surgeon-Major Ross on the development of the proteosoma in the mosquito in its bearings on the ætiology of human malaria, desire to convey to him through the President its deep sense of the obligations which all students of tropical pathology are under to this distinguished observer, and to assure him of the interest and hope with which it follows his work.

(2) That this Section records its warmest thanks to the President for the very able manner in which, notwithstanding much physical suffering, he has presided over its proceedings, and for having placed before the Association the latest discoveries of Surgeon-Major Ross with which his own name is so inseparably identified.

In seconding the resolutions, Sir JOSEPH EWART said that he warmly approved of the complimentary observations of Dr. Davidson on the investigations in

which Dr. Ross is now engaged, and trusted that the natural relations between the mosquito and the malarial parasite will soon be settled by him. It was scarcely necessary to remind the Section of the great work of research and discovery made by Dr. Manson in the domain of the filaria. His professional and clinical labours recently undertaken in connection with two of the London Schools of Medicine were much appreciated. Sir Joseph was glad to recognise the large attendance, the admirable papers, and genuine enthusiasm at the meetings of the Section, which has been to himself, and many others, a welcome addition to the organisation of the Association. He, therefore, hopes that at future meetings of the Association a place will be found for a Section on Tropical Medicine.

Inspector-General TURNBULL, R.N., proposed, and Dr. ANDREW DAVIDSON seconded, that a vote of cordial thanks be accorded to the Hon. Secretaries of the Section of Tropical Diseases for the admirable way they had fulfilled their duties.

Reviews.

TROPICAL DISEASES: A MANUAL OF THE DISEASES OF WARM CLIMATES. By Patrick Manson, M.D., LL.D.Aberd. London: Cassell & Company, 1898. Cr. 8vo., 624 pp. Price 10s. 6d.

It is seldom that we have perused a text-book on medicine with so much satisfaction and profit as we have that of Dr. Manson's "Manual of the Diseases of Warm Climates." The author is to be congratulated on the clear and interesting manner in which he has succeeded in conveying all he has to say on the chief diseases peculiar to warm climates, and in placing before the profession a volume which will be particularly useful to the workers in this domain of medicine. The volume is divided into seven sections. Section I. is devoted to Fevers, under which are chapters on malaria, yellow fever, plague, dengue, Mediterranean fever, Japanese river fever, Nasha fever, Kala-azar, tropical typhoid, heat stroke, and unclassed fevers. Section II. deals with General Diseases of Undetermined Nature, such as beri-beri, epidemic dropsy, and negro lethargy or sleeping sickness. Section III. takes up Abdominal Diseases, under which are comprised cholera, dysentery, endemic gangrenous rectitis, hill diarrhœa, sprue, tropical liver, abscess of the liver, infantile biliary cirrhosis, and ponos. Section IV. treats of Infective Granulomatous Diseases, such as leprosy, yaws, verruga Peruana, ulcerating granuloma of the pudenda, and oriental sore. Section V. is on Animal Parasites and Associated Diseases, Section VI. on Skin Diseases, and Section VII. on Local Diseases of Uncertain Nature. The volume has nearly one hundred illustrations, the chapters on malaria being especially rich in that respect. Dr. Manson defines the malarial parasite as a protozoal organism of warm climates, which, although ordinarily living in external nature, is capable of becoming parasitic and of multiplying in man, giving rise to fever.

A very full description of the intra-corpuscular or human cycle of the plasmodium is given, showing how the parasite maintains itself and multiplies in the human body. The organ or tissue it selects in its latent condition, or the exact conditions which cause it once more to resume active, propagating, circulating life are stated to be unknown, while the extra-corporeal life of the parasite is still the subject of investigation and of theory. Dr. Manson has advanced in this volume his mosquito theory in explanation of the manner by which the malarial parasite leaves the human body to follow an extra-corporeal life. It is pointed out that the flagellated bodies are never seen in newly-drawn blood, and that they come into view only after the specimen has been mounted some time. It is Dr. Manson's view that the flagella are flagellative spores, the extra-corporeal homologues of the intra-corporeal spores; in fact, that the flagellated bodies form the first phase of the extra-corporeal life of the plasmodium. His theory is that the mosquito is the intermediate host of the malarial parasite, and that man becomes infected by drinking water contaminated by the mosquito or by inhaling the dust of the mud of dried-up mosquito-haunted pools, or in some similar way. Surgeon-Major Ross's experiments in support of this view are given, so are those of MacCullum's, but the opposing opinions of Marchiafava, Bignami, Blanchard, and others are not omitted. The latter consider that the crescents, spheres, and flagellated bodies are degenerated, dead, or moribund parasites. The volume was published previous to the latest important experimental observations of Surgeon-Major Ross, on the development of the proteosoma in the mosquito, which strengthen in a remarkable manner Dr. Manson's contention. A table giving the characteristics of the various parasites will be found useful to the practitioner, while the chapter on treatment, the time and manner in which quinine is to be given and the precautions to be taken in malarious countries are eminently practical. In a book in which the subjects are so carefully treated it is difficult to select any specially for remark. The chapter on Malta fever is particularly interesting to military surgeons, as this disease is extremely prevalent at times in the garrisons of Gibraltar and Malta, but as it is probable that it occurs also in other parts of the tropical world, the short but excellent account of the disease will prove valuable to practitioners elsewhere. The chapters also on animal parasites and associated diseases are instructive, and their study is of importance, because the presence of these parasites give rise to many obscure diseases. Filariasis, Bilharzia, guinea worm, ankylostomiasis, are in turn skilfully handled, and described in a manner that leaves a clear impression of these diseases. Taken as a whole the volume is one which we can thoroughly recommend to the student of tropical medicine, and to the medical man who either practises or intends to practise in the tropics. The book is opportune at a time when so much attention is being directed to the necessity of the study of tropical diseases, and so many are asking what is the best book to read on the subject.

EXERCISE FOR HEALTH: ITS SCIENCE AND PRACTICE. By H. H. Hulbert, M.R.C.S., L.R.C.P., and Luis J. Phelan, London. The Whitely Exerciser, Lt., and C. Arthur Pearson, 260 pp. Illustrated.

Residents in the tropics are placed at a great disadvantage compared with home dwellers in regard to exercise. In the equatorial belt, where a maximum of heat obtains all the year round, and even in the regions of the tropics having a "cold" season for eight months of the year, ordinary English out-of-door sports are well nigh impossible. Any suggestion or device whereby suitable exercise is obtainable in warm climates is a welcome contribution. This drawback to health with which tropical residents have to contend, is largely provided for by the plan recommended in this admirable book.

Sports such as cricket, football, tennis and racquets are not without their risks in the tropics. To "field" all day, exposed to the full force of a tropical sun, to engage in a fast game of football, or to even play in a match, at the milder game of tennis, are pregnant with possibilities of dangers which detract from their usefulness to maintain health in warm climates. During the "cold" season these games no doubt recommend themselves to the younger men, but for men and women beyond or approaching the "forties," they are for the most part unadvisable. But even for young men, whilst the hot season lasts, physical exercise is reduced to a minimum. This is a misfortune and a source of danger to

health, which until lately has been unsurmountable. Of all forms of exercise devised of late years, none so highly recommend themselves as that provided by the "Whitely Health Exerciser." It consists of a simple and practical device whereby a complete system of scientific muscular movements is easily obtainable. The apparatus is capable of bringing into play the various muscles of the body in an infinite variety of carefully selected movements. The instrument is cheap, occupies but little space, weighs less than two pounds, and is at once portable and handy to take about. There is no complicated machinery to get out of order—a most important consideration, more especially in up-country districts. It merely consists of a long elastic cord running over pulleys, and can be fixed up in one's bedor bath room in a few seconds. The book giving instructions is clearly written and contains valuable information concerning the general maintenance of health, which is both authoritative and explicit. The illustrations are ample, instructive and readily understood. The physical health in warm climates can be maintained by the use of the "Exerciser," without the dangers attending exposure to the sun, which are so vital a deterrent to the full benefits obtainable from out-of-door sports, when practised in the tropics.

No one intending to take up his or her residence in the tropics, or to proceed on a long sea voyage, should leave home without being provided with both the "Whitely Health Exerciser," and the book of instructions of how to use it.

A Manual of Jail Hygiene for the Use of Medical Subordinates. By Surgeon-Captain W. J. Buchanan, B.A., M.B., B.Ch., D.S.M., I.M.S. Bengal: Secretariat Press. 1898. Price R3.

This little volume is modestly stated to be intended for the use and instruction of medical subordinates in Bengal jails, whose duties we note from their enumeration to be of a very responsible nature. The medical subordinate is the executive officer who supervises, or is expected to supervise, all the arrangements for the health and comfort of the prisoners, and whose responsibilities are not confined to the medical or surgical treatment of the prisoners who are ill, but also include the prevention of sickness and the maintenance of cleanliness and sanitation of the jail premises and the personal hygiene of the prisoners. With such wide and important duties a manual which will serve as a practical and safe guide to him must be a most valuable acquisition, and we have no hesitation in stating that Surgeon-Captain Buchanan's manual meets a distinct want. In small compass it deals with all important matters relating to the health and comfort of the prisoners, and is full of excellent advice and direction, which is thoroughly practical and which will prove useful not only to medical subordinates but also to medical officers connected with jails. We see that it is specially written for Indian jails, but the principles laid down apply equally well to all jails in the tropics, and we hope that Dr. Buchanan's book will secure this wider circulation.

News and Notes.

A bill has been introduced into the House of Commons whereby it is intended to legislate upon the use of foreign medical degrees in Great Britain and Ireland. The spirit of the intended legislation is, that any person attaching to his name letters indicating a medical diploma, shall, unless such diploma has been obtained in the United Kingdom, place and clearly indicate the source from which it has been received.

This question of the registration of diplomas comes home to members of our profession resident in the British Colonies. We are aware several have local regulations as to the right of persons possessing foreign diplomas and degrees to practise, but how far this is general we cannot ascertain. We would be greatly beholden to medical men in the Crown Colonies and Protectorates, more especially, were they to forward information to the Editors of this Journal, so as to enable them to be accurately informed as to the local rules in force. Armed with such information, it would then be possible to deal with the subject, and bring it before the proper authorities. Perhaps local secretaries of Medical Societies would kindly supply us with the regulations appertaining to their particular Colonies.

Dr. Thomas L. Bancroft, of Burpengary, Queensland, Australia, in the *Australian Medical Gazette* of June 20, 1898, records the results of his investigations into the "life history" of Filaria Bancrofti and Filaria inimitis, and has been able to verify some of Manson's statements regarding the former. His observations up to date are as follows:—

(1) Metamorphosis of filaria sanguinis hominis takes place in the muscles of the thorax of two species of mosquito, viz., culex ciliaris and culex vigilax.

(2) Upon the death of the mosquito, the young filariæ do not escape from its body into water, and therein live a free life, as Manson and others thought, but they invariably die.

(3) Those embryo filariæ, which are destined to develop, leave the stomach of the mosquito at an early date, and take up their abode in the muscles of the thorax. After the meal of blood has been digested, the stomach and even the abdomen is free from filariæ.

(4) The young filariæ live a day or two after the death of the mosquito, but should the latter fall into water they die immediately.

We have received numerous useful and welcome hints in regard to the management of the Journal of Tropical Medicine. One of the most important is that the Journal ought to be issued weekly. This request shows how earnestly a journal dealing with tropical medicine is wished for, and is a great stimulus and encouragement to the promoters of the Journal. We may say at once that the original idea was a quarterly publication, but in the meantime, for at any rate twelve months, one journal per month will be issued. The corresponding journal in Germany is a bi-monthly periodical, but as tropical diseases are of greater consequence to the British than to the German Empire, a more frequent issue seems justifiable. The Editors will be pleased at all times to receive suggestions from contributors concerning the work of the Journal and the lines along which it should be conducted.

Fever Specific.—*Chilian Solanaceæ.*—One of the most valued popular remedies in Chili is the so-called "Natri," which probably includes at least three species of the Solanaceæ, *Solanum crispum*, *S. gayanum*, and *S. tomatillo*. It is largely employed as a remedy against fevers, &c. Ramdohr has examined

this drug, and finds in it an alkaloid which gives most of the reactions of solanine. It melts with decomposition between 230°—235°, is soluble with ease in hot alcohol, almost insoluble in water and ether. The salts are all easily soluble in water, with the exception of the gallate and the picrate. From the alcoholic solution of the alkaloid, it can be obtained in fine needles. The gold salt contains 16·6 per cent. of gold. Elementary analysis yielded discordant results, owing probably to the impurity of the substance obtained.—*Pharm. Zeit.*

Sir William McGregor, M.D., K.C.M.G., C.B., has retired from the post he has so long and so ably held as Lieutenant-Governor of British New Guinea. Sir William McGregor graduated at Aberdeen as M.B. in 1872, and M.D. 1874. He joined the Colonial service in Fiji, and was elected Lieutenant-Governor of New Guinea when first it was taken over by the British Government. The retiring Lieutenant-Governor has not only proved himself an administrator of a high order, but has added handsomely to our scientific knowledge of this, until now, little-known country.

The question of vaccination and anti-vaccination is so prominently before the public in the meantime, that it would be well were those responsible for vaccination in the Colonies to relate their experiences. We shall be glad to receive communications in reference to vaccination in the Colonies, so that the opinion of medical practitioners abroad may be ascertained. The relation of the natives to the medical acts in regard to vaccination should be given.

Plague.—What may be regarded as the æstivo-autumnal lull in plague is now happily with us, both in China and India. A year or two ago we were fain to believe that with the subsidence of plague during the hottest weather the disease had disappeared for good; experience has taught us, however, that the belief was a delusion, and that with the cooler weather a recrudescence may be looked for.

Medical practitioners resident abroad are particularly requested to see that their addresses are correctly and fully given in the Medical Directory. Communications should be sent to the Medical Directory, 7, Great Marlborough Street, London, W.

The question of improving the methods of transport for medicines and medical appliances in connection with Field Hospitals for the British Army in India is at present being considered by a special commission.

The proposed amendments to the Cape of Good Hope Pharmacy Laws which were down for discussion, have, owing to the recent defeat of the Colonial Government, been dropped.

Papers Promised.

(1) River Pollution in Mauritius; (2) Reforms in the Government Medical Department in Mauritius; (3) Discomforts and Dangers to Health on Board Passenger Lines of Steamers; (4) Some of the Sanatoria in the British Colonies. By D. E. Anderson, M.D.Paris, M.B., B.A., B.Sc.Lond.

On Yaws, and other papers. By A. Barber, M.R.C.S.

(1) Lymph Sinus; (2) Perforation of Duodenum by Ascaris Lumbricardis; (3) A Case of Small-pox. By E. A. Chartres, L.R.C.S.I. and L.R.C.P.I.

(1) Malarial Neurosis; (2) Substitutes for Quinine; (3) Chronic Dysentery; (4) Night and Day Blindness following Beri-beri; (5) Organic Extracts in the Treatment of some Tropical Diseases. By Robert W. Felkin, M.D., L.R.C.P. and S.Ed.

Anti-malarial Drugs. By Surg.-Major A. Duncan, I.M.S.

(1) Case of Obstinate Ringworm of the Face; (2) Case of Leprosy treated as Specific for three to four years, until Microscopic Diagnosis was made. By J. C. Graham, F.R.C.S.Ed., M.D.Bonn.

(1) Notes on various Subjects; (2) Whooping Cough in Negroes; (3) Malarial Fevers; (4) Intestinal Worms in Children. By St. George Gray, M.B., B.Ch. (Univ. Dub.).

Acute Ozæna among Natives in India. By Sophia Grumpelt, M.D.Bern., L.R.C.P. & S.Ed.

Leprosy in the Canary Isles. By Stanford Harris, M.D., M.R.C.S., L.S.A.

Causes of Death in Malaria. By W. E. de Korte.

The Endemic Neuritis of Jamaica. By G. V. Lockett, F.R.C.S.

The Influence of Malaria on the Surgery of the Urinary Organs. By W. A. Mackay, M.D., F.R.C.S.

Notes on Three Cases of Black-water Fever. By G. F. Reynolds, M.R.C.S.Eng.

Malarial Eye Affections. By M. T. Yarr, F.R.C.S.I.

A Note on the Dosage of Quinine in Malarial Fever. By W. Jones Greer.

Malaria. By Surg.-Major A. Duncan, I.M.S.

(1) Native Diseases in Nyassaland; (2) Malaria in Central Africa; (3) The Rate of Quinine Excretion. By S. Kellett Smith, F.R.C.S. (Angoniland Exploration, 1895-96).

Communications, Letters, &c., have been received from:—

A.—Dr. R. Adamson (Labuan, Borneo); Mr. D. Alexander (Jamaica, W. T.); Mr. Ameresekere (Udugama); Dr. D. Anderson (Paris); Dr. E. Oliver Ashe (Kimberley); Editor of "Australasian Medical Gazette."

B.—Dr. A. Beveridge (India); Mr. A. Barker (Borneo); Dr. R. Bell (Jamaica); Mr. T. Mulock Bentley (Orange Free State); Mr. Bishop (Sierra Leone); Dr. Blackburn (Cape Colony); Mr. Wm. Boase (British Guiana); Mr. R. Lee Bolton (Smyrna); Mr. P. Hamilton Boyden (Portsmouth); Surg.-Capt. W. Buchanan (Bengal); Dr. Buschan (Stettin); Mr. Frederick Burge; Miss Benson, M.D. (Bombay); Dr. Barnes (China); Mr. Burge (Shanghai); Dr. Bankart (Malta); Dr. Brown (Amritsar); Dr. Bennett (Trinidad); Dr. Brander (Belfast).

C.—Dr. Frank Cantlie (Swatow); Dr. Chassaud (Smyrna);

Surg.-Col. O. H. Channer; Mr. Chartres (N. E. India); Dr. Clare (Jamaica); Dr. G. Clarke (Cachar); Dr. A. J. M. Closky (Selangor); Mr. A. P. Coates (Cape Colony); Dr. Craig (India); Mr. C. Couran (Madras); Dr. Cooke (W. Indies); Dr. Cran (Brit. Honduras): Miss Corthorn, M.D. (Bombay); Mr. Comley (Calcutta); Lt.-Col Channer; Dr. Cowan (Bengal).

D.—Dr. Dalzell (Natal); Surg.-Lt.-Col. Dantra (Mandalay): Dr. Davey (Transvaal); Dr. Andrew Davidson (Edinburgh); Dr. Dalgetty (India); Mr. R. F. Dedu'ckson (Calcutta); Dr. Drummond (Ceylon); Dr. A. Duncan (Almora); Lt.-Col. Donovan; Dr. D'Erf Wheeler (Jerusalem); Dr. de Leon (Jamaica); Mr. Davis (British Honduras); Surg.-Major Dimmock (London).

E.—Dr. Elliott (Gold Coast); Dr. Edgar (Perak); Dr. A. J. Elliot (Assam); Dr. Edington, Dr. Eyre (Cape Town); Dr. Edkin (Trinidad).

F.—Dr. Farmer (Mysore State); Dr. Robert Felkin (London); Dr. Fells (S. India); Major R. H. Forman; Mr. Gundry Fox (Perak); Dr. Francis; Mr. Fritz (W. Indies); Mr. W. H. Fry (Singapore).

G.—Dr. Graham (Sumatra); Dr. St. Geo. Gray (St. Lucia): Mr. Greenshields (Michigan); Dr. Grosett (Jamaica); Dr. Sophia Grumpelt (Cape Colony); Dr. Galloway (Singapore).

H.—Mr. Hallwright (British Honduras); Mr. Harrison (Belize); Mr. Howard (Cape Colony); Dr. Stanford Harris (Las Palmas); Capt. Hughes (Aldershot); Major T. G. Harwood (Aldershot); Mr. E. S. Humphrey (W. Australia); Mr. H. Hudson (South India).

J.—Surgeon-Major-General Jameson, C.B. (War Office); Mr. E. Joseph (Trinidad); Mr. Joslen (Jamaica).

K.—Dr. Kennedy (Demerara); Dr. Keyt (B. Honduras); Surg.-Lieut. Knapp (Cape Colony); Mr. T. H. Knott (Andover); Dr. de Korte (Cape Colony).

L.—Dr. Law (Berbice); Mr. Lockett (Jamaica); Dr. Lund (Transvaal); Dr. Lyall (Swatow); Mr. Charles Lyons (Johannesburg); Mr. Leask (Singapore).

M.—Surg.-Col. K. Macleod; Dr. W. A. Mackay (Huelva); Dr Digby Macphail (Jamaica); Surg.-Capt. R. H. Maddox (Bombay); Colonel Maturin (Colchester); Surg. W. B. Maurice, R.N. (Portland); Mr. J. Preston Maxwell; Mr. P. Michelli (Dreadnought Hospital, Greenwich); Dr. Middleton (Singapore); Mr. Arthur Milner (Trinidad); Mr. H. Milton (Cairo); Dr. A. Morrison (Alexandria); Dr. Murison (Cairo); Mr. Edw. Mahon, R.N.; Miss Mitcheson, L.R.C.P. and S. (N. India); Dr. P. M. Muttukumaru; Dr. Macdonald (Cape Colony); Dr. McNicol (Hoogli); Mr. G. Moberly (Natal); Dr. Mackinnon (East Africa).

N.—Dr. C. Neill (Bengal); Dr. F. Neal (British Guiana); Dr. Nightingale (Siam).

O.—Mr. Ozzard (Berbice); Mr. Ormond (Natal).

P.—Dr. G. Park (Penang); Dr. Paterson (Hebron); Dr. Pisani (India); Dr. Polson (Johannesberg).

R.—Mr. Reynolds (Gold Coast): Mr. Russell (Upper Assam); Mr. Rice (Stourport).

S.—Dr. B. Scheube (Greiz); Dr. Strachan (Brockley); Dr. Sandwith (Cairo); Surg.-Capt. Sutherland (Saugor); Staff-Surg. Stephens (Constantinople); Dr. Stubbert (Sullivan Co., N.Y.); Mr. Sichel (Bighi); Mr. S. Kellett Smith (Liverpool).

T.—Dr. Turner (Gibraltar); Dr. Alex. Turnbull; Mr. Tottenham; Dr. Thornhill (Ceylon); Dr. Torrance (Syria); Dr. H. F. L. Taylor (Edinburgh); Mr. A. R. Todd (Jamaica); Dr. Thomson (Hong Kong).

V.—Dr. S. Vine (Jamaica).

W.—Mr. V. L. Watts (Bengal); Dr. Wingate (Amoy); Dr. W. de W. Wishart (Demerara).

Y.—Major Yarr; Mrs. Yardley (India); Major G. H. Yonge (London).

Births, Marriages and Deaths.

The charge for inserting announcements of Births, Marriages and Deaths, is 3s. The notice should be accompanied by a remittance.

BIRTHS.

ADAMI.—On July 25, at Peel Street, Montreal, Canada, the wife of Professor J. G. Adami, M.A., M.D., of a son.

CLARK.—On July 17, the wife of Sir Jas. R. Andrew Clark, Bart., of Tidmarsh, Berks, of a son.

CURRIE.—On the 5th inst., at Edinburgh, the wife of Oswald James Currie, M.B., M.R.C.S., Maritzburg, Natal, of a son.

PISANI.—On July 10, at Aligarh, N.W. Provinces, India, the wife of Surgeon-Major L. J. Pisani, F.R.C.S., Indian Medical Service, of a son.

MARRIAGE.

ATKINSON—EASTMOND.—On August 3, at St. John's Cathedral, Hong-Kong, by the Rev. R. F. Cobbold, M.A., John Mitford Atkinson, M.B.Lond., Principal Civil Medical Officer, Hong-Kong, son of the Rev. S. Atkinson, M.A., of Portland, to Clara, eldest daughter of James Eastmond, of Puddyngton, Devonshire.

DEATHS.

MACCALLUM.—On July 1, at the Government Civil Hospital, Hong Kong, Hugh MacCallum, Secretary to the Sanitary Board, Hong Kong, aged 45.

PECK.—On July 28, at Malta, Surgeon William Peck, R.N.

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2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

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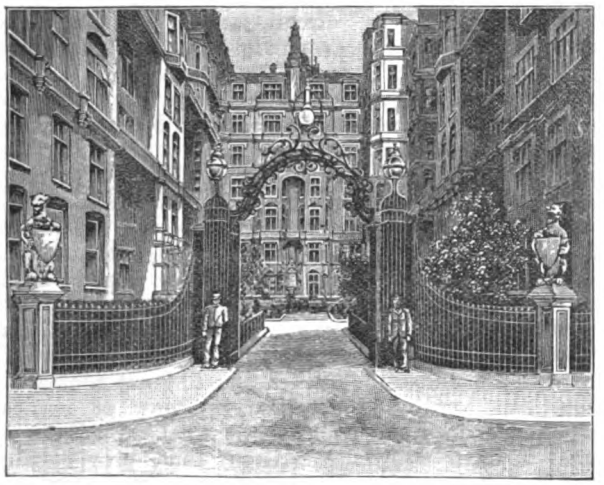

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SPECIAL ACCOMMODATION FOR INVALIDS.

Original Communications.

ENTERIC FEVER AMONG BRITISH SOLDIERS IN INDIA.[1]

By Sir JOSEPH FAYRER, Bart., K.C.S.I., M.D., F.R.S., &c.
Honorary Physician to H.M. the Queen; late Physician to the Secretary of State for India in Council.

IN the year 1895 there were 71,031 British troops serving in India, distributed in four commands, as follows :—

Bengal	22,259
Madras	13,417
Bombay	15,622
Punjab	19,713

Of these, 1,870 were admitted to hospital for enteric fever, of which 477 died. Thus by far the largest death rate was due to enteric fever, as is shown by comparison with the other death causes among that body of men during the same year. There were—

	Admissions.	Deaths.
Ague, Remittent fever, Malarial cachexia	24,794	39
Enteric fever	1,870	477
Simple continued	1,532	3
Dysentery	2,521	74
Hepatitis and liver abscess ...	663	102
Pneumonia	310	36
Phthisis	325	34
Sunstroke and heat apoplexy ...	243	60
Syphilis (secondary)	6,029	15
Cholera	44	32
Small-pox	19	2
Killed in action	—	13
Other causes	65,485	197
Total	103,835	1,084

[1] Dublin Congress of the Royal Institute of Public Health, August, 1898.

These troops were stationed all over India, from the Madras Presidency to the frontier and hill stations, thus being in a great variety of climates, from the humid heat of Southern India to the hot, arid atmosphere of the plains of North-West India, and the temperate climates of the hill stations.

Enteric fever is the great scourge of our young soldiers in India, and our army is essentially a young one, as will be seen by the following figures, showing the average strengths at different ages for the year 1895 :—

Under 20 yrs.	20—25 yrs.	25—30 yrs.	30—35 yrs.	35—40 yrs.	40 yrs. and upwards.
1,887	35,628	23,537	4,779	1,363	401

Of the total of 1,084 deaths, or 15·28 per mille, occurring among 71,000 chiefly young, vigorous, selected lives, 519, or 7·31 per mille, were due to fevers, of which 477, or 6·72 per mille, were returned as enteric, 42, or ·59, as malarial and simple continued fevers.

It will be obvious that, whatever the original cause may be, the influence of age in determining the incidence and severity of the disease is great. The susceptibility of the young soldier under 24 years of age is much greater than that of older men, and suggests the expediency of not sending men to India till after this most susceptible period has passed. The first and second years of residence add to this liability, for with increasing age and length of service there is a notable diminution in the cases of, and mortality from, enteric fever.

The ratio of mortality per mille was greatest from 20 to 24 years of age, the total number of deaths being 293, or 8·22 per mille; out of the total number that died at all ages from enteric fever 77 per cent. were between the ages of 20 and 24. The ratio of mortality also was highest in the first year of residence, the total number of deaths being 141, and the rate per mille 10·83; of the total number that died of enteric fever at all periods of residence, 37 per cent. were in the first year of residence.

As to the influence of locality on the disease, the death-rate from enteric fever in all the Indian commands was as follows for the year 1895 :—

	Per mille.
Gangetic Plain and Chota Nagpore ...	37·8
Indus Valley and N.W. Rajputana ...	27·0
Hill Stations	26·6
Upper Sub-Himalayan	24·9
S.E. Rajputana, Central India, and Gujerat ...	24·7
Deccan	21·7
Hill Convalescent Depôts	16·6
Southern India	10·3
Western Coast	9·6
Bengal and Orissa	11·0
Burma Coast and Bay Islands ...	8·9
Burma (inland)	4·9

As to season, the six months from April to September (inclusive) for the period 1886—1895 showed the greatest number of deaths, with two maxima, in May and August.

With regard to milk, which, in this country at least, is so often regarded as a vehicle for the morbific agent, it is to be noted that in India women and children, who probably drink more milk than men, suffer from enteric fever in a much smaller ratio than the men, and their comparative immunity is given as a reason for not considering milk to be a frequent

vehicle of the infection, leaving it to be inferred that additional causes arising out of the exposure, surroundings, and occupations of these young and susceptible men must be held accountable for the greater frequency of the disease in them. In the period 1886—1895 there were among the men 19·6 admissions and 5·28 deaths per mille; among the women 6·8 admissions and 1·82 deaths; among the children 3·5 admissions and 0·69 deaths.

The greater prevalence of enteric fever in India as compared with some other countries is shown in the following comparative statement:—

Deaths per 1,000 from enteric fever in 1894.

India.	United Kingdom.	Gibraltar.	Malta.	South Africa and St. Helena.	China and Straits Settlements.
5·75	·21	1·27	1·52	2·55	None.

In view of all these facts a question of interest arises: Is this enteric fever etiologically and pathologically identical with the enteric fever of Europe and other temperate climates, which is generally now considered to be dependent on a microbe for its origin? Or is it not possible that the phenomena, which so closely resemble, if they are not altogether identical with, those of the typhoid of this country, may be developed by causes connected with high temperature, moisture, malaria, and other climatic conditions which act so potently in India or other hot climates on the susceptible constitution of the European in early life, and which, under the added influence of impure water and insanitary surroundings, become intensified in their action?

My own opinion has long been that it is so, for it is difficult to divest oneself of the impression derived from examples of this disease which occurred where it was impossible to trace it to fæcally contaminated or otherwise impure water, and where the persons infected have contracted it in localities never before occupied by men, that the peculiar phenomena which have given the disease the name of enteric may have been developed by other conditions than those which seem to be the cause of it in this country.

Other writers have held the same view, for example, Professor McConnell, who said:—"I am inclined to believe that the evidence of a specific poison is not nearly so generally available here as in Europe; that probably climatic influences, *plus* want of proper sanitation, give rise to it in India; that in not a few cases the etiology seems to differ from that assigned to it in Europe, namely, specific fæcal contamination; and that it may possibly arise from climatic causes, combined with non-specific fæcal evacuations or other poisonous material, the result of an insanitary condition of dwelling houses, cesspools, drinking water, and other sources of contamination." Dr. Bryden, who said:—"I made the generalisation that the typhoid fever of British soldiers in India is primarily due to climatic influences." And Dr. Marston, who said:—"Indian experience has compelled me to recognise that those views as to the causes of enteric fever are too exclusive, and quite inadequate to account for the facts; they do not cover anything like all the facts, and they are irreconcilable with some of them," and many others to the same effect.

Is it not possible, it may be asked, that malarial infection may give rise to these phenomena, and that a certain number of cases which began with the symptoms of simple malarial, intermittent, or remittent fevers may under certain conditions assume the enteric form. Such is the view that I have long been compelled by circumstances to take, and apparently much the same view was held by Leon Colin, Woodward, Gordon, and others, and such is probably the explanation of the fever now reported as prevailing among the American troops in Florida, which is designated as "malarial typhoid." The previous as well as the subsequent history of many cases seem to warrant this conclusion, and whilst fully admitting the bacterial origin of the typhoid of this country, I am under the impression that a considerable proportion of the 477 men who died in India in 1895 with enteric symptoms may have owed these conditions to other causes than a bacillus, and that recent investigations which seem to trace all enteric fever to a bacillus, though entitled to great consideration, can hardly be accepted, in view of clinical experience, in India at least, as final. I adhere to what I have elsewhere written, that, provisionally speaking, two forms of fever with enteric symptoms and lesions may be admitted.

(1) The specific enteric fever, or typhoid.

(2) Malarial, or tropical enteric, or typhoid; the distinction of one from the other being difficult and not always practicable. Of course it is quite possible that other forms, the result of the action of both poisons, or of pythogenic and miasmatic causes combined with heat and other climatic influences, may occur.

I submit this with much deference for further consideration, and am well assured that from the able bacteriologists who have been and are now interested in the subject, which is one of much importance from many points of view, we shall ultimately receive a definite reply.

I have restricted this short paper to few facts respecting enteric fever as it prevails in India, and have stated the view which my own personal experience has suggested as to its possible etiology. The whole subject of Indian and tropical fevers, however, needs further elucidation, and this paper is merely offered as formulating propositions that may educe discussion which will throw further light on the subject.

[All the statistics in this paper are taken from the *Annual Report of the Sanitary Commissioner with the Government of India for* 1895, with the exception of those relating to average strength at different ages, which are taken from the *Army Medical Report for the Year* 1895.]

GASTRIC JUICE IN THERAPEUTICS.—Freimont, in the *Gazette Medicale de Paris*, mentions the potency of fresh gastric juice taken from the stomach of a dog. A case of cholera morbus was cured in a few hours by its exhibition; and in acute enteritis, enlargement of the liver and the emaciation consequent on typhoid, the remedy is declared to be effective.

MALARIAL SCURVY.

By Colonel K. Macleod M.D., LL.D.

Professor of Clinical and Military Medicine, Army Medical School, Netley.

The subject of scurvy has occupied a very prominent place in Indian medical literature. An excellent synopsis of all that has been written on the matter up to the year 1886 will be found in the late Dr. Norman Chevers' "Commentary on the Diseases of India"[1]; and a good description of the disease as observed in India, especially in Central and Western India, is given in the late Surgeon-General Sir William Moore's "Manual of the Diseases of India."[2]

The circumstances under which scurvy has been observed to occur in India on a large scale may be summarised as follows:—

(1) Sailors arriving in India after long voyages frequently suffered in former years from scurvy in very typical and severe forms, sometimes to such a degree as to render the navigation of their ship difficult or impossible. In later years, owing to shorter voyages, improved hygiene, better dieting and the use of lime juice, sea scurvy has become rare in the mercantile marine; but cases are still met with among sailors landed from sailing vessels in the hospitals of Indian seaports.

(2) Soldiers used similarly to suffer from scurvy on sailing transport vessels. Steam transport and better feeding have put an end to this.

(3) Outbreaks of scurvy have occurred among European troops from time to time, both in cantonment and on service. The Burmese wars of 1825 and 1835 were peculiarly disastrous in this respect; the scorbutic deterioration caused by improper feeding, exposure, fatigue, and climatic influences manifesting itself in extreme cases in the shape of sloughing dysentery and gangrenous ulcer. In recent times scurvy has very rarely occurred in the European Army in India.

(4) Native troops have suffered severely from scurvy both in cantonments and on service. In the Burmese wars they were more seriously affected than their European comrades, and even during the frontier operations of recent years, scurvy has been very common among them, though in milder forms. Dieting arrangements are, except on service, left to the men, and they are prone to save money by restricting their food. Hence they still frequently become scorbutic, more especially in times of famine, when the price of provisions rises, or at stations foreign to them.

(5) Scurvy used to be a scourge of Indian prisons, and both directly and indirectly to give rise to great mortality. Improved hygiene and dieting have largely reduced the number of cases of scurvy among native prisoners, but scorbutus is still responsible for a very material share in the pathogeny of jail maladies.

(6) Among Indian populations scurvy prevails extensively in certain places and at certain times. In the north-western portion of the Indo-Gangetic valley —in Sind and Marwar—the disease may be said to be endemic.

Deficient and improper food and brackish water are the special causative conditions. In these and some other parts of India scurvy in mild form is habitually present, but when scarcity or famine arises the scorbutic dyscrasia presents itself almost universally, and in those aggravated forms, ulcerative and phagedænic, which imply profound tissue enfeeblement, or poisoning, or both. Sometimes the disease has presented itself with such generality and severity as to suggest the idea of its being epidemic.

Most of the scurvy which is met with in Indian jails is, in these days, imported from without, and is simply an index of very extensive scorbutic disease among the native population. Indian statistics are silent as regards the extent of prevalence of scurvy in the native community at large, but the official returns for the year 1896[1] include 115 cases and 4 deaths among 110,090 prisoners; 534 cases and 10 deaths among 128,187 native troops, and 18 cases and no deaths among 70,484 European troops.

The foregoing paragraphs refer to true scurvy, mainly of dietetic causation, often conditions—such as impure (especially brackish) water, climatic inclemencies, hygienic defects, exposure and fatigue—aiding in its development. But there is in India a state of blood and tissue deterioration very closely resembling the scorbutic, if not identical with it, associated with, if not produced by, malarial disease, which claims special attention and study. This state is characterised by anæmia, marasmus, enlarged spleen, fever of irregular type, spongy and ulcerated gums, which bleed spontaneously or after very slight mechanical injury, a tendency to bleeding from other mucous surfaces and from sores and wounds; anasarca in some cases, diarrhœa and dysentery occasionally, and great impairment of tissue vitality resulting in ulcerations and gangrene on very slender provocation. Cases of this sort occur in malarious years and seasons, and are met with in all parts of India. In severe outbreaks of malarious disease such as occurred in Burdenau and the adjoining districts in the 'sixties and 'seventies, this form of malarial cachexia was peculiarly prevalent and severe, but it is always present in India and constitutes the chief variety of scurvy observed in Indian jails. Dr. Allen Webb[2] of the Bengal Medical Service was the first to point out the association between malarious spleen and scurvy, and he held that this association was a causative.

A good description of this special form of scurvy will be found in the *Indian Medical Gazette* for August, 1886,[3] by Dr. R. D. Murray, who points out that in these cases the gum disease is the chief index of the scorbutic dyscrasia, that post-molar ulceration is a frequent occurrence, and that the petechiæ and

[1] "A Commentary on the Diseases of India," by Norman Chevers, C.I.E., M.D., F.R.C.S. London: J. & A. Churchill, 1886, p. 336-355.

[2] "A Manual of the Diseases of India," by W. J. Moore, C.I.E. Second Edition. London: J. & A. Churchill, 1886, pp. 460-490.

[1] Report of the Sanitary Commissioner with the Government of India for the year 1896.

[2] "Pathologia Indica," by Allan Webb, B.M.S. Calcutta: Walker & Co., 1848.

[3] "Malarial Scurvy and the Post-Molar Ulcer," by Surgeon R. D. Murray, M.B. *Indian Medical Gazette*, July, 1886.

muscular and articular effusions which mark severe cases of true scurvy are very rare.

In this class of cases, as occurring among natives, it is difficult if not impossible to dissociate dietetic and malarious influences, and to say how much of the scorbutic dyscrasia is due to one and how much to the other. Is the scorbutic condition a pure outcome of the blood and tissue deterioration set up by the malarious infection? or is it a manifestation of a latent scorbutic taint elicited and enhanced by the malarial disease? or is it due to dietetic defect and error, such as is common with natives while suffering from disease —to innutrition consequent on the combined effects of malaria and bad feeding?

Among the invalids received in the Netley Hospital from India there is always a large proportion of soldiers who have suffered from malarious disease, and who present on arrival some form or degree of malarial cachexy. In many of these, swollen, spongy and ulcerated gums, with occasional bleeding, are observable, and their condition is as a rule associated with enlarged spleen and anæmia. Now the British soldier is well fed in India, in health and in sickness, in quarters and in hospital, and in the causation of this condition the dietetic element may safely be eliminated. If so, replies to the foregoing questions may be supplied by experience gained from a community in which scurvy is not likely to be present in latent form, or to be developed by improper feeding during illness.

I have selected the following as a well-marked illustration of the "malarial scurvy" met with in the Netley Hospital.

Lance Corporal I. C., 2nd Royal Munster Fusiliers, aged 23, nine years' service, of which 6$\frac{4}{12}$ were spent in India—at Cawnpore, Barrackpore, and Dum Dum. Invalided from last-named station March 10, 1898; admitted to Netley Hospital April 16, 1898.

History.—Admitted into hospital at Barrackpore four times in October and November, 1897, for quotidian ague. His spleen became enlarged, and he got emaciated and anæmic. On admission at Netley he was wasted and pallid, suffering from fever of an irregular type, with great enlargement of spleen. He had occasional attacks of diarrhœa, and in May presented symptoms of congested lungs with hæmoptysis. In June his *condition* was as follows:—Emaciation and anæmia persist; gets a slight rise of temperature (100°) every second evening; teeth covered with sordes; gums spongy and ulcerated, bleed readily; spleen enlarged and tender, reaching almost to the middle line; appetite good; bowels normal; no cough; no sign of active lung disease; no tubercle bacilli in sputum; no anasarca or dropsy; urine normal; no spots on skin or any sign of muscular or articular swellings (no history of either); no jaundice; liver normal.

Examination of Blood.—No parasites in red corpuscles; white corpuscles few; lymphocytes only; coagulability decidedly impaired (8 minutes); acidity normal; considerable diminution of lime salts.

Treatment and Progress.—He was treated with fresh vegetables and fruits, quinine and iron and chloride of calcium. He is now (September, 1898), about to leave the hospital, having been pronounced by a Board unfit for further service. He is free from fever; spleen smaller and softer, but still perceptible below cartilage of ribs; gaining weight; no pulmonary symptoms; much less pallid; gums less swollen and healing; no sordes on teeth; some bleeding still goes on, especially at night; no spots or swellings. Clinically the state of the gums in this case was precisely the same as in genuine scurvy of dietetic causation. The latest theory regarding the pathology of scurvy is that which will be found here clearly and ably propounded by Professor A. E. Wright, in a paper appended to the Report of the Army Medical Department for the year 1895.[1] Dr. Wright holds that scurvy is an acid intoxication, due to consumption of food yielding more mineral acid than can be neutralised by the alkalis of the food and blood, and the waste products of the body (ammonia). This acid intoxication is indicated by excessive acidity of blood and urine, and defective coagulability of blood. In this case there was no excess of blood acidity, but there was decided reduction of coagulability and diminution of lime in the blood. The paucity of white corpuscles was also a marked feature. As far as this observation goes, therefore, it would lend support to the view that the conditions known as "malarial scurvy" is different in nature and causation from true scurvy; but even if future observations justify the conclusion, there still remain the large class of cases in which the malarial and scorbutic elements are combined, and of which Chevers writes:—"Scorbutus often gives deadliness to Indian fevers, and Indian scurvy never rages more obstinately than among the subjects of malarious cachexia."

REMARKS ON THE NATURE OF ZANZIBAR FEVER.

By Gerald Sichel, F.R.C.S.Eng., Surgeon R.N.

Sent for publication by the Director-General, Medical Department, R.N.

These remarks are the outcome of an experience gained chiefly on board one ship (H.M.S. "Gibraltar") during a stay of about four and a-half months (1896-7). We arrived at Zanzibar during the "lesser rains" or "vuli," and stopped there during what is considered the most unhealthy season of the year. As regards the climate (observations taken at 8 a.m. each morning), the temperature averaged 81° to 82°, barometric pressure 30·07″ to 30·08″, and humidity 83 to 84 per cent. of saturation.

Another important fact to bear in mind is, that Zanzibar is a coral island surrounded by coral reefs.

A letter from Captain Wharton in Darwin's "Coral Reefs," p. 256, says: "Zanzibar seems to me to have undergone several motions of subsidence and upheaval, the latter being the latest; it appears now for many years to have been nearly stationary." It seems possible that the well-known unhealthiness of this coast is due to that fact, viz., that upheaval was the last motion of the reef, whereby dense masses of

[1] On the "Pathology and Therapeutics of Scurvy," by A. E. Wright, M.D., Appendix No. XIII. to Army Medical Department Report for the year 1895.

organic matter are placed in conditions no longer compatible with life, but which are capable during their decomposition of emitting injurious emanations, and that owing to the dense structure of their surroundings this emission probably goes on for years.

Now three points about the fever met with at Zanzibar struck me very forcibly:—(1) Quinine was generally useless; (2) the absence of definite aguish attacks; (3) the irregular and often undulant course of the pyrexia. The first and third facts, coupled together involuntarily, turns one's thoughts to Mediterranean fever; the first and second are both against a malarial origin. The fact that I never found anything abnormal in the blood of any of the patients I examined goes for little, owing to the fact that my highest objection was only $\frac{1}{6}$, and moreover, it was practically my first experience in searching for Laveran's parasite. In connection with the first hypothesis, namely, that this fever is allied to, if not identical with, Mediterranean fever, it will be interesting to compare the various general conditions existing at Malta—the home *par excellence* of this fever—and Zanzibar. First, however, I must adduce two facts in favour of my hypothesis, that the latter island is practically impregnated with decomposing organic matter. In the first place I would point out the blackened rocks and low cliffs to be met with anywhere on the coast, and even in the centre of the island; these are evidently of old coral formation, and the discolouration looks as if it were due to nothing less than sulphurous fumes. In the second, in one of the numerous small coral islands surrounding Zanzibar, and one often resorted to by the white inhabitants in search of a sea breeze and health—it is said that the gas can actually be seen bubbling up through the sand.

Well, against this condition of things, we have in Malta an island of soft sandstone, which from hundreds of years of faulty drainage is probably a veritable sponge, soaking in gross organic impurities.

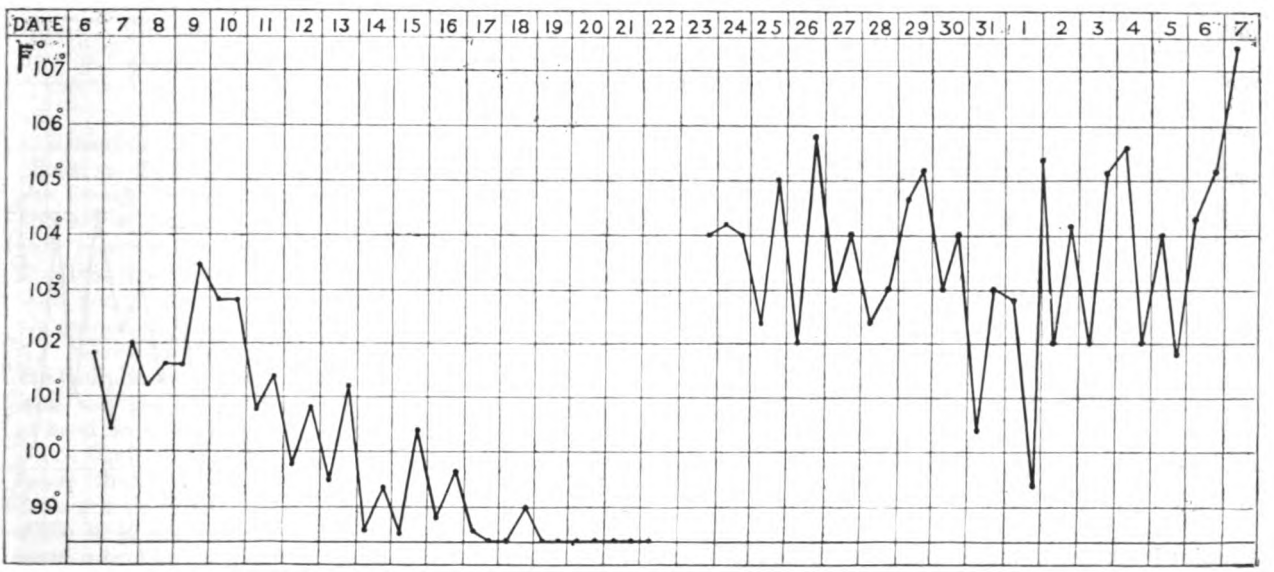
For the comparison of the meteorological data, I have taken May, June, and July of this year (1898), the averages of which work out as follows:—temperature 73°, barometric pressure 29·74″; relative humidity, taking saturation at 100, 82.

I have a strong opinion myself that the humidity of the atmosphere has some relation to the prevalence of Mediterranean fever, and it will be seen here that the humidity at Malta and Zanzibar for the periods taken is almost identical.

At Malta both the temperature and the barometric pressure were considerably less than at Zanzibar.

Now as to the fever itself, my observation that quinine was usually quite unavailing was borne out by Dr. Charlesworth, the Consular surgeon, who told me that this coincided with his own experience.

Cases.—No. I.—W. B. R., aged 28, admitted to sick list December 6, 1896. This patient was on the sick list from October 15 to 17 in the Red Sea, with vertigo due to heat. On December 6 he again went sick with headache and fever; neither liver or spleen

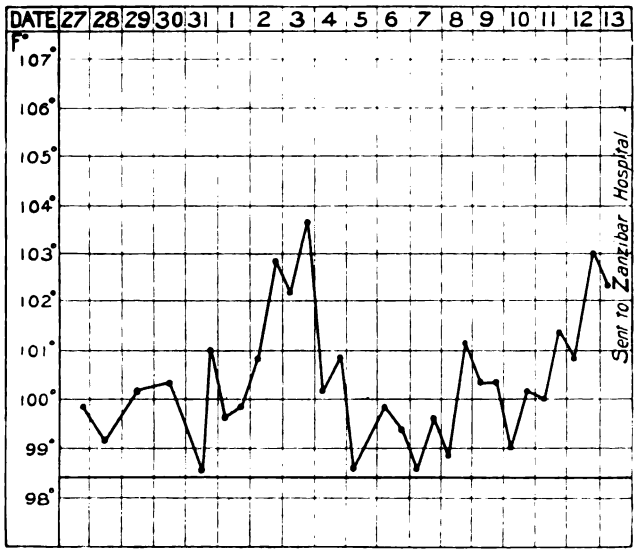


were enlarged, and on quin. sulph. grs. x., t.d.s., he appeared to recover and was sent to duty on December 22; on the 23rd, however, he returned with temp. 104°, although he had only been employed in the fresh air and not below deck. He was at once sent to the French hospital. There his temperature remained continuously high, at first amenable to antipyrin and large doses of quinine, and accompanied by a considerable amount of delirium at night. Two days after admission the urine was found to contain a large quantity of albumen, and this persisted to the end. On the 29th and 30th he was packed in ice. He steadily lost ground, pneumonia and other signs of failure set in; he took ample nourishment throughout, and stimulants as well, but in spite of this he gradually sank and died January 7, 1898.

Had this case occurred in the Mediterranean, it would almost certainly have been returned as a malignant form of Mediterranean fever; in the second part of the attack, or relapse, there are two waves to be seen, in the second of which he died from hyperpyrexia. I do

not think that this appearance is solely due to the ice-pack, as the greatest fall occurred two days after it had been left off. Another point is the comparative comfort of the patient until the very end. This is, I think, a condition more marked in Mediterranean than any other fever. He was an anæmic, weakly subject.

No. II.—E. S., aged 32, also a weakly subject, was put on the sick list on Oct. 27, 1896, suffering from subacute rheumatism of both ankles. He had frequently had rheumatism before and often suffered from sudden attacks of syncope on the slightest exertion. A slight whiffing apical systolic bruit, just traceable into the axilla, was heard on one day only—but this disappeared. On two occasions his urine

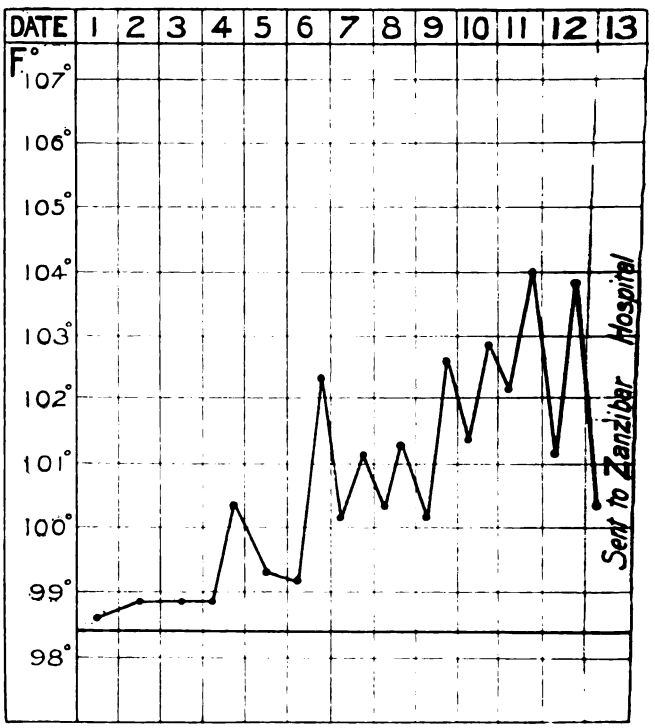


contained albumen, the second time possibly due to the large amount of salicylate of soda he was taking. He was very susceptible to cinchonism, 20 grains of quinine giving him deafness, buzzing in the ears and dyspnœa.

The swelling of the ankles and the fever resisted all treatment. He complained of excoriation of the scrotum from rubbing of his clothes, and two very unhealthy ulcers formed there; these much improved under treatment. I could find no malarial parasites in the blood. He was sent to the hospital on shore on November 13. There the ankles slightly improved, but he developed synovitis of left knee. He continued very weak, and was invalided from the station on November 30, 1896. This case was ailing when we arrived at Zanzibar, but still, as he seemed to develop the trouble whilst there, I think he may fairly be included here. The pyrexia, whilst under my observation, showed two marked undulations.

The resistance of the rheumatism to salicylates and of the temperature to quinine look remarkably as if the case were really one of the rheumatic variety of Mediterranean fever. It is only just to here remark that H.M.S. "Gibraltar" was sent straight from the Mediterranean to Zanzibar.

No. III.—C. B., aged 15, admitted to the sick list November 1, 1896. The course of the fever suggested typhoid, but there were no spots or abdominal symptoms. Splenic dulness was enlarged to one finger's breadth from the costal margin, and later right up to the edge of the ribs. No malarial parasites could be made out in the blood, and urine was free from albumen. He was sent to the French Hospital, where his temperature continued to vary from 100·4° in the mornings to 103° to 104° in the evenings until November 24, after which it remained normal in the mornings and 101° to 102° in the evenings. He was greatly weakened, and was invalided from the station November 30, 1896.



The above chart will show how the temperature at first suggested typhoid. Anyone at all familiar with Mediterranean fever will know how difficult the diagnosis between the two diseases often is. Beyond the commencement of the pyrexia, however, there was nothing to suggest that this was enteric.

No. IV.—C. H., aged 24, admitted November 15, 1896, discharged to the French Hospital November 17. Looked strumous, but had no physical signs of phthisis. Splenic dulness increased forwards about one finger's breadth. Gradual onset, general febrile symptoms. Pyrexia entirely unaffected by quinine the two days he was under treatment before being sent to hospital.

Although I have notes of only one more case—of no

special interest—numerous other minor attacks of fever occurred over which quinine exerted little or no effect.

This fact, together with the others mentioned above, point to East Coast—or Zanzibar—fever being of the same nature as Mediterranean.

Of course, in a ship, it is impossible to carry out proper bacteriological tests, but if ever I should serve on that coast again I shall certainly do my best to obtain a series of Widal's reactions—should it be possible to do so—in any cases I may have.

The cases of fever were by no means confined to the ship, but were also plentiful ashore, and as far as I could ascertain were of much the same type—if such a term can be applied to a fever of so many varieties.

HÆMORRHAGE FROM THE BOWELS IN MALARIAL DISEASE.

By Capt. S. F. Clark, M.D., R.A.M.C.

Station Hospital, Hong Kong.

I should like to make some remarks upon a complication of malarial disease, viz., hæmorrhage from the bowels, which deserves more attention than it appears to have received from the profession. I say this because, though the life of a patient attacked by this bleeding is placed in great peril, yet many text books of medicine either ignore the subject altogether, or else do little more than merely mention it. More surprising still, however, is the fact that the complication has escaped notice by many medical men resident in the tropics. Why this should be so is hard to say, for anybody who has treated a case is not likely to forget the hand-to-hand struggle with death that ensued. It may be that medical men have not published their observations of such cases, while it is also conceivable that of late years such cases may have become of more frequent occurrence. Support is given to this view of the matter by the fact that some medical officers I have served under, of high rank and great Indian experience, had not met with this malarial complication until quite lately. I served in India for six years, and though I saw and treated hundreds of cases of malarial disease, it was not until my last year in Karachi, Sind, that I met with a case of hæmorrhage from the bowels due to malaria. This was towards the end of 1893. In 1894 I saw a man at Derlali who was convalescent from this disease, and in 1896 a very severe case occurred at Dover in a soldier just returned from West Africa. In Karachi the number of cases quickly reached double figures, and though the amount of malaria in the station was great and the type severe, yet I cannot explain why I passed five years in India without seeing or hearing of this complication, and then met with quite a run of it. The Karachi hospital records did not show any case of this hæmorrhage as having occurred in previous years, and it had not been noticed among the large native population of the town.

A typical case may be described thus:—A man is brought to hospital complaining of "fever and ague," and on examining him he is found to have an elevation of temperature, and his skin is hot and dry to the touch. He states that he has been suffering from ague a good deal lately, and the case appears to be simply one of malarial fever of an ordinary type. In all probability the patient is vomiting the greenish matter common in the hot stages of ague. He is put to bed and sooner or later expresses a desire to evacuate the contents of his bowel; this desire may be preceded by crampy pains in the abdomen. When he uses the bed pan the stool will be found to be copious, quite liquid, and of a bright red colour, with little shreds of solid matter floating in it. The copious nature of the stool causes alarm, which is not lessened when after a short interval another similar motion is passed, followed by more at short intervals. It would appear as if the whole of the blood in the patient's body was being passed *per rectum*. An act of vomiting is very liable to be followed by an evacuation of blood from the bowel. The stool comes away quickly and easily, without pain. In some cases the bloody motions are preceded by two or three liquid stools devoid of blood, but in other cases the first motion passed contains blood. If the hæmorrhage is not checked the patient becomes collapsed and dies, but the great majority of cases can be saved—even some which appear hopeless. I consider the presence of vomiting a very serious matter; it greatly hampers treatment, as any medicine given by the mouth is apt to provoke vomiting and bring on a movement of the bowels, and consequent loss of blood. The restlessness of the patient is also calculated to induce an action of the bowels. If the treatment causes the stools to come at longer intervals and to be less in amount, the patient can be saved, for the bleeding will soon cease altogether, while if the hæmorrhage gets out of control the patient will certainly die. The later phases of the case therefore vary with the effect of the treatment, and are similar to the symptoms attending great loss of blood in any other form. But even though the patient is almost pulseless and apparently in a hopeless condition, yet his life should not be despaired of.

The treatment of this grave affection must be prompt and energetic. In the first place, absolute quiet of the body must be obtained as far as the restlessness of the patient will permit of it. He must be laid flat on his back in a comfortable bed, and should be guarded from all causes of mental excitement. When he has a motion a slipper bed-pan must be very gently put in position to receive the evacuation. The theoretical treatment would no doubt be to let the patient stool into the bed and endeavour to catch the discharge in absorbent wool, &c.; but I do not think this is necessary. No cleansing operations should be done after the bowels have moved.

To check the bleeding both external and internal treatment is available. The whole abdomen should be covered by an ice bag or bags—the ice in small pieces and constantly renewed — melted ice is not wanted. I consider this external application of ice to be of the utmost value. Astringents—notably

tinct. opii and liq. ext. ergot.—should be given by the mouth. It is now that the presence of vomiting will be realised. In the injection into the tissues of ergotinum we have a powerful aid independent of the presence of vomiting. I consider that the use of this drug should never be omitted; it can be injected into the thigh muscles without any moving of the patient. All these drugs should be used freely and in full doses. Rectal injections of iced water I consider to be worse than useless.

As a rule the patient does not require any nourishment, but if the case is prolonged Brand's essences should be given in small quantities. The question of alcoholic stimulants is a difficult one, as their use may possibly increase or renew the blood flow. I think they ought to be withheld as long as possible.

The after-treatment includes rest in bed with avoidance of movement. Astringents ought to be given for two or three days after the bleeding has ceased, and quinine should be freely administered. The ice bags may be done away with twelve hours or so after the last appearance of blood. The food should consist of Brand's essence of chicken, while milk may also be used. Once the hæmorrhage has thoroughly ceased it has no tendency to recur.

Pathology.—In the one fatal case of which I have any knowledge it was found on examination that from stomach to cæcum the whole intestinal tract was in a condition of extreme acute congestion, gradually increasing in intensity from above downwards, while the mucous membrane was thickened, opaque, and of a deep purple colour, with almost black patches on the *valvulæ conniventes*. About five or six inches down the jejunum were a series of very small shallow ulcers with thin ragged edges. The same condition existed throughout the large intestine, except that there were no ulcers. It is evident that the great hæmorrhage that occurs cannot be solely from these few small ulcers; in my opinion it comes from the vessels of the acutely congested intestinal mucous membrane, and I shall now touch upon this point.

I believe that the intestinal hæmorrhage is due to changes in the blood caused by the malaria, just as hæmorrhages occur in the acute infectious diseases. Roberts includes as a cause of melæna "diseased conditions of the blood," and Whittaker says that hæmorrhage from the bowels in acute infectious diseases is due to disorganisation of the blood by micro-organisms which "touch the life of the blood corruptibly." In malaria we have Laveran's micro-organisms present. Osler states that they destroy the red blood corpuscles, therefore surely we have here the micro-organisms "which touch the life of the blood corruptibly," and so cause the hæmorrhage from the bowels.

How does the blood reach the bowel when *post-mortem* examination does not show any great breach of surface? Simply by making its way through the vessel wall without any solution of continuity occurring in that wall. In an inflamed tissue, not only fluids, but the blood corpuscles themselves make their way through the walls of the vessels, and under the microscope this process can actually be seen going on in the inflamed mesentery of a frog. I consider that in this way a large quantity of blood escapes into the cavity of the bowels from the acutely congested capillaries distributed to the intestinal mucous membrane.

Papers read at the Annual Meeting of the British Medical Association.

PLAGUE IN INDIA.

By Professor W. J. Simpson, M.D.

In opening the discussion on the plague in India there is one point upon which I am sure we are unanimously agreed, and that is the great admiration we all feel for those Englishmen and Englishwomen—and by English I include Irish and Scotch—who have displayed so much devotion and courage under circumstances of most exceptional difficulty and alarm. Officers, both civil and military, soldiers, ministers, missionaries, nurses and doctors, have distinguished themselves in no common degree in their fight against an invisible and destructive foe. As a profession we may be proud of the achievements of the medical men, both European and Native, official and non-official, who, short handed, have laboriously done all in their power to mitigate the effects of the disease and check its ravages; and equally with the profession I would include that essential auxiliary, the staff of sisters and nurses, whose noble work in the hospitals in nursing the plague-stricken Indians ought never to be forgotten. Such exceptional medical services, whether scientific, preventive or curative, it is to be hoped will be liberally recognised by some mark of distinction, both by the Home and Indian Governments.

In the short time at my disposal it will be necessary for me to limit my remarks on plague to only a very few subjects, and consequently I shall not deal with many of the well-known facts concerning this disease, nor with the clinical aspects of its different varieties, nor with the rapid advances in our knowledge of its bacillary nature, since the discovery of the microbe by Kitasato; nor even, enticing though it be, with Haffkine's brilliant discovery of a plague prophylactic, which in the observations that have been made shows a reduction in the mortality between the inoculated and the non-inoculated of over 80 per cent. I do not touch on this subject, because I observe that the distinguished scientist has sent a paper on plague to this meeting of the Association.

I shall accordingly confine my remarks first of all to some of the phenomena connected with plague that are still obscure, and which require special research for their elucidation; and secondly, to the increasing importance to India and to England of India's possessing a thoroughly trained health service, in which shall be included along with the European, the best native medical element of the country.

With reference, then, to my first subject, it may be taken as an axiom, I believe, which no one doubts, that sanitary measures exert a most powerful influence on plague; that social conditions also play an important part; that the incidence of the disease is more severe on the inhabitants of dark, damp and filthy dwellings, such as are to be seen in their worst form

in the large cities of the East; that overcrowding favours its spread, and that sanitation in its widest sense is the best agent to combat the disease. At the same time, I think it must be admitted that our knowledge of the disease is very imperfect, and that there are other powerful factors which, owing to unfamiliarity with the disease, there is a tendency to minimise or ignore, and which must be counted with and studied if the disease under the present conditions of the East and West is to be brought under control. In considering these factors it may be necessary to revise some of our views concerning plague.

Not long since it was a current and favourite doctrine that the plague of London was stamped out by the fire of London, which gave a healthier city. Such an explanation appealed to the sanitarian, but when tested as to its truth the fact could not be overlooked that plague continued in London for thirteen years after the fire, that it disappeared as quickly from other towns in England as from London, and that the newly built London was little better than the old, for Sir Christopher Wren's plans were not carried out. Again, in the recent outbreak in Bombay, plague was considered a local disease which would not spread. Its manifest slowness in reaching any general proportions was taken as a sign of its incapability of diffusion, the fact being overlooked that this is one of the important characteristics of most plague epidemics. It is easy to be wise after the event, and none can gain credit now by showing that plague has spread over the whole of Western India, even up to the North-western Provinces. It is possible, however, that some light may be thrown on this subject by viewing it from its epidemiological aspect, and in this connection it will be useful to compare the liability of India to plague with that of Western Europe.

The information concerning early outbreaks of plague in India is very meagre and furnishes little material for comparison with the outbreaks in Europe, yet the little that is to be gathered seems to support the view that plague in India synchronised often if not always to some extent with the great diffusions or pandemics in Europe, and in this respect India, like Europe, was subject to destructive occurrences at long intervals, and shared in those sudden cessations of the disease which were conspicuous in Western Europe in the 17th century. Though the reports are meagre, there is no doubt that plague is not new in India. It is recorded that the Justinian plague of 542 A.D., which is supposed to have arisen in Ethiopia and Egypt, not only spread to the West along the coast of Africa and over Europe, but also to the East over Syria, Persia and the Indies. Plague, which from the earliest times has been associated with trade, merchandise and commerce, or with movements of large bodies of men, followed in this pandemic the general lines of intercourse of those days. Although no special pandemic is noted between 542 and 1334, the epidemics in Mahommed Tughlak's time, and later in 1399, being probably parts of or remnants of the black death, which is believed to have arisen in China, and is stated to have been very destructive to the inhabitants of India, yet the disease must have been well known to the Hindus, for one of the Purans, written at least 800 years ago, gives instructions to the Hindus as to the precautions they are to take in the event of an outbreak of plague, and one of these instructions is particularly interesting, because it shows that the authors were familiar with one of the methods of spread of the disease and with the precaution that was to be taken against it. It is to the effect that *whenever they observe a mortality among rats they are to leave the locality*. If we were to judge from a perusal of some of the recent literature on plague, the connection between rats and plague is a new discovery, whereas it is only a re-discovery of a fact which was known to the Hindus and as I shall later show, has been observed from the most ancient times. Plague is also noted as prevailing in some parts of India in the 15th and 16th century.

Plague prevailed in Bombay City and its environments in the 17th century, from 1689 to 1702, forming a part of that general eruption which is recorded as having prevailed in Western India, and possibly even earlier in some parts of India, from 1684 to 1702. It corresponded in time with a wide diffusion of plague in Persia, the eastern branch of an activity in Syria, which had its western branch in a wide diffusion in Europe in the 17th century. Surat at that time was commercially, for India, a more important town than Bombay, and was attacked with plague in 1684, *i.e.*, five years before Bombay. It possessed all the unwholesome conditions which have been observed to favour the prevalence and virulence of plague. Crowded and unclean, the streets were usually narrow, and in many places covered with excrement of men and beast. Fryer, who visited Surat some time before the outbreak, wonders that a city whose people make the streets a dunghill should never have been visited by the plague. The disease, when it was imported in 1684, continued for six years without interruption, varying in intensity at different seasons of the year. Subsiding during the rainy season, viz., from June to September, the epidemic broke out with fresh fierceness in October, and again abating the greater part of the cold and hot seasons, raged with renewed fury towards the end of May. The death-rate at times, on a very modest calculation, amounted to 300 a day. The same remarkable immunity of Europeans was noticed in the epidemic of Surat as that exhibited in the recent epidemic in Bombay. It is mentioned that up to 1689 no Englishman had been attacked in Surat, which is in striking contrast to what happened in Bombay in 1690, for in regard to this city it is recorded that of 800 Europeans only fifty were left—six civilians, six commissioned officers, and not quite forty English soldiers. Bombay, that had been one of the pleasantest places in India, was brought to be one of the most dismal deserts. There is no record of the mortality among the natives in Bombay, a fact which indicates that mortality in the interior of the country, unless on an enormous scale, would not be likely to attract attention.

After the epidemic in the 17th century the plague seems to have disappeared from India as completely and as rapidly as it did from Western Europe, for it is not until 110 years later, at the beginning of the 19th century, that a small part of Western India, viz., Kutch, Kattiwar, Gujerat, and Sindh, were

again affected with the disease, which continued from 1812 to 1821. It is deserving of notice that this epidemic occurred at a time when plague became widely diffused in the Levant, spreading to the Lower Danube, Asia Minor, Armenia, and Northern Africa, and lasting nearly twenty years.

Nothing more is heard of the disease on the Western side of India until 1836, when the Pali plague broke out in Marwar in Rajputana, and lasted until 1838. The epidemic, which was limited in its nature, also corresponded in time with a fresh and comparatively limited activity in the Levant, which affected the Turkish dominions in Europe and Asia as well as Egypt, and it is to be observed that the disappearance of plague in Rajputana coincided with its decline and ultimate disappearance in the Levant. Plague was, however, discovered in 1823 as prevailing in the extreme North-west of India in the province of Kumaon and Gharwal, which adjoin one another, and are on the Southern slopes of the Himalayas. There is no information as to how long this centre of plague had existed previous to its recognition. It is possible that as the plague of 1896 in Bombay found its way in 1897 as far as Jullundur in the North-west, that the plague in Kumaon in 1823 was only a part of that which had prevailed in Western India in 1821. Whatever may be the date of its origin, there can be little doubt that Kumaon is now an endemic centre, plague having occurred in limited outbreaks even as recently as 1893. The last outbreak, which was in July and September of 1893, occurred in a valley some 6,000 feet high. Fortunately, this centre is a comparatively inactive one as regards its powers of diffusion, which is in favour, as I shall afterwards show, of its being a branch of the parent stock in the Levant. An epidemic in Hansi, in the province of Delhi, in 1828-29, and in Rohilcund, around Bareilly, in 1836-38, probably owed their origin to Kumaon. In connection with the possible antiquity of Kumaon as a plague centre, there is the fact that plague was epidemic in Delhi in the time of Jehangir. On the other hand, Delhi at that time was a large commercial emporium, having much dealings with the West.

The intervals of freedom from plague, so far as is known, are as great in India as in Western Europe, and would indicate that the disease is as much an exotic to India as to Western Europe. The tendency to linger a number of years in one locality or district can scarcely be taken as evidence of endemicity, but rather as manifestations of the same invasion in which the germs have not succumbed to the influence of the new environments. The same is seen with cholera, whose home is fairly well defined. Bombay, with its freedom from plague for one hundred and eighty-four years, shows a longer interval than Moscow, in 1771, which had not been attacked for one hundred and fifty years; or Marseilles, in 1720, after a lapse of seventy years; or London, in 1499, after an interval of one hundred and fifty years. These long intervals are worthy of attention because they show a vulnerability of towns, which from their long freedom might be considered invulnerable.

Moreover, the cessation of plague in Western India at the beginning of the 18th century was apparently as complete, as rapid and as remarkable, as the cessation of plague in Western and Central Europe at the end of the 17th century. At this period plague disappeared from the greater part of Western Europe in the course of ten years, and completely in thirty. A still more remarkable disappearance is that which took place towards the middle of the 19th century. Then, in the course of five years, from 1839 to 1844, plague disappeared from its old haunts in South Eastern Europe, the Levantine Countries and Egypt. Hitherto it has been impossible to satisfactorily trace these sudden disappearances to special measures devised for that object, and it must be confessed that it is not possible to be satisfied with the accuracy of the current explanations to which these disappearances have from time to time been attributed. In all the explanations it seems to be the case of the fire of London over again, only on a more extended scale. A favourite explanation, and one that has been repeated just lately by an eminent authority, is advancing civilisation. It is a pleasant and comfortable hypothesis, but it may be asked what advancing civilisation got rid of plague in India in the 16th and 17th centuries, and of plague in the Levant and Egypt in the middle of the 19th century in the course of five years?

The Levant and countries adjoining have been the centres of plagues for at least three thousand years, the first notice of the disease being in Syria, when the Philistines, after defeating the Israelites at the battle of Ebenezer, were affected with plague, which attacked city after city, causing a deadly destruction. As a propitiatory offering, the Philistines made *images of their emerods and images of their mice that marred the land.* It is evident from this reference that the mice were thus early considered to play an important part in plague epidemics. The plague of the Levant has lost not only its powers of diffusion but the power of retaining its hold on countries in which it had appeared for centuries almost as regularly as the seasons. Nor does there appear to be any indication at present to regain these powers, for in the several recrudescences which have taken place in Mesopotamia, Asia Minor, and in Northern Africa, it has been pointed out by Tholozan that they are restricted outbreaks partaking more or less of a local nature, showing no special aptitude for diffusion, and though retaining their fatality, localising themselves independently of quarantine. The plague at Resht and at Astrakan arrested itself before quarantine was introduced. The same cessation of plague of a localised character appears to have occurred in the western part of India, for after 1840 no more is heard of plague until 1896, and then not in connection with a recrudescence or fresh activity of a widely diffusive nature in the Levant, but with a fresh activity of a widely diffusive nature in China. The plague of 1896 in India had not the character of recent recrudescences in Mesopotamia, Persia, and Kumaon, which were largely local in their nature, but it possessed, and still possesses, powers of diffusion, which characterised the outbreak in China.

Apart from other considerations as to means adopted to prevent the spread of plague, it is an important question epidemiologically, whether this pandemic

of the far East has enough diffusive power to pass on into the Levantine countries and thence into Europe. If we were to judge by the events at Jeddah, where the plague, though imported, seemed to acquire no foothold, the answer might be no; but the data at present are insufficient for so decided an opinion. One of the features of plague is that it is a disease which is slow in its advancement. All that can be said is that this new recrudescence in the far East has apparently nothing to do with the old centres in the Levant, and it is necessary to be particularly careful not to be too much influenced by the experiences which the Levantine plague has presented to us for the past hundred years, otherwise there is the danger of repeating the mistake that was made in Bombay in 1894, of considering it a local disease unlikely to spread. The plague of the Levant during the past century was totally unlike the present plague that has arisen in China. The Levantine plague was, and is, distinguished as an inactive, contracting plague, belonging to a species that has had its day and is dying; the Chinese as an active expansive plague, full of potentialities, and only requiring the opportunities to manifest itself. The rapid spread of the disease in China and now in Western India is sufficient evidence of vitality and diffusive power. When plague broke out in Bombay in 1896 it spread from that city in every direction, infecting nearly the whole of the Bombay Presidency, Kutch and a part of Sindh. As shown by Surgeon-Captain Grayfoot, a large number of the localities were infected by imported cases, and that in many a considerable time elapsed between the first recognised case and the first indigenous case, and that again some time intervened between the first indigenous case and the subsequent epidemic. Thus at Sholapore the first recognised imported case was on December 17, 1896; the first recognised indigenous case on September 28, 1897, *i.e.*, more than eight months later; while the disease did not become epidemic until November, 1897. In districts adjacent to one another it was frequently noticed that the migration of rats, or the "rat's progress" as it has been called, seemed to play an important part in the spread of the disease.

Plague spread in a north-easterly direction as far as Jullundur in the North-west, where it was never permitted to assume an epidemic form. Possibly the disease was more easy to control and stamp out in the North-west, because villages and not large towns were infected and obviously the conditions are not alike; but the success, I think, may in no small degree be attributed to the enlightened policy of the Lieut.-Governor and his confidence in his medical advisers from the commencement. Immediately plague broke out in Bombay Sir Anthony Macdonnell sent Mr. Hankin from the Agra laboratory to study the plague bacteriologically, and allowed him to remain there until the North-west Provinces needed his services. Prompt attention to first cases, segregation, evacuation of the village, camping out, isolation of the village and disinfection, were the principal measures adopted, and Surgeon-Lieut.-Col. Thomson and his assistants are to be congratulated on the successful manner in which they have combated plague in the North-west.

The time and manner in which Bombay became infected is of great epidemiological and practical interest. Unfortunately both time and manner are involved in obscurity, and are likely to remain so. It seems to be the fate of most epidemics of plague in large towns for their origin to be obscure. Apart from the fact that the presence of plague is always very reluctantly acknowledged, because of the important interests that are likely to suffer, and that the disease is extremely slow in manifesting itself to any alarming extent; the obscurity may be due to the fact that some of the lower animals are affected with the disease in an unrecognised form, that the pneumonic variety in man, which was shown by Surgeon-Captain Childe to be produced by the bacilli attacking the lungs, may be mistaken for some other form of lung disease, and that there are mild forms of plague which are not easily recognised. The identity of the disease in rats with the plague in man was demonstrated by Dr. Surveyor, the bacilli from both responding to the same tests. Possibly the Chinese view regarding the gradation of animals infected, and the relation of the disease to the soil, though somewhat fantastically expressed, has much truth in it. In treating of the plague in London in 1603, Lodge mentions rats and moles and other creatures accustomed to living underground, forsaking their holes and habitations, and attributes it to corruption in the soil. Pigs have been shown by Wilm to be affected with the disease. Boccaccio mentions the death of two hogs from plague which he witnessed in Florence in 1348. Snakes are recorded as dying from the disease, and Nuttall has produced the disease in snakes experimentally. In many epidemics epizootics are also observed either to precede or coincide with outbreaks. Surgeon-Lieut.-Colonel Weir, the Health Officer of Bombay, observed this in Bombay, and gives statistics showing that in August preceding the outbreak of epidemic plague in September there was a heavy mortality amongst cattle, sheep and goats. Monkeys were affected in Hurdwar, and it is a curious fact mentioned in Surgeon-Major Lyon's report that the grey monkey was much more susceptible to the plague than the brown monkey. With these facts before us, it is evident that plague is not a simple disease to be easily dealt with administratively.

It is certain that the recognition of plague in Bombay in September, when it was described as a mild form of plague, was not its first manifestation, and the date has gradually been pushed back to August, July, June and May, and even to February, when, as described in Mr. Birdwood's able lecture at the Society of Arts, Dr. Kay, medical officer to the G.I.P. Railway Company, treated several plague cases at the Bycalla Infirmary of the Railway Company, which though he did not recognise them as plague cases at the time, his after experience convinced him had been really cases of plague. It is possible that the date may have to be pushed back even farther than February, 1896, for as Editor of the *Indian Medical Gazette*, I received in 1896 a letter from a medical officer on the Malabar Coast, describing some cases of peculiar fever with glandular enlargements under his care, I think in 1895, and over which he was much puzzled; unfortunately I mislaid the letter and was unable to

recollect the medical officer's address. Perhaps the mention of it at this meeting may bring this important matter to the medical officer's notice.

That a disease, such as plague, may exist in a mild form for a long time before it manifests itself, either in its more virulent type, or in a local outbreak of considerable dimensions, may be gathered from the occurrences in Calcutta. The facts briefly stated are, that the Shropshire regiment which distinguished itself in Hong Kong, in cleansing plague-infected houses, lost three of its number in that colony from the disease, while double that number were attacked. From that time the regiment at intervals suffered in small groups from fever accompanied by glandular enlargements. This illness they brought with them to Calcutta, in January, 1895, and then other drafts of soldiers, who had never been to Hong Kong, but who were brought in intimate association with their affected comrades, were attacked with a similar malady. At first the disease was diagnosed as syphilis, then as malarial fever with bubo, and ultimately the cause was registered as unknown. Some medical officers considered it to be a new disease; one of the medical officers was attacked in June of 1896, and the glands of the groin, axilla and neck were affected. In October, 1896, besides an imported case of illness from Bombay, with fever and glandular enlargements, a number of cases occurred in Calcutta, but with symptoms of a more acute form, and of less duration than the cases in the Shropshire regiment, which was still continuing to get fresh cases. There were fever, white-coated tongue with red tip and edges, congested eyes, dull intellect and glandular enlargements, mostly in the groin; children and young adults were chiefly affected. An adult who was affected and died, had all the typical symptoms of plague. From experience gained later in Bombay and Poona, I have not the slightest doubt that two other suspected cases which proved fatal were cases of plague, and that the other cases of fever with glandular enlargement were benign plague, more severe in its character than some seen by me in Bombay and Poona. A peculiarity of these mild cases was that in some, a bacillus similar to the bacillus of plague was found in the blood. It could not be cultivated, however, unless, as it seemed to me, large quantities of blood or serum was drawn. In one portion of the town, somewhat later, there was a glandular and very fatal illness among rats, whose organs were filled with diplobacilli, similar to the plague bacilli. Sick rats left their holes and slowly hobbled about or rested, apparently in a dazed condition. As many as 100 affected rats were counted in one small grain depôt in one day. The houses were treated as plague-infected, a campaign was waged against the rats, and evidently with success, for the epidemic among the rats ceased. It is necessary to state here that my views regarding the nature of this glandular sickness among the inhabitants was not held by a higher authority, and that isolation of such cases was considered unnecessary. On the other hand my further experience convinces me that the medical men who agreed with my views were right in their diagnosis, while some of the microscopical specimens sent to Kitasato elicited the opinion they were probably plague bacilli. Unfortunately the cultures I took to Haffkine were old and gave negative results. In connection with the occurrence of such cases in Calcutta, and their nature, the Report, dated July 16, 1897, of Dr. M. J. Rosenau, the Quarantine Medical Officer to the supervising Surgeon-General of the United States Marine Hospital Service, which I came across the other day, is interesting. It is as follows:—

"National Quarantine Station,
"Angel Island, Cal.,
"*July* 16, 1897.

"Sir,—I have the honour to report the British ship 'Annie Maud,' one hundred and forty-three days from Calcutta, was placed in quarantine to-day for disinfection. A short while after leaving Calcutta one of the crew was taken ill with swellings in the axilla, groin and elbow, and died. Two more of the crew suffered with buboes, from which they recovered.

"Very respectfully,
"M. J. Rosenau,
"*Passed Assistant Surgeon, U.S., M.H.S.*"

One other point is deserving of notice, viz., that in 1897 there was an epizootic outbreak among cattle in the same locality in which the rats had died some months before, and though it was called rinderpest at the time, the symptoms differed somewhat from the ordinary cases under that name, and *post-mortem* examination showed a very congested and enlarged condition of the mesenteric glands.

Plague has now manifested itself in larger proportions in Calcutta, and within three months has caused 128 deaths. It is to be noted that the cases are no longer mild, and that they are well distributed over different parts of the town. There was the same difference of opinion in regard to the nature of these cases of fever with glandular enlargements as in 1896, until Haffkine decided the question by reporting the nature of the bacillus.

The early cases in March and April of 1898, in Calcutta, were set down as cases of bubonic fever, which recent statements would lead one to believe is quite common in Bengal, and is only malarial fever accompanied by buboes. All that can be said about such cases, and I have made careful inquiries, is that some of the most experienced practitioners in Bengal have not come across these cases until a short time ago. The same erroneous mode of viewing mild cases of plague found favour in the Pali epidemic of 1836, but it was conclusively shown by Dr. Forbes that no such fever attacking the lymphatic glands was known in the Pali district or in that part of India, except in connection with plague. Dr. Forbes divides the Pali plague seen by him into four forms: (1) an ordinary bubonic; (2) a more violent and malignant; (3) a most fatal pneumonic; and (4) an extremely mild form in which the glandular swellings made their appearance with little constitutional disturbance and were attended only by languor, debility, and a general feeling of indisposition. They went on slowly to suppurate, and health was gradually restored. Dr. Forbes' description of this mild form is similar to that given by Foderé as applying to the benign plague observed in the Levant and in Marseilles in 1720, and concerning which Foderé declares that it is no

less plague than the other forms, and equally demands the attention of the physician and of the magistrate. Similar cases were described by Dr. Duthieul as occurring in Mesopotamia in 1856, and these were observed in Benghazi, in North Africa, in Astrakhan, and in the early period of the Vetlianka outbreak in Russia. It is remarkable also, as pointed out by Cantlie, that for some years preceding the outbreak in China there had been in Hong Kong, South China and Singapore, a prevalence of a previously unknown affection distinguished by fever and glandular enlargements. One of these cases imported from Singapore was seen by me in 1897 in Calcutta, at the request of a medical man there, and it corresponded with the chronic affection from which the soldiers of the Shropshire regiment was suffering.

The fatality of plague naturally attracts the most attention, and unless the malady is fatal it has hitherto been considered to be any disease but plague. This view, however, requires to be revised, for when opportunities arise for special observation mild cases and mild epidemics are found to prevail alone, or co-exist with those of a severe type. The outbreak among the Souttars of Kosumba Village, inquired into and reported on by Surgeon-Captain T. E. Dyson, Deputy Sanitary Commissioner of the Gujerat district, illustrates one of these points. Here, according to Dr. Dyson, "the disease was of a mild type, characterised by slight fever of two or three days' duration, and the formation of buboes chiefly in the groin. Fully three-fourths of the thirty-one cases which occurred were of this type, and during one visit to the village I found two boys, about 12 years of age, with buboes in the groin, whose fever had been so slight as to escape observation, and they had not been recognised as plague." Surgeon-Lieut.-Col. Weir, Surgeon-Lieut-Col. Dimmock and others, much engaged in plague work, have observed and recorded such cases.

It appears to me, then, that it is in the milder types rather than in the severe forms, in the pneumonic forms and in the disease among animals, that the obscure beginnings of some epidemics of plague, in a previously healthy locality, should be searched for, and that the sanitary officer of the future, if he is to deal with plague in its early stages, must be well versed in the different types of the disease in man and in its manifestation in animals.

In other respects our knowledge of plague is defective; for instance, the mode of entrance of the microbe into the human body, the medium or media by which it gains that entrance, and the life history of the microbe outside the human body are still unknown, and consequently the plague is being fought under the most disadvantageous conditions. There are theories and explanations, but there is nothing on a sound basis. With the exception that the microbe of plague has been discovered, the existing conditions in regard to plague are much the same as those which prevailed fifty years ago in regard to cholera, before Snow's discovery that contaminated water carried the poison. Previous to then the measures introduced to combat cholera were of a general hygienic nature, the special being submerged in the general and not infrequently overlooked, while much energy and money were expended on the rectification of hygienic defects which had little or nothing to do with the spread of the disease. This must always be the case until our knowledge concerning an epidemic disease is complete.

Most of our knowledge concerning plague is in a fluid condition, principally because it is a new disease to the present generation of medical men, and because the opportunities afforded for its study in India have not been taken sufficient advantage of. In fact, they are great opportunities lost. Good work has undoubtedly been achieved by the different Foreign Commissions sent out by the several European Governments, and by the scientific Committee appointed by the Government of Bombay in corroborating and adding to the researches of Kitasato on the bacillus, but the essentially English method introduced by Simon and his contemporaries, and which is continued with such excellent results by the Local Government Board, has not been applied—I refer to the regular and systematic investigation of plague by special and thoroughly trained medical officers, case by case and outbreak by outbreak, including all the circumstances connected with each, and combining with this research laboratory work. This ought to be done, no matter what is the cost, for at the most it can only be insignificant to the losses which India is being now subjected to. It was reckoned that Bombay at the time of the height of the plague lost £100,000 a day. Over 100,000 persons have died of the plague in Western India. The other day I heard Lord Reay declare that plague is the most important problem that the Government of India has to deal with. There can be no doubt that this is so, politically as well as socially, and I would plead for the inhabitants of India, and for the sake of those who have commercial and social interests with her, and for the sake of humanity, that every effort which science and money can afford should be made to learn more about this disease.

This is impossible under the present arrangements, owing to India possessing no trained sanitary service. This is a subject I dwelt on very fully in 1894 at the Indian Medical Congress, and there was a resolution of the Government of India that such a service was to come into existence in the year 1900. The plague has probably upset the arrangements intended to have been made before such a service can be established, the first arrangement being of the education in sanitary science in medical schools of the members of the proposed service. Plague has emphasised the absolute necessity of this service and the utter helplessness of India to combat disease without such a trained service. I divide the medical part of a sanitary service into three branches—(1) the administrative, (2) the investigative, and (3) the scientific—which should be in close connection with one another, and in large towns should form parts of one large department. Broadly in England, the administrative is represented by the local health officers and their subordinates, the investigative by the medical inspectors of the Local Government Board, and the scientific by the laboratory researches made into the causes of disease in connection with special investigations of either the medical inspector or local health officer.

In the absence of a proper sanitary service in India,

laymen had to direct the operations against plague in Western India, and in a number of instances soldiers had to be employed for house-to-house inspection.

Apart from the fact that it is impossible to turn men suddenly into well-trained sanitary inspectors, there was always the risk, among a highly imaginative people, unaccustomed to see the military in their houses, of considerable alarm being created, which would be intensified by the wildest rumours; while it was certain to give a handle to the many political agitators, who are only too ready to seize every opportunity for brewing discontent and mischief. However suitable the system might be for a military station, it could not be adopted for Indian towns generally. The policy which has kept the military apart from the people except in cases of riot and other exceptional instances appears to be one on a very sound basis. Plague no doubt is an exceptional event, but it is too much connected with the domestic habits of the people to be dealt with except by agents to whom they are accustomed. To the members of the British Medical Association, as well as to those accustomed to the methods adopted in England with regard to epidemics, the system of placing the control and direction of an infectious disease into the hands of laymen must seem extraordinary. The following extract, taken from one of the despatches published in one of the blue books in 1897, will serve to illustrate the system. It is as follows :—"In view of the rapidity with which plague was spreading in the Satara district, a committee under the Presidency of the Honourable Mr. Spence, and comprising the following officers, Mr. Lely, I.C.S., Mr. R. A. Lamb, I.C.S., Mr. A. C. Logan, I.C.S., and Lieut.-Col. J. W. Wray, all of whom have had wide experience of plague measures, was appointed to devise measures for the prevention of the spread of the disease." I am sure the exceptionally able and distinguished administrators on this Committee, and who belong to a Government which is splendidly organised in other respects, would be the first to acknowledge that an organisation composed of laymen is not fit to deal with epidemic diseases. The system can only be excused under the exceptional circumstances of there being in the country no properly trained sanitary service whose duty would be to control epidemics. A native and European sanitary service is needed to protect the civil population against the invasion and ravages of disease, just as a European and native army is required to protect them from the invasion of foreign armies.

Later on in the epidemic, by the despatch of Army medical officers and Indian medical officers from other parts of India, and by the sending out of a large contingent of medical men from England by the India Office, a nucleus of an administrative department was formed. But with reference to the Army medical officers and the Indian medical officers, it was only robbing Peter to pay Paul, for many districts requiring European medical officers were denuded of them. As a matter of fact, without plague duties the medical officers of the Indian Medical Service have for many years been overworked and are not sufficient in number for the ordinary duties which have been assigned them and have a well founded grievance in their inability to obtain the leave and furlough due to them.

If we now turn to the investigative branch, consisting of a body of highly trained men whose sole occupation is to search out the causes of an epidemic and inquire personally into the manner in which it spreads among the inhabitants, it is found that no such branch exists in India. From the foregoing it will be seen that two of the most important branches of a sanitary service in India have still to be formed.

As regards the scientific branch, the Government of India and the Government of the North-West Provinces have been fortunate in possessing for some years past the services of Surgeon-Lieut.-Colonel D. D. Cunningham and Mr. Hankin. Two laboratories, however, in a vast country like India, do not meet its requirements. Moreover for the past eighteen months Professor Cunningham's laboratory has been closed, owing to his retirement and there being evidently no one to take his place. When the plague broke out in Bombay the Government of India very wisely requisitioned the services of M. Haffkine, who was on a visit to the country pushing forward his anti-cholera inoculations. And in a short time M. Haffkine announced his brilliant discovery of a new method of combating plague. This discovery was not, I would point out, made suddenly after the Government of India requested him to go to Bombay and provided him with a laboratory, but it was the result of many years' work in the laboratory, beginning at least ten years previously from the time M. Haffkine directed his attention to anti-cholera inoculations. I mention this because there is a disposition in some quarters to consider laboratories expensive luxuries, unless some discovery of the first magnitude is speedily made.

The Government of India has always been imbued with the great importance of establishing laboratories in India, but the state of finances has invariably blocked the way. This difficulty, however, disappeared in a most charming and unexpected manner in 1897, when a number of the Princes in India expressed their desire to commemorate the sixtieth year of Her Gracious Majesty's reign by establishing a Health Institute for India, which should have M. Haffkine as its first director.

The spontaneity of the offer and the generosity with which it was made was enhanced by the peculiar appropriateness of this royal memorial to our beloved Empress Queen, whose reign has been so distinguished for the advances made in scientific and preventive medicine. The Princes were anxious that the site should be chosen and the foundation of the Health Institute laid on the day of the Jubilee. Owing to delays over which possibly the Government of India had not full control, in consequence of its plague and famine work, the auspicious day was allowed to pass and no foundation stone was laid. Everyone acquainted with India will know the importance of an auspicious day. It is the symbol of success and good fortune.

Exciting events follow quickly on each other in India and crowd out the preceding. War, in this case, with its distractions and expenses, burst out in the North-West Frontier, and the golden moment was lost for the establishment of a magnificent Health Institute, which, while serving as a noble monument of the loyalty and liberality of the Princes, would at

the same time be for India a landmark in the Victorian era. Let us hope, however, that the check, however regrettable, is only of a temporary nature, though it requires a sanguine mind to overlook the fact that delays mean new interests arising, a certain amount of chagrin, and the imperilling of a noble project.

A similar fate, owing to similar causes, appears to threaten the Pasteur Institute of India, for which R.77,000 was collected over two years ago, and a site offered by the Punjaub Government. A letter from the Committee of Management of the proposed Pasteur Institute was addressed, over eighteen months ago, to the Government of India, asking for their sanction to this site. At a recent meeting of the Committee the members had to adjourn because no reply had been received to their letter.

I have given these instances, not because I think the Government of India unfavourable to these proposals, but as illustrating the fact which is too familiar with those interested in sanitary progress in India, that under present arrangements even the most important sanitary matters cannot have the attention paid to them which is necessary, and that the only remedy is a properly constituted sanitary service, similar to that which I drafted in my address in 1894, and which was approved of then by the Government of India.

As regards plague in India, it appears to me that the disease has come to stay, at least for a considerable time, and it is consequently important on this ground alone, quite apart from other reasons, that a trained sanitary service should be established, and while doing all that is known to check its ravages, it is also necessary to systematically investigate and study the disease from every point of view, which cannot be done without laboratories and a specially trained service.

MALARIAL AFFECTIONS OF THE EYE.

By M. T. Yarr, F.R.C.S.I.

Major R.A.M.C.; Chief Clin. Asst., Royal Ophthalmic Hospital, Moorfields; Member Ophthalmological Society, &c.

Text-books on diseases of the eye as a rule dismiss the subject of malarial eye-affections in a few words: in fact, I am only aware of one[1] in which anything like an adequate summary of the state of present knowledge of this important class of disease is given. When we consider the large amount of space often devoted in these works to diseases and congenital defects of excessive rarity—to the curiosities of ophthalmology, so to speak—the absence of detailed reference to malaria becomes astounding. On the other hand, a few able monographs on the subject are in existence, and a mass of evidence in the shape of articles on symptoms, notes of cases, &c., may be laboriously disinterred from the ophthalmological literature of the last thirty years.

Most medical men in practice in the tropics are familiar with a distinctively malarial class of eye-diseases, with characteristic symptoms, characteristic ophthalmoscopic signs, and above all, characteristic pathology; and few writers on tropical diseases omit more or less detailed allusions to the subject—indeed, so far back as 1833 we find a special "amaurotic" form of malarial fever described[1]—but only too often the value of their remarks is minimised by vagueness of description and unscientific phraseology, those convenient but antiquated terms, "amaurosis" and "amblyopia," being employed to cover a multitude of sins of omission and inaccuracy. Some idea of the importance of these affections, as well as of their wide-spread prevalence, may be gathered when we find it stated by Poncet[2] to whose able and laborious researches we owe most of our knowledge of the subject—that he found fundus lesions visible by the ophthalmoscope in no less than 10 per cent. of malarials examined by him in Algiers, and by the microscope found fundus lesions *the rule* in necropsies on cases of malarial cachexia and pernicious malaria. It is therefore evident that malaria—which, like syphilis, spares no organ—does not spare the eye.

In the brief space at my disposal I can only endeavour to bring together, in the form of a concise summary, facts, hitherto to be found only in scattered form, supplemented by such observations as my personal experience in the tropics, at Netley, and at Moorfields enable me to make.

I purposely omit reference to malarial albuminuric retinitis and diseases of the anterior half of the eye, as not presenting, in my opinion, signs sufficiently distinctive to justify, in the present state of our knowledge, an attempt to group them under the head of affections characteristically malarial.

Malarial eye-lesions all originate in circulatory troubles and may be conveniently classified under the following heads:—

(1) Neuritis.
(2) Retinal Hæmorrhages.
(3) Retino-Choroiditis.
(4) Effusions into the Vitreous.

(1) *Malarial Neuritis.*

From one of my Hong Kong case-books I take the following brief notes of a typical case of malarial neuritis:—

Private A. F., admitted to hospital complaining of supra-orbital pains, dimness of vision, and photophobia.

During preceding twelve months had been in hospital seven times with malarial fever, no attack of exceptional severity; he is thin, anæmic, spleen slightly enlarged; temperature normal, urine normal. Vision $\frac{6}{60}$ each eye; fields normal; colour perception unimpaired; disc raised, reddish-grey, margins blurred; rest of visible fundus normal save for a slight haze. He was placed on light, nutritious diet—chicken, fish, beef-tea, and given quinine. A week later the visual acuity had improved to $\frac{6}{18}$, but reverted after three days to $\frac{6}{36}$, and varied between that and $\frac{6}{24}$ for some time; five weeks after admission it suddenly became $\frac{6}{12}$, at which it remained. The supra-orbital pain and photophobia ceased a fortnight after admission. After seven weeks in hospital he was

[1] "Traité Complet d'Ophtalmologie," par L. de Weeker and G. Landolt (article by J. P. Nuel).

[1] Stosch, "Febris Intermittens larvata amaurotica," Carper's *Wochenschrift*, 1833, 11. No. 8.

[2] *Ann. d'Oc.*, 1878, t. lxxviii., p. 201.

sent to the sanatorium on the "Peak," and from thence invalided to England a month later with "malarial cachexia." Examined again before leaving, visual acuity $\frac{6}{12}$, fields normal, with the exception of a very slight contraction on the nasal side; colour perception normal; fundus normal, with the exception of a slight greyness on the temporal half of optic disc.

Signs and symptoms binocular throughout. No history of syphilis.

I have selected the above case as embodying most of the characteristics of the disease.

(1) It will be seen that the patient had suffered from repeated attacks of malarial fever. This is the case as a rule; it almost invariably occurs in those whose systems are broken down by many—it may be slight—attacks of fever. I have seen one case of neuritis commencing in the course of a pernicious attack, but such cases are not common.

(2) In the commencement, supra-orbital pain and photophobia are almost constantly present; night-blindness frequently.

(3) Colour perception remains unimpaired, except in the rare cases ending in complete atrophy.

(4) The variations in the visual acuity in the course of the malady form the most characteristic symptom and distinguish malarial neuritis from all other forms. Sulzer, in a careful *résumé* of the subject,[1] lays stress on this point. A diminution of the visual acuity to $\frac{1}{10}$ can rise in two or three weeks to $\frac{1}{2}$ or $\frac{2}{3}$, falling again perhaps in two or three days. Macnamara[2] described a case in the Calcutta General Hospital of a young girl of 13, admitted with a visual acuity = perception of light only—who was discharged five weeks later with normal vision.

(5) Fields intact, or only slightly contracted.

(6) The fundus changes visible with the ophthalmoscope include swelling of the papilla, which assumes a greyish-red colour, œdema of the circum-papillary retina with effacement of the papillary margins, and enlarged and tortuous veins. The peculiar colouration of the papilla—"teinte rouge-grisatre"—due to parasites in its capillaries, is pathognomonic. In about a third of the cases tiny peripheral retinal hæmorrhages are also found.

(7) About 80 per cent. of cases terminate in a partial atrophy, indicated by varying diminution of visual acuity, irregular contraction of the field, and slight greyness of the disc; many end in apparently complete recovery, some rare cases go on to complete atrophy.

Pathology and Morbid Anatomy.—For our knowledge of this branch of the subject we are mainly, if not entirely, indebted to Poncet[3] who systematically examined with the microscope the eyes in all cases of death from malaria at the military hospital of Philippeville (Algiers). His researches show conclusively that the changes in the disc and retina in this disease are due primarily to melanæmia with increased vascularisation, the subsequent atrophy or partial atrophy being explained by consecutive endoarteritis of the vessels. A perpendicular section through the retina shows the raised, swollen papilla ("tete de hanneton"), its little capillaries stuffed with leucocytes each containing a central spot of black pigment—red corpuscles excessively rare; the same pigmented leucocytes (when Poncet uses the word "leucocytes" he undoubtedly means in most instances malarial parasites) fill the retinal vessels, and when hæmorrhages exist, they are found to be due to emboli of these leucocytes with consecutive extravasations.

The affection is always binocular, although it does not usually begin in both eyes at the same time.

Sulzer believes that a certain proportion of these cases have malaria as a predisposing cause only, the exciting cause being the indirect action of sunlight, and adduces certain cases seen by him in Borneo in support of his view, but his arguments are not convincing and the evidence seems inadequate.

(2) *Retinal Hæmorrhages.*

Two varieties of retinal apoplexy are found in association with malaria: (*a*) minute peripheral; (*b*) large peri-papillary and macular.

Minute hæmorrhages in the ciliary zone of the retina are frequent in acute attacks of fever: they are often so very minute and so far forward as to be easily overlooked. Poncet found them in all cases of death from malaria. They may accompany or follow neuritis,[1] but often form the only apparent lesion of the fundus. It seems probable that many of the transient disturbances of vision so commonly seen in malarial fevers are due to slight œdema of the retina, followed by these tiny hæmorrhages.

The large peri-papillary and macular hæmorrhages are much less frequent, and like the neuritis which they sometimes accompany, are usually seen only in malarial cachectics. These are of much graver import, always causing some impairment of vision and occasionally even absolute loss. I believe many of the cases of "sudden and persistent amaurosis" described by writers on malaria to be due to macular hæmorrhages. The only case of such sudden amaurosis I have seen was in Hong Kong, in a soldier suffering from advanced malarial cachexia who subsequently died; in the course of one night his vision in the right eye was reduced to perception of light, in the left to $\frac{6}{36}$; examination showed a large macular hæmorrhage in the right, with several small hæmorrhages between papilla and macula in the left; diffuse haziness of retina in both.

In a case of malarial cachexia invalided from the Indian Frontier, which I recently saw at Netley by the courtesy of the Director-General, Army Medical Staff, there were several hæmorrhages arranged in a curiously symmetrical manner along the inferior temporal vessels in both eyes; V.A.: in R. $\frac{6}{18}$, in L. $\frac{6}{24}$; urine normal.

Microscopic examination shows these retinal apoplexies in malaria to be due to infarcts of parasites followed by extravasations (*vide supra*).

(3) *Retino-Choroiditis.*

In about 20 per cent. of acute intermittents, generally towards the end of the hot stage, patients complain of supra-orbital pains, tenderness on pressing

[1] "Troubles de la Vision dans l'Impaludisure," *Arch. d'Ophtal.*, 1890.

[2] *Medical Times and Gazette*, May 2, 1868.

[3] *Vide supra.* Also "Atlas des Maladies Profonder de l'Oeil," par Perrin and Poncet.

[1] Gueneau de Mussy, *Journal d'Ophtal.*, t. 1., p. 5, 1872.

the eyeballs, photopsies and photophobia. Examination then discloses a general hyperæmia of the fundus, mainly venous; red, slightly swollen papilla, surrounded by a grey veil; and general haziness of the retina which appears to have an undulating surface —"dunes" with intervening depressions.

This œdematous state of the ocular membranes—the first stage of malarial retino-choroiditis—generally subsides without leaving any appreciable trace. In a certain number of cases, however, more especially in those who have had repeated attacks of fever, and are falling into the condition of malarial cachexia, the symptoms persist, punctate peripheral hæmorrhages appear, and a chronic, slowly progressive retino-choroiditis is set up, ending in capillary atrophy of the choroid and much loss of vision. Out of thirty-eight cases of malarial cachexia which I examined at Netley, in April last, I found this condition of capillary atrophy of the choroid in three—nearly 8 per cent. I have been able to watch the progress of such a case in a man—a discharged soldier who had suffered much from malaria in Burmah—who has been attending Moorfields for the last twelve months; the fundus is now of an almost uniformly grey colour, as though powdered over with pepper, the disc is pale and the arteries reduced to fine threads; the distribution of the choroidal vessels is mapped out with extraordinary clearness, the vessels seem almost white with a central red streak; pigment-layer of retina and chorio-capillaries atrophied; vision $\frac{4}{60}$ in one eye, $\frac{6}{60}$ in the other; some myopic astigmatism, but the correcting glasses only improve to $\frac{6}{36}$ and $\frac{6}{18}$; fields irregularly contracted. When first seen he had only general haziness and loss of lustre of retina, with V. $\frac{6}{12}$ in each eye with correction.

The following description by Poncet[1] of the retina of an Algerian colonist who died from a pernicious attack of malarial fever shows clearly the nature of the circulatory changes in the acute stage of this affection. The patient was much emaciated, pale, and anæmic, and had had several attacks of fever.

"In all the capillaries such a quantity of pigmented elements is found that each vessel looks as though formed of a mosaic with little black points. Red corpuscles are rare. Each little black point is a pigment molecule in the protoplasm of a white corpuscle; very rarely the pigment is free in the capillary. Two points are very clearly demonstrated. First, the extraordinary number of leucocytes; second, the enormous quantity of pigment. It is easy to understand the obstacle to the circulation caused by this mass of leucocytes in the capillaries. This abundance of pigmented leucocytes (parasites?) is found in the entire retina, at the periphery as at the papilla, and explains the peri-papillary œdema and dirty grey aspect of the papilla in these cases of malarial cachexia."

The subsequent capillary atrophy of the choroid and partial atrophy of the optic nerve is due to chronic inflammation of the choroidal and retinal vessels, set up by the irritation of the plasmodia, ending in atrophic changes.

[1] Planche, lix., fig. 1. Atlas de Perrin et Poncet.

(4) *Effusions into Vitreous.*

White Infiltration of Vitreous.—This rare and curious affection was first described by Seely,[1] and consists in an infiltration of the vitreous, forming in stages, causing almost complete loss of vision for a time, and giving a characteristic white reflex with reflected light. Seely attributes it to a serous infiltration, due to chronic paludism. In his two cases the progress of the disease was oscillating for several months; eventually the visual acuity became normal under prolonged quinine; in one case mobile opacities persisted.

Sulzer describes three cases of this disease. In the first two the ocular affection had existed some weeks before they came under his notice; in the first the eyes had been blinded during the night, with only a day's interval between each; in the second there was an interval of several weeks. The first was complicated by intense ciliary and supra-orbital neuralgia on both sides, with tenderness of the eyeballs and limited and painful motility. Both cases were malarial cachectics, with intense anæmia. Visual acuity was reduced to perception of light. The diffused vitreous infiltration, which at first gave a perfectly white reflex in all directions, was not completely absorbed in either case. During the eighteen months under observation this was replaced by moving flocculi arranged like a spider's web, with slight turbidity of the vitreous in the meshes, so that the fundus was only indistinctly visible, although the white atrophic colour of the papillæ could be made out. The right eye of the first case became blind, the left counted fingers at a short distance. The second counted fingers at 60 cm. when last seen. The third case was seen a few days after the onset of the disease, when only the right eye was attacked; it presented a diffuse and equal infiltration of the posterior segment of the vitreous with a faint peripheral red reflex on complete dilatation of the pupil; V.A. = fingers at 20 cm. Four weeks later the left eye was similarly attacked. In three months, under quinine, total reabsorption took place, leaving V.A. normal.

Penoff[2] also described diffuse opacities of the vitreous in malarials, but they were complicated by affections of the uveal tract.

I have never seen a case of white infiltration, and such cases of vitreous opacities in malarials as I have seen have been preceded or accompanied by uveal or other lesions only indirectly due to malaria.

It will, I hope, be clearly understood from the foregoing necessarily imperfect sketch, that the classification I have attempted to make is based only on the prominence of one set of signs or another in the majority of cases; for instance, a sharp line of demarcation cannot always be drawn between neuritis, retinal hæmorrhages, and retino-choroiditis; many cases occur in which all three affections are associated or form stages in one process.

It remains only to enumerate some of the rare or obscure affections mentioned by writers on malaria.

[1] W. W. Seely, *Transactions of American Oph. Soc.*, 18th Annual Meeting, 1882, p. 345.

[2] *Centralblatt fur pract. Augen.*, 1879, p. 80.

Sudden and persistent Amaurosis without visible Fundus change.—Well authenticated instances of this are on record: they can only be attributed to some obscure focal brain-lesion.

Periodic Amaurosis.—See remarks on œdema of retina and retinal hæmorrhages. Possibly also due to quinine.

Sudden Amaurosis ending in Atrophy.—Possibly due to hæmorrhage into the sheath of the optic nerve: occasionally cases of quinine amaurosis.

Persistent Central Scotoma.[1]—I have never seen a case that was not due to macular hæmorrhage.

Periodical Blue Vision.—Baas[2] describes a case of malarial fever in which this curious symptom was present. I am unable even to conjecture an explanation.

The treatment of malarial eye troubles is the treatment of malaria; most essential of all is the early removal of such cases to a non-malarious country. A prolonged course of iodide of potassium is generally of benefit in hastening the absorption of vitreous opacities.

Heurteloup's artificial leech to the temples, protection from light, blisters, are useful in relieving local symptoms.

Quinine Amaurosis.

Before concluding, it is necessary to say a few words on *quinine amaurosis*.

Slight degrees of this, caused by spasmodic contraction of the arteries, are amongst the familiar experiences of all practitioners in malarious countries. A certain amount of amblyopia is almost invariably present in cases of quinism, synchronous with the aural symptoms. In very susceptible individuals this may amount to absolute blindness, persisting for hours and even days. In severe cases varying amounts of concentric contraction of the field remain, central vision, colour, and light senses being as a rule unaffected. In slight degrees of quinine amaurosis the ophthalmoscopic appearances are normal; in severe cases, with persistently contracted fields, pallor of the disc and thready vessels are found.

I am indebted to the kindness of Mr. Treacher Collins for the details of the following remarkable case. The patient has been attending Moorfields at irregular intervals for the last four years. I first saw him three years ago.

W. J., aged 29, came to the out-patient department on June 8, 1894. He stated that three months before he had taken six-pennyworth of quinine (120 grs.) for a headache; vomiting, lasting eighteen hours, followed, with acute head-pains but *no* deafness; his "sight seemed to go all at once," and he could not distinguish light from darkness with either eye; four days after sight began gradually to return, and in three weeks' time he could read; he returned to his employment as a carman and felt little trouble till a week ago, when sight began to fail again. On examination, V. $\frac{6}{9}$ each eye (telescopic); fields concentrically contracted to the size of threepenny pieces; discs a dead white; vessels threads, fundi anæmic, white lines along arteries. When next seen, on June 28, 1894, V. was reduced to hand movement. After this it began very slowly to improve under nitro-glycerine tabloids; slight momentary improvement could always be obtained by inhalations of nitrite of amyl. When last seen, eight months ago, V. $\frac{6}{18}$ and J.$_2$ in R.; $\frac{6}{9}$ and J.$_1$ in L. (telescopic); field and fundus unchanged.

Such cases are happily of extreme rarity, in this country, at all events. Mr. Swanzy, in the last edition of his work on "Diseases of the Eye," states that he has seen only one case of quinine amaurosis with permanent serious defect of vision.

De Schweinitz made some interesting experiments on dogs in order to determine the lesion in quinine blindness,[1] showing conclusively, first, that the prolongation of quinine-amaurosis produces true atrophy; and second, that thrombosis of the central vessels may be expected in severe cases.

Treatment.—Stoppage of quinine; this, with tonics and nitro-glycerine, generally leads to ultimate cure. I need hardly add that in treating ocular affections in malarials, the possibility of the symptoms being produced by quinine should never be overlooked.

[1] Teillais (de Nantes), *Ann. d'Oct.* xcv., p. 234.
[2] *Klinische Monatsblatter fiir Augenheilkunde*, 1885, p. 240.

[1] *Transactions of the American Ophthalmological Society*, 1891.

THE TESTING OF HAFFKINE'S PLAGUE-PROPHYLACTIC IN PLAGUE-STRICKEN COMMUNITIES IN INDIA.

By W. M. Haffkine, D.S.C., C.I.E.
AND
Surg.-Maj. Bannerman, M.D., I.M.S.

The details of the preparation of this plague-prophylactic were given in the *Brit. Med. Jour.* and *Ind. Med. Gaz.* in 1897.

In 1896 experiments such as the following were performed. Of twenty rats taken from a ship newly arrived from Europe or some place where there is no plague; ten are inoculated with the prophylactic and 10 are not. Then the whole 20 are put together and a plague-stricken rat is introduced amongst them. In course of time 8, 9, or all the unprotected rats die, while none or possibly but one of the protected dies.

In January, 1897, a large number of leading European and native gentlemen offered themselves for inoculation to prove the harmlessness of the method. Others were similarly inoculated before the inmates of Biculla House of Correction, Bombay, where plague had broken out, and as a result half the inmates volunteered and were inoculated on January 30, 1897. On the day of inoculation 6 cases occurred of which 3 proved fatal. Amongst the inoculated one had a bubo and 2 others developed buboes on the same evening. All three died.

From this date the inoculated and uninoculated lived under exactly similar circumstances until the epidemic stopped eight days later. Of 173 unprotected 12 were attacked with plague and 6 died. Of 148 protected 2 were attacked and none died. This was encouraging.

During the same year 8,200 persons were inoculated in Bombay, of whom 18 (all natives) were attacked and only 2 died, and *these two developed* symptoms

twenty-four hours after the inoculation, probably having been infected before inoculation.

At Mora in the Kolaba district, near Bombay, there was a population of 1,000 souls, of whom 429 were protected by inoculation. Of these protected persons only 7 were attacked and none died. Of the uninoculated 26 were attacked and 24 of these died.

In Lower Damaon, a Portuguese colony, 2,197 were inoculated, 6,033 remaining uninoculated. A careful investigation carried out by Professors Koch and Gaffky of the German Scientific Mission, Surgeon-Major Lyons and Mr. Haffkine, showed that from March to May, 1897, of uninoculated 1,482 died. On similar grounds one would have expected the inoculated to have lost 332, if the prophylactic was without effect; but only 36 actually died. This is equivalent to a reduction of mortality of 89·2 per cent. in favour of preventive inoculation.

In Lanowlin, a small hill station near Poona, with a population under 2,000, a recrudescence of plague occurred. The officers of the Research Laboratory took charge of two wards of the town most severely attacked, and made a house to house visitation, making a careful census of inhabitants and inoculating day by day.

Of 323 protected 14 were attacked and 7 died. Of 277 unprotected 78 were attacked and 58 died, a reduction in mortality of 85·7 per cent.

At Kirkee, near Poona, the native artillery (1,530 men, women, and children) were attacked under the fullest military sanitary supervision. One out of every 6 were attacked, and of these 2 out of every 3 attacked died. But for this military sanitation doubtless these figures would have been higher.

Out of these 1,530 individuals 671 were protected by inoculation and 859 not, though all were under similar circumstances. Of 859 uninoculated 143 were attacked and 98 died. Of 671 inoculated 32 were attacked and 17 died, a reduction in mortality of 77·9 per cent.

In Umarkadi Jail were 400 inmates, and before inoculation 3 had died of plague. They were paraded, and every other man as he happened to be on parade was inoculated. All then lived under identically similar circumstances. The uninoculated should have had 7 deaths to make them equal to the others, but they had no deaths.

At Undera (near Baroda), out of a population 1,029, 79 had died of plague; 513 were inoculated and 437 not, an equal number of age and sex being taken and all living under similar circumstances.

Of 513 inoculated 8 had plague and 3 died. Of 437 *not* inoculated 28 had plague and 26 died, a saving of 26 lives and a reduction in mortality of 89·65 per cent.

This difference was even more strongly marked when individual families were dealt with. These figures were verified by Surgeon-Major-General Harvey, Surgeon-Major Bannerman, Surgeon-Captain Dyson and Mr. Haffkine.

In the Khoja Mussulman community of Bombay, 5,184 were inoculated, and 8,146 remained uninoculated. In the inoculated 3 deaths from plague occurred and 4 from other causes. In the uninoculated 177 deaths occurred from all causes.

Finally, the result of inoculation with Haffkine's prophylactic against plague confers an average reduction in mortality of 86 per cent. on the mortality of unprotected persons.

APPEARANCE OF PIGMENTATION IN LYMPHOCYTES IN RELATION TO THE DIAGNOSIS OF MALARIA.

By Dr. Patrick Manson, LL.D.

In the *Annales de l'Institut Pasteur* of December 25, 1897, Vincent, in the course of an interesting article, "Du processus leucocytaire dans la malaria," states that the phagocytic function, as regards the malaria parasite, is performed almost exclusively by mononucleated leucocytes (micro- and macrophages). He further states that occasionally, though rarely, the small lymphocytes have the property of including the parasite, and in an illustration he figures a lymphocyte showing a speck of black pigment lying in the narrow peripheral cytoplasmic zone.

Most observers are agreed as to the activity of the large mononucleated leucocytes in malaria. Metchnikoff, however, explicitly states that the lymphocytes are completely passive in this respect, and are never phagocytic in malaria. So far as my observations have gone they bear out Metchnikoff. I believe that Vincent, as well as others who share his opinion with regard to the phagocytic action of the lymphocytes, have been misled by a feature which must be regarded as being perfectly normal to a considerable proportion of the lymphocytes. This feature may be observed in all bloods, whether from malarials or from persons who may never have been exposed to malarial influences. The lymphocytic pigmentation, described and depicted by Vincent, is therefore a physiological and not a pathological pigmentation.

If we examine properly prepared blood slides from healthy persons, we find that in 20 to 30 per cent. of the lymphocytes a minute black dot (in some instances very minute, in others of considerable magnitude) can be seen. Occasionally there are two such black dots, very rarely three. They lie in the cytoplasm and usually close to the nucleus. By raising the lens their intense blackness becomes merged into a brightly refringent speck. In accurate focus they are as black as, and quite indistinguishable from, malarial melanin. In those works on the histology of blood which I have consulted I find no mention of this speck. Friends, with a special knowledge of the histology of the blood, to whom I have spoken on the subject of this lymphocytic speck, say they are not aware of its having been described, and cannot suggest an interpretation. It is difficult to believe that this appearance can have been overlooked by the many careful observers who in recent years have made a special study of the blood, but it is certain that there is no allusion to it in the text books, nor in works specially devoted to malaria.

I have thought it right to call attention to the matter, as it is more than likely that, in attempting diagnosis, some may be misled into giving a diagnosis of malaria on finding this appearance of pigmentation in the lymphocytes.

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THE

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SEPTEMBER, 1898.

PROFESSOR KOCH ON MALARIA.

THE lecture on malaria delivered by Professor Koch to the German Colonial Association in Berlin was the outcome of his recent visit to Eastern and Central Africa, at the instance of the German Government, to inform himself regarding the prevailing diseases of those regions and the means of guarding against them. Citing some instances of the terrible effects of malaria which he had observed in Eastern Africa, Dr. Koch expressed his opinion that the German Colonial possessions would never be a source of real satisfaction to the German people till they succeeded in getting the upper hand of this disease, and that this end would not be obtained until we knew the real nature of malaria and how it originated and spread. The progress already made in this direction was the object of the lecture. Texas fever, which in some respects is like malaria, was first described because he thought that having been more thoroughly investigated it threw some light on the dark points of malaria. It was not in Texas, but in the Northern States of America that attention was first drawn to Texas fever. Smith had found that the red corpuscles of the blood of animals suffering from Texas fever contain the parasite, the pyrosoma bigeminum. Texas fever, or rather fever with the pyrosoma bigeminum, has been recognised in South Africa, Australia, Roumania, in the low lands along the Danube, and in the Campagna near Rome. In German East Africa Dr. Koch succeeded in proving its existence, and he repeated the experiment of Smith in infecting healthy animals with Texas fever by means of ticks from sick animals. The important point, however, is that the ticks that Dr. Koch used were the descendants of the ticks which had fed on sick animals, thus proving the possibility of the transmission of blood parasites by descendants of ticks. With this preface Dr. Koch proceeded to speak of malaria, and in East Africa he believes that there are four forms of malarial fever, two, however, being only important. These are the ordinary tertian, which constitutes 10 per cent. of the malarial fevers, and tropical malaria, which constitutes 90 per cent. In the latter he succeeded in proving, in harmony with the regularity of the attacks, the development of the parasites is just as regularly related to the attacks as the Italian investigators proved it to be in home malaria, and, that, as in ordinary tertian, sporulation coincides with the beginning of the attack, and quinine must be given, if it is to do its work efficiently, some hours before, so in tropical malaria the parasites are most sensitive to quinine when the large rings appear in the blood. Acting on this principle Dr. Koch appears to regard it with unusual success, except in cases of relapses. To prevent the latter he gives one gramme every fifth day. He, however, admits that further investigation and research are needed on this point. Dr. Koch has never seen a case of malaria breaking out when one gramme of quinine was taken every fifth day. We fear that his experiences are somewhat unique in this regard, and that further experience will lead him to modify his views on this point. Dr. Koch has some very decided views about black-water fever, which he considers to be nothing more nor less than quinine poisoning, and that where quinine is used somewhat more cautiously, and arsenic and methylene blue employed, black-water fever will vanish altogether

from the category of the tropical diseases proper. Possibly Dr. Koch has not been long enough in the tropics nor has had sufficient opportunity of studying the disease to enable him to come to so important a conclusion, but it will be well for medical men who come across these cases to study them carefully in order to refute or confirm his views. In dealing with the origin and transmission of malaria Dr. Koch favours the mosquito theory, and regards the experimental testing of it as extremely important, because it would give us quite definite guidance for action. No mention is made of Surgeon-Major Ross's experiments, but a suggestion is thrown out that the circumstances may be similar to those found experimentally in Texas fever, viz., that the mosquito receives the parasites, transmits them to its eggs and young larvæ, and only the next generation is able again to infect with malaria parasites.

CONDITIONS OF SPANISH MILITARY HOSPITALS IN SPAIN, CANARY ISLANDS AND CUBA IN 1897.

By Reinhold Kuge.
Staff-Surgeon German Navy.

During a journey last autumn and winter, I had the opportunity of inspecting Spanish military hospitals in various regions of the world, and, as the war between Spain and America has arrested general attention, it may be interesting to see how far the Spaniards are provided in respect to sanitation.

In September, 1897, I was in Vigo, a small town with about 30,000 inhabitants, in the north-west corner of Spain, lying by a deep, well-protected bay, and which in summer is much resorted to as a watering-place. I had already seen the Civil Hospital, named "Elduayen," after its founder, and found it well fitted up. I then without difficulty obtained permission to inspect the military hospital. The hospital lies on the quay, close to the landing-stage of our boats, and was formerly a chapel.

The various wards were situated round a square, badly-kept and dirty court-yard. They were desolate, low-pitched large apartments, from the walls of which the cement had fallen, and on the ceiling of which the supporting beams could be seen uncovered and uncleaned. The planks of the floor were full of holes and dirty. The iron bedsteads were all more or less crippled and the blankets much torn. There were no tables for the sick.

On the other hand, the hospital dispensary was well fitted up, the reason being that it was much patronised, not by the hospital invalids, but by the officers of the garrison, who, with their families, are entitled to have all drugs, &c., from thence at a low charge.

The dispenser showed me over the hospital. There was no military doctor; they were all in Cuba or in the Philippines. A civil doctor was in charge. The chief illnesses were glandular inflammations, abdominal catarrh and sexual diseases. There was no theatre, so that every case for operation had to be sent to Coruña.

Unfortunately I could gain no idea of the treatment adopted. I did not see the w.c., I only smelt it. There was a small room, scantily furnished, for crockery and washing up.

In October, I was in Las Palmas (Canary Isles). Here the invalid soldiers were in a wing of the town hospital; there was no independent military hospital. The entire arrangements and the general conditions were so primitive that I cannot pass them by.

The hospital, a straggling, irregular building, resembled from the exterior, a prison rather than a hospital. In the building were included the town hospital, an asylum for poor incapacitated old women, a boarding-school for better-class girls, a school, and a day-nursery. Along with these, the invalid soldiers were housed.

In the infirmary division of this hospital were assembled in and out patients, children and adults together, the males and females only being separated. The wards were long, narrow and dark. The floor was badly paved with bricks, the windows far too small; there was but little attempt at cleanliness, and no convenience for personal care. There were neither male nor female attendants or nurses to keep the wards

clean; the doctors, as well as the patients, were dependent on persons only slightly ill to do this.

The gloomy wards had from thirty to thirty-five beds, not too clean, and the apartment was not properly ventilated. One ward was occupied by soldiers, and was somewhat better than the others. The illnesses were of the same nature as in Vigo, but here diarrhœa had broken out.

The kitchen was well arranged but the diet bad. Fish not even properly scaled, and meat all sinews and bone; the potatoes only were eatable.

Here also no operations were possible; when I saw the miserable theatre I did not wonder when the doctor told me if any operations were undertaken the patients died of *sepsis*.

In January, 1898, I was in Havana. I only had a superficial glance at the Military Hospital, but saw over the Field Hospital of Alfons XIII., in Fort Principe. The fort lies on a steep hill in the north-west of the town near the sea. It took me two and a half hours to inspect this giant hospital. It consisted of a number of wooden buildings connected by covered ways, with plenty of light and air, and open to the fresh sea breeze. There were 2,900 sick and wounded in the hospital, cared for by 13 military and 7 civil doctors. In the dispensary there were two dispensers and thirteen assistants; they had to prepare 7,000 prescriptions daily. Most medicines came ready from Madrid and had only to be issued; quinine was ordered by the ton. Besides the 20 doctors, there were 47 sisters, 150 hospital assistants, and 170 nurses.

The arrangement was as follows: Each block of the Hospital was fitted up on an average for 24 men; hammocks (made of sail) were used instead of beds as being cooler. Every invalid had a bed-table, as also a commode.

In each block there was a room set apart for the assistant, and opposite to this a smaller apartment for the necessary instruments, medicines, bandages, &c.

The theatre was very simple, but conformed to all modern requirements. There was a dark room for the examination of eyes; and I noticed that all surgical instruments came from Paris.

Dazzling cleanliness reigned everywhere. Besides general hospitals there were isolated sections for infectious diseases. At the time they were untenanted, as there was not one single case of yellow fever or small-pox. There was a bath-house with arrangements for immersion, sitz and douche-baths.

Cooking, in the excellently appointed kitchen, was done with steam. I tasted the food repeatedly and found it good and tasty. The cooking appliances and the apparatus for disinfection came from New York, as did those for the excellent steam laundry. All these arrangements and apparatuses were in separate independent buildings. There was also a large engine in use to raise the necessary water for this institute to two water towers, whence it was conducted, with the requisite pressure, to all parts of the hospital. All w.c.'s were fitted with plugs for rinsing.

Of the illnesses, intermittent fever and diarrhœa took the lead. Of course the proportion of these to the wounded was very small. At the surgical station there were only isolated cases of resections of large joints and amputations. All healed well; there was very seldom a case of illness from wound infection. Wound tetanus, which is more frequent in military than in civil practice, had not occurred at all, and only isolated cases of erysipelas had been observed.

In the mixed station I saw three severe cases of syphilis: itch was the most frequent of skin diseases.

Besides this, the largest military hospital, there were, at the time of my visit, 5 more military hospitals in Havana. These 6 hospitals were occupied by 9,000 sick and wounded. In Cuba altogether there were 60 military hospitals.

As I left the hospital in the evening I came across a transport of 411 sick and wounded. Some walked up the hill, or rather dragged themselves along, some were driven up in carts, and some in covered ambulances, which looked like coffins. The poor fellows all looked, more or less, like chronic invalids in the last stage of exhaustion.—*Archiv für Schiff- und Tropen-Hygiene*, August, 1898.

THE SICK AND WOUNDED IN THE AMERICAN ARMY.

In another column we publish an account of some of the military hospitals in Spain and her colonies. It would appear that in the case of Spain the further from home the better are the soldiers cared for; for the military hospitals of Havana are replete with all modern sanitary requirements. It must, however, be remembered that the sick and wounded Spaniards have been fortunate, inasmuch as the troops were operating at a base, and that they were received directly into Base or Station Hospitals.

Field hospitals were, from the nature of the campaign, but little required by the Spaniards, so that in comparing the Spanish and the American hospital equipments this fact must be borne in mind.

Very different is the case in the American army. Camps were hurriedly formed, field hospitals had to be got together under great difficulties of transport, and the very *verve* and "go" of the American soldier are adverse to the satisfactory working of a commissariat, or a medical, department in the field.

The so-called breakdown of these departments is told us, not by the pen of the foreign critic, but on the word of the candid, perhaps the too candid, American authorities themselves.

But whatever may be said of the department there is nothing but praise for the work of the individual surgeons. In a trying climate they have each hazarded life and limb to keep pace with the abnormally rapid advance of the troops. The desire of the American to "get there" is apt to lead to the neglect of, what the fighting line term, the non-combatant branches.

As so often happens in armies better equipped to take the field than was the invading army at Santiago, the medical department is passed over in times of peace with a patronising smile; and it is only when the calamities, incident during a campaign occur, that it is expected to behave with the thoroughness of the more pampered departments of the army. It was not the lack of surgeons nor the want of their presence in the most dangerous parts of the fight that constituted the so-called failure of the medical department. It was the insufficiency and bad quality of the rations, the impossibility of obtaining transport for the sick and wounded, the general unpreparedness of the military authorities for undertaking an aggressive campaign in a deadly climate, that caused what is termed a breakdown. We are informed on reliable authority, that the medical department of the American army, though overworked, did and is doing splendid service, and the malignant criticism too often in evidence is for the most part undeserved.

Under the skilled direction of Surgeon-General Sternberg, the surgeons of the American army did valuable work in an emergency, which few can appreciate unless they had been in a position to judge of its inherent difficulties.

Articles for Discussion.

THE SIESTA.

The word siesta is of Spanish origin; it is derived from the Latin word sexta (hora) the sixth (hour) after sunrise, the hour of noon. Not only has the word been adopted into the English language, but the custom of taking the "siesta" has come to be a habit in most of our tropical colonies and states. It would be well were the medical profession in the tropics to express their opinions on this custom, and to state what are its hygienic effects. The question is, does the siesta contribute to the better health of those who follow the habit? Habit it must be called, for in several of our British colonies it is no longer practised. Time was when the siesta was the invariable custom. In the early days of our knowledge of tropical colonisation life the habit was encouraged and considered to be necessary, but from whatever reason the indulgence has grown into desuetude in many places where it formerly obtained. It is chiefly in the busy business centres where the plan of taking a mid-day sleep has become a thing of the past. In

India it was once regarded as essential to the daily routine of life to rest and sleep in the middle of the day, but of late years many have dispensed with the luxury. As in India, so in the Straits Settlements and China, men no longer think, or find, the siesta a necessity. In less active centres, however, such is not the case, and, when we travel outside British spheres of tropical life, the observance is the rule. In the French Provinces of Indo-China all business is suspended during the mid-day hours, and the streets of the capital are void of all signs of life. At that hour Saigon answers to the description of a famous traveller who declared it to be "a city of empty *cafés*." Throughout the tropics generally, it may be said that where commercial activity is apparent the siesta has been abandoned, at least by the men; but where time is of but little account, it is followed as of old.

Most women, even in busy centres, however, preserve the habit, and both men and women indulge themselves in the ingratiating custom in the quieter districts. Many regularly undress and systematically lie down with definite purpose. They consider they are obeying a natural call, and adding to their powers of resisting the inroads of climate by thus conserving force and energy. The question therefore comes to be—is the old plan better, or is it advisable to resist the habit, and get through the day without sleep? Is the new fashion, of which active business men are the exponents, more salutary, or are these men by working in their offices during the heat of the day injuring their health?

The hour selected at which to lie down is after the mid-day meal; immediately after is the rule. The moment "tiffin" is over the privacy of the bedroom is sought, the blinds or shutters are arranged so as to exclude the light, and, if the street or surroundings are noisy, provision has to be made to shut out the disturbing sounds. For one and a half to two hours is this state of things maintained, and after a cup of tea or coffee the daily round is again assumed.

It will be observed that it is after a full meal that the siesta is taken. This in itself is not considered either theoretically or practically a hygienic course to pursue, yet it is done in the middle of the day, although all shun it in the evening after late dinner. Moreover, "tiffin" is usually a full—a very full—meal, a meal which causes many ailments on account of its excess. He, or she, who would study and follow the best regime in tropical life, will curtail the amount to merely the bare necessaries—a light luncheon without stimulant. This salutary rule is, however, only practised by those endowed with exceptional powers of restraint, or who have learnt the lesson by bitter experience. After this meal of several courses, with wine, beer or spirits as adjuncts, and a cup of strong coffee as a finishing beverage, it is not to be wondered at that men do not care to work, but betake themselves to rest and sleep.

Is the siesta a necessary adjunct to health? I have no hesitation in answering in the negative and declaring against the custom. The men and women who best maintain their health and vigour do not take a siesta. In consequence their night sleep is more sound and refreshing, and their digestion will longer withstand the tendency to dyspepsia, so common an ailment in warm climates. It is allowed that the European requires more sleep in the tropics than in temperate climates; but there is ample time to have sufficient sleep at night. Late hours are the exception in tropical life; ten o'clock is the hour for retiring, and sunrise, practically 6 a.m., the time for getting up. Eight hours is ample sleep for all but young children, but if one or two hours are taken in the middle of the day, sleep is apt to desert the night hours, and to make it restless and unrefreshing. Where life drags tardily along, and the weary tropical day hangs heavily on the European, the siesta helps to pass away the hours, but that does not in anyway justify the custom. Where work has to be done the siesta has been neglected, and the men who forego it are the active members of the community. This is surely sufficient testimony against the custom, and should aid us in making up our minds that the siesta is not an absolute necessity, and that insomnia, that purgatory of tropical life, is more often to be ascribed to the siesta than to any

other of the disturbing environments of a tropical night.

(Correspondence on the subject is invited.)

J. C.

PROTECTION OF BRITISH TROOPS BY MEANS OF THE MEDICAL SUPERVISION OF WOMEN.

A MEMORIAL, signed by upward of one thousand mothers in various parts of the country, has been forwarded by the Countess of Carlisle to Lord Salisbury, protesting against the principle involved in protecting the health of British troops by the system of medical supervision of women in the cantonments. This is no doubt a counterblast to the memorial, drawn up some months ago, by a number of women in Britain requesting that the Contagious Diseases Acts for soldiers in tropical countries be re-instituted. There seems to be a widespread belief that, by allowing venereal disease to prevail unchecked, men will be scared for fear of contracting the disease into abstaining from illicit intercourse and, as a corollary, compelled to marry. Psychologically, there is little relation between illicit intercourse and marriage. The instinct implanted in mankind, in common with all animals, to continue the species, is not satisfied by promiscuous sexual intercourse. There is a deeper and more enduring principle involved and one which is inseparable from animal life. Promiscuous intercourse never has and never will supplant the state of matrimony. The law of natural selection comes into the question of marriage. In that sphere sexual passion, in its vulgar sense, plays altogether but an obscure part. Natural selection obeys a higher law than mere sexual gratification; the two are inseparable, but not co-equal, in bringing about the married state. Marriage, by our modern social laws, unwritten though they are, grows more and more impossible during the "twenties." The average age at which men marry in Britain is 27, but this standard is arrived at from two very different grades of society. In the middle classes the average age of marriage for men is much higher; but amongst the working classes, more especially amongst the manufacturing classes, it is the rule for men to be married between the ages of 19 and 23. Oriental nations induce their children to marry at even earlier years—soon after puberty. They have established the custom so that their sons may be spared other and less manly forms of gratifying their sexual instinct. The argument then lies between these two great questions. By our social surroundings marriage is postponed until towards the age of 30, and between adult years and the period of matrimony are men to be condemned to the physical sins too often the accompaniment of so-called continency, or are they to be allowed to contract disease without the intervention of the State? The religious bearing, as it affects the individual, becomes a matter of personal concern; but as regards the family and thereby the national health, it has to be met and dealt with on broad sanitary principles.

OPHTHALMOLOGY IN ITS TROPICAL BEARINGS.

MALARIAL AND QUININE AMAUROSIS.—An instructive series of papers under the above title, by Dr. Fernandez of Havana, Cuba, has just been brought to a conclusion in the *Journal of Eye, Ear, and Throat Diseases* (Baltimore, U.S.A.) The author gives details of 37 cases in all: of these 25 are cases of quinine amaurosis of an unusually grave type, 7 malarial neuritis, 1 malarial hæmorrhages; the causation of the ocular trouble in the 4 remaining cases seems doubtful. The most remarkable fact brought to light is the extraordinary prevalence of quinine amaurosis—ending in too many cases in complete atrophy—in Cuba. The explanation is not far to seek: the word "heroic" is an absurdly inadequate term to apply to the doses of quinine habitually prescribed in malarial fevers by many of our Cuban *confrères*, in fact they would be incredible were they not vouched for by Dr. Fernandez. The following cases are taken almost at random:—Case VII. Child of 2; 1 gramme of quinine in the twenty-four hours for three or four days, in addition to inunctions. Case X. Child of 7; 4 grammes at a single dose. Case XI. Man of 42; quinine in 8-gramme doses. Case XVI. Woman of 32; "42½ grammes distributed about in the twenty-four hours." Case XVIII. Man of 53, treated by quinine in 8-gramme doses. As there was no improvement "12 grammes of quinine were then ordered." Those in attendance were told by the physician when this instruction was given that the patient might become blind, but that nevertheless it was necessary to run the risk in order to stop the febrile process. "The amaurosis in this case was total. . . . ." Like the profane man in the story who "could not do the subject justice," Dr. Fernandez abstains from comment.

PATHOLOGY OF PTERYGIUM.—Dr. X. da Costa, in the *Revista Portugueza de Medicina e Cirurgia* (Lisbon), describes two cases of pterygium in which histological examination showed a distinct epitheliomatous structure in the head of the tumour, and passes in review similar cases in ophthalmological literature (Snellen 1, Steiner 1, Bisti 1). He believes in the possibility of the epitheliomatous transformation of the head of a pterygium by the proliferation and normal evolution of its epithelium. He also discusses at length the connection between pinguecula and pterygium.

TREATMENT OF PTERYGIUM.—H. M. Starkey of Chicago (Annual Meeting of the American Medical Association, 1898), has been treating small pterygia, not encroaching much on the cornea, with a weak galvanic current. A platinum needle, as the positive pole, is passed through the pterygium in three lines, 2 mm. apart, and a current of

three milliampères passed for one minute at each place. In 50 per cent. of the cases the growth entirely disappeared; in 30 per cent. it was so much diminished in size as to require no further treatment; in 20 per cent. the growth, though reduced in size by the electrolysis, had subsequently to be removed by some other form of operation. Small pterygia, not encroaching much on the cornea, and particularly those with considerable enlargement of the vessels, are the most suitable. It is not advised, except as a palliative in broad fleshy pterygia, or in those encroaching much on the cornea.

TREATMENT OF TRACHOMA.—M. Eberson (*Klin.-Therap. Woch.*), lauds the use of ichthyol in trachoma and all conjunctival catarrhs, with or without corneal complications. He uses a 50 per cent. aqueous solution, to which a little glycerine is added, applied and left a minute or two.

M. Pergens, in the *Klinische Monatsblatter für Augenheilkunde*, and Prof. Deneffe in the *Bulletin de l'Académie Royale de Belgique*, strongly recommend the use of "Protargol," the new silver salt, in trachoma, in preference to the old nitrate. The main advantages they enlarge upon are: (1) that it is painless; (2) more penetrating, as it does not coagulate albumen; and (3) does not stain linen.

M. T. YARR,
Major R.A.M.C.

Review.

YELLOW FEVER IN THE WEST INDIES. By Izett Anderson, M.D. London: H. K. Lewis. 106 pp.

Dr. Izett Anderson's book is a faithful summary of the clinical symptoms and treatment of yellow fever. The author has had an experience of over thirty years in the West Indies, and his statements and advice are worthy of the greatest respect. The symptoms of the different stages of the disease are given succinctly and lucidly, and many clinical aids to diagnosis are set forth with the readiness and authority of ripe experience. The treatment of yellow fever is ably handled, the author giving not only his own methods but the methods of past and present practitioners. Dr. Anderson advocates calomel and quinine, in heroic doses, in the very early stages of yellow fever, whilst yet the urine is normal. In the second stage, where renal symptoms are in evidence, opium is stoutly condemed and carbolic acid, in an alkaline mixture, highly recommended.

Dr. Anderson is of opinion that yellow fever is non-contagious; that the doctor and the nurse are not specially exposed to the disease by attendance on the sick, in fact, that the hospital is the last place at which the disease is likely to be contracted; or as Dr. Bailey puts it, "the hospital seems to afford a certain amount of protection." Dr. Anderson ascribes environment, and not personal contact, as the primary factor in the spread of the disease; and when yellow fever is contracted at some distance from the infected area, as on board ship, he agrees with the pithy expression of Dr. Bailey—"It was a transmission of a 'section of the climate' of a yellow fever district, the ship brought something from the climate and not from the sick." In this way Dr. Anderson explains transmission of infection by blankets, clothing, &c. It may be thought a crude term by the bacteriologists, but it conveys a concrete impression which fits the received idea as to the means of spread.

It is remarkable that the two most fatal diseases known, plague and yellow fever, are not considered to be contagious, and that the attendants upon the sick in hospitals devoted to the treatment of these pests do not readily contract the disease. In fact, the staff of the hospitals show a lower rate of infection and mortality than any other section of the community.

We cannot too strongly recommend Dr. Izett Anderson's book to the young practitioner proceeding to countries within the yellow fever area, and to all those interested in tropical medicine generally.

News and Notes.

CHOLERA is officially reported to have assumed an epidemic form in Madras. A few cases have occurred among the emigrants awaiting embarkation to Natal.

IT is interesting to note that although vaccination was introduced into Japan in the latter half of the present century, it is now not only compulsory, but re-vaccination is insisted upon every five years.

WE would warn medical men, who look upon the Transvaal as a place affording a "grand" opening in which to commence the practice of their profession, to inquire of those acquainted with the present state of the country before doing so. Since the Jameson raid the country is not only poorer, but the numbers of the inhabitants are diminishing; Pretoria, Johannesburg and Bloemfontein are suffering from a "plethora" of doctors.

FOR the medical arrangements during the recent Soudan campaign, which has just culminated in the victory of Omdurman, we have nothing but praise. The Royal Army Medical Corps has initiated its existence with credit. Although the wounded in the Anglo-Egyptian army were far below the numbers the medical staff were prepared to deal with, the excellent condition in which the army was brought into action, after months of desert marching and camping, was owing to the advice of the Medical Department being followed in every particular.

AMONGST the wounded we regret to know that Lieut.-Col. A. T. Sloggett, senior medical officer of the First British Brigade, was wounded. He was hit in the left breast, and although the wound is serious it is satisfactory to hear that he was capable of being removed to the hospital at Obadich. The latest information concerning Lieut.-Col. Sloggett is, we are glad to say, that he is doing well.

MAJOR PINCHES, R.A.M.C., attached to the 21st Lancers, had a narrow escape of his life. Major Pinches joined the regiment in its famous charge. During the fight his horse was ham-strung and he fell amongst the Dervishes. Luckily Serjeant-Major Brennan saw the fallen officer, and cutting his way through the ranks of the enemy managed to remount Major Pinches behind him and bear him to a place of safety.

THE SALE OF POISONS IN HONG-KONG.—At a meeting of the Hong-Kong Sanitary Board, on July 28, the draft bye-laws for regulating the sale of poisons were submitted and adopted by the Board, subject to the approval of the Legislative Council.

VACCINATION AGAINST ENTERIC.—Voluntary vaccination against enteric fever has been started in several Indian cantonments.

A NOVEL LANCET.—A native *hakim*, or surgeon, in a little native state in the Himalayas, just beyond Simla, has cultivated a very long nail on the fore-

finger of his right hand, which he keeps very sharp and uses as a lancet as occasion requires.

SHOLAPORE PLAGUE HOSPITAL.—The lady superintendent of the above hospital thus describes how she found it. "The wards were huts, composed of matting walls, fastened together with bamboos, roofs of grass, and earth floors; slits in the matting for windows, and most of the wards had no doors. The beds were made of a framework, and short legs of rough wood, laced across with rope from side to side. . . . There were patients on the floor, patients in the beds and, for want of space, the cots were so close together that it was quite impossible to get between them. The floors were strewn with food and dirty dressings and piles of filthy clothes belonging to the patients' friends. The floors were also covered with expectoration."

THE Hakim of Port Saïd, a venerable but unwashed old humbug, receives his patients reclining on the floor smoking a hookah. His stock in trade consist of a few brass jars containing little bags of dust from Mecca, strips of parchment bearing verses from the Koran, some shells from the holy cave of "Yeddah," and a lot of similar so-called remedies. All these articles are for "outward application only." The bags of holy earth, for instance, are worn round the neck to cure a fever, the strips of parchment with a scrawl on them are to be applied to painful parts to cure rheumatism or similar evils, while holy shells are to be worn by women to prevent abortion.

LARD AS AN ANTIDOTE TO STRYCHNINE.—Dr. W. D. Turner (*Indian Lancet*) was led by the accidental observation of the recovery of a dog from strychnine poisoning after eating about four ounces of lard, to make a series of experiments on dogs and other animals. He records thirteen experiments in all—three on dogs, three on hens, one on a crow, four on hogs, and two on calves. In all cases, except the first two hogs, the animals recovered, although the lard was not administered until after convulsions had well set in. Of the four hogs two died from two grains each of strychnine with only six ounces of lard administered as an antidote, the last two, however, recovering from the same dose of strychnine after taking twelve ounces of lard.

SOUTH AFRICAN EXHIBITION, Graham's Town, Cape of Good Hope, opens December 15, 1898, for five weeks.

DEATH OF AN ARMY SURGEON FROM TYPHOID.—Among those who died on board the United States hospital ship "Relief," which arrived on August 19 from Porto Rico, was Surgeon-Major Lawrence S. Smith, of the First Regiment, Pennsylvania Volunteers. His death was due to typhoid fever, which he contracted in attendance upon the men of his command.

STORY OF TROPICAL DISEASE IN BRITISH SCHOOLS OF MEDICINE.—Early in the year, a letter was addressed from the Colonial Office to the leading Medical Schools of this country and the General Medical Council, drawing attention to the desirability of extending the teaching of tropical medicine in the British Medical Schools, and asking them to give the matter their earnest and favourable consideration. From the purport of the letter it is evident that Mr. Chamberlain is much interested in the matter, and is anxious to do everything in his power to extend the benefits of medical science to the natives of the Tropical Colonies and Protectorates, and to diminish the risk to the lives and health of those Europeans who, as Government officers or private employés, are called upon to serve in unhealthy climates. We understand that a number of the medical schools, while recognising the desirability of providing at suitable institutions means for the systematic and clinical study of tropical medicine, is of opinion that it would serve no good purpose to make it a part of the ordinary curriculum. This is an opinion with which we entirely agree. For every school, in the absence of any facilities for the practice and clinical study of tropical diseases, to encumber the student's curriculum with more subjects would be but to add the proverbial straw. At the same time we think it would be no hardship to future medical men intending to practise in warm climates, that before any civil appointment is given them or permission granted to practise, they should furnish evidences of their having studied tropical diseases. The facilities for such study is within their reach at the New School of Tropical Medicine at Greenwich. The missionaries are hoping to be able to send shortly to Khartoum a medical missionary.

A GERMAN Commission, with Professors Koch and Pfeiffer as the directors, is to proceed to German East Africa, India, and New Guinea to study malaria and tropical diseases generally. At present Professor Koch is in Italy studying the Italian methods.

A DEPUTATION, consisting of Rev. Dr. McMurtie, Mr. Ewing, Rev. J. Travers and Mr. Cantlie attended at the Foreign Office on September 14, 1898, to urge the Government to despatch experts to investigate malaria and black-water fever in British Central Africa.

Mr. Bertie, on behalf of the Government, informed the deputation that a Commission consisting of three persons, Dr. Stephens, Dr. Christopher and Dr. Daniels of British Guiana, had been already appointed and were about to start for British Central Africa.

One of the Commission is to proceed to Calcutta to study the methods followed by Surgeon-Major Ronald Ross in the investigations of the mosquito-malarial fever.

ON August 1, Brigade-Surgeon-Lieutenant Colonel Preonath Mookerjee, Civil Surgeon of Sagaing, died from a carbuncle, after a lingering illness. The late surgeon was highly respected in India.

Communications, Letters, &c., have been received from :—

A.—Dr. William Ayres (Uganda Protectorate); Dr. E. Grace Adams (Punjaub); Dr. Winslow Anderson (San Francisco.

B.—Surg.-Capt. W. J. Buchanan (Dacca); Mr. W. M. Borcherds (Cape Colony); Dr. G. P. Banerjee (Rajputana).

C.—Dr. Curwen (Peking); Dr. P. Cousland (Swatow); Lieut.-Col. A. Crombie (Inverness).

D.—Director-Gen. Med. Dept. Admiralty.

H.—Surg.-Capt. E. Wickham Hore (Bushire); Staff Surgeon P. B. Handyside (Cape of Good Hope); Mr. W. W. Hoare (North Borneo); Surg.-Lieut. S. Hunt (Poona); Mr. J. Hervan (Assam).

J.—Dr. Robert Johnson (Assam); Major T. M. Jones (Poona).

K.—Mr. Thomas H. Knott (Andover); Dr. William Kirk (Shanghai); Dr. A. Keogh (Bengal).

L.—Dr. Ligertwood (Chelsea): Dr. Landsborough (Formosa).

M.—Dr. J. C. Müller (Delhi); Dr. C. G. Monro (Blairgowrie); Dr. Moffat (Uganda); Lieut.-Col. K. McLeod, Mr. P. Michelli (Greenwich); Dr. R. Macdonald (Queensland); Dr. Masani (Harda); Dr. Mense (Cassel); Mr. Müller (London).

N.—Dr. H. A. A. Nicholls (Dominica); Dr. Nisbet (Queensland).

P.—Mr. Patenon (Grenada); Dr. Peypers (Amsterdam); Dr. Power (Argentine Republic); Dr. Plaxton (Jamaica).

R.—Surg.-Capt. Ernest Roberts (Henley); Dr. Renner (S. Leone); Dr. Palmer Ross (British Guiana); Dr. Ker Ramsey (Victoria); Dr. William N. Robertson (Queensland; Dr. Leonard Rogers (Bombay).

S.—Dr. Stokes (Pilgrim's Rest); Mr. W. P. Seed (Coolgardie); Dr. J. Scott (Bussorah); Dr. Sandwith (Cairo); Dr. Stephens (Constantinople); Mr. Stow (Hawaii); Dr. O. Sapara (Lagos); Fleet-Surg. Gerald Sichel, R.N.; Dr. Sambon (London).

T.—Dr. W. Thomson (Monte Video); Dr. E. A. Travers (Silangor); Staff-Surg. V. G. Thorpe, R.N.

W.—Dr. von Winckler (Demerara); Dr. Browne Webber (Nicaragua); Mr. Morgan J. Williams (Rhodesia).

Y.—Major G. H. Younge (Chatham); Major Yarr (London).

Papers Promised.

Abscess of Liver bursting through Lung. By Joseph Scott, M.B., C.M.

(1) The History and Pathology of Yaws; (2) The Nature of Yaws; (3) Yaws and Syphilis; (4) The Treatment of Yaws. By H. A. Alford Nicholls, C.M.G., M.D., C.M., M.R.C.S.Eng.

Kala-azar of Assam. By Dr. H. Thornhill.

(1) Methods of using Quinine; (2) The Value of Prophylactic Issue of Cinchona Preparations. By Surg.-Capt. W. J. Buchanan.

Immunity of the Natives of India from Typhoid. By Lt.-Col. A. Crombie.

The Distribution and Harmfulness of the Anchylostomum. By Leonard Rogers, M.D., M.R.C.P. F.R.C.S. (I.M.S.).

On the Sequelæ of Sunstroke. By Dr. Sambon.

The Unclassified Fevers of Hot Climates. By A. Crombie, M.D., Brig. Surg., Lt. Col. (I.M.S.).

Births, Marriages and Deaths.

The charge for inserting announcements of Births, Marriages and Deaths, is 3s. The notice should be accompanied by a remittance.

BIRTHS.

Clair-Smith.—On August 22, at Motihari, Bengal, India, the wife of William P. Clair-Smith, of a son.

Mahoney.—On August 17, at Bolarum, Hyderabad (Deccan), the wife of Assistant-Surgeon E. Mahoney, Hyderabad Contigent, of a daughter.

Woolbert.—On August 9, at Fair View, Landour, the wife of Surgeon-Major H. R. Woolbert, I.M.S., of a daughter.

MARRIAGES.

Boyd—Beveridge.—On July 30, at St. Mary's Church, Addington, Christchurch, N.Z., by the Rev. W. S. Bean, vicar, Alexander Brooke Boyd, M.A., M.B.Oxon, of Richmond, Nelson, to Margaret Miller, eldest daughter of Thomas Beveridge, of Fendalton, Christchurch, and formerly of Balado, Kinrosshire.

Buchan—Hume.—On September 10, at the Parish Church, Lynton, by the Rev. W. E. Cox, rector of the parish, assisted by the Rev. H. Ensor, Charles Forbes Buchan, eldest son of the late Charles Forbes Buchan, M.D., R.N., to Margaret Hume, second daughter of Captain Walter Hume (late 38th and 75th Regiments), of Rock Lodge, Lynton, North Devon.

DEATHS.

Miles.—On July 18, at his residence, Kensington Road, Bangalore, of Bright's disease, Surgeon-Lieutenant H. H. Miles, I.S.M.D., aged 51 years.

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1.—All communications will be acknowledged in the Journal under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the Journal of Tropical Medicine should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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THE DISTRIBUTION AND HARMFULNESS OF THE ANCHYLOSTOMUM.

By Leonard Rogers, M.D., M.R.C.P., F.R.C.S.,
Indian Medical Service.

Since the anchylostomum was shown by Griesinger to be the cause of "Egyptian Chlorosis," and more especially since the St. Gothard tunnel outbreak, the harmfulness of this intestinal worm has been well known. It is, however, only within recent years that the very wide distribution of this parasite has been recognised by many workers, among whom may be mentioned Lutz in Brazil, Sir William Kynsey in Ceylon, Rudduck in Assam, Dobson in various parts of India, and Galgey in the West Indies.

Very different opinions are, however, held as to the exact degree of injury produced by the presence of this parasite in varying numbers. Thus Sandwith of Egypt and Thornhill of Ceylon write very strongly on the terrible amount of disease and death caused by this worm. On the other hand, Macdonald of Ceylon wrote that in a great number of cases the worm does very little harm, though capable under certain conditions of destroying life; Rudduck, who discovered this worm in Assam, is of the opinion that a healthy person can support a fairly numerous colony of them without damage, and the parasite only assumes importance when the patient is lowered by some other disease; and lastly Dobson, of the Indian Medical Service, after showing that upwards of 80 per cent. of the healthy inhabitants of most parts of India harbour this worm in numbers varying from units to hundreds, very pertinently asks, "At what stage does the parasite become injurious?"

In the course of my recent investigation of the epidemic malarial fever of Assam I had occasion to make some observations and experiments on this point, and I therefore propose in this paper to examine the evidence on both sides, and to see if the truth does not lie somewhere between the two extreme views quoted above.

In the first place it is certain that if these blood-sucking worms are present in very large numbers for a long period of time, they will cause a severe anæmia and other symptoms, which if not recognised and properly treated may cause death. In the St. Gothard outbreak as many as two and even three thousand of these worms were found in some of the cases. But if such a large number of the worms can be withstood for several months, is it not evident that the loss of blood caused by small numbers, such as from 20 to 50 will be repaired by the system, and no disease will result? Dr. Dobson's observations, as well as those of McConnel in Calcutta and Bilharz in Egypt, both of whom found this worm in the great majority of *post mortems*, after death from accident or disease, prove that this worm may be present in comparatively small numbers in the majority of a population without doing any harm. I was able to confirm this in Assam, for I found this worm in 66 per cent. of 50 healthy men to whom I gave thymol. In order to form some idea of what numbers are required in order to produce anæmia, I examined the blood of many of these men with the hæmocytometer and hæmoglobinometer, notes being taken of their condition and previous history with regard to malaria, &c. I found that in the case of men who passed 20 or fewer of the worms, their hæmoglobin averaged the same as healthy men who had none. Again one man, who passed 110 anchylostoma and 104 flukes (amphistomum hominis), had an average amount of hæmoglobin, and that although he had been more than three months in jail, where he drank only filtered water, so that he must have harboured this large number of blood-sucking parasites for more than that period of time without his blood having deteriorated. Another man with an average amount of hæmoglobin, who had been in jail for nearly three months, passed one anchylostomum and 42 flukes. It is evident then that the systems of these men had been able to completely repair the loss of blood caused by such large numbers of parasites acting for several months. Once more, 293 anchylostoma were passed by a man who had been in jail two-and-a-half months, yet he showed no clinical signs of anæmia, and his hæmoglobin was found to be only 15 per cent. below the standard of healthy Assamese people.

On the other hand it was found that in the blood of apparently healthy men who had suffered from malarial fever for one week or more within the previous two years, the hæmoglobin was below the normal standard, this being especially marked in all cases where the spleen was at all enlarged. It is evident then that a very small amount of malarial fever exerts a much more deleterious effect on the blood than does a fair number of anchylostoma.

Again Dobson found over 100 of these worms in seven healthy men, one of whom passed 230. It is, then, evident that from 100 to 300 anchylostoma may be present for upwards of three months without producing any evident anæmia. When then does the parasite become dangerous to man? No very exact line can be drawn, but the opinion of several writers on this subject that 500 of the worms must be present for from six months to a year in order to produce anæmia may be accepted as sufficiently near the mark for purposes of discussion. For an anæmia so produced, the term anchylostomiasis may be fittingly applied. On the other hand the mere presence of a few, or even a considerable number of these worms, does not constitute anchylostomiasis, which term means disease produced by this worm, for the loss of blood caused by them may have been fully repaired by the system, and no disease may have resulted.

Much of the confusion in which this subject is involved is due to this fact not being borne in mind, as a perusal of the present literature of the subject will show. To give an example, Dr. Ortho Galgey of St. Lucia has recorded "Short notes of 130 cases of anchylostomiasis." An analysis of these shows that 43 of them were admitted for diseases other than anæmia or malaria, many of them not showing any signs of anæmia. Another 31 were admitted for malarial diseases, while 19 more had malarial symptoms. The number of anchylostoma passed after thymol by these cases was recorded as follows. In one case "hundreds" and in another "enormous quantities," in two "very large quantities," in six "large quantities," in 26 "quantities," in 79 "many," and in 16 "a few." A clue to what these terms mean is afforded by the fact that in several of the cases in which "a quantity" were passed, none were obtained after the administration of the first two doses of 20 grains each, and it was only after a second similar treatment that "a quantity" came away. Now it is quite certain that if as many as 50 of the worms had been present, some of them would have come away after the first administration of the drug, so that we may safely take this term to mean something less than 50, while many probably means about 20. It appears then that in this series only eight cases passed more than 50 worms, while certainly in 95, or more than three quarters of them, less than 50 worms must have been present. I feel sure, from my experience in Assam, that if Dr. Galgey would give thymol to a series of healthy people, he would find either "a few" or "many" of the worms in as large per centage of them. Now the diseases for which many of these cases were admitted to hospital ranged from measles to locomotor ataxy, yet they are all included under the head of anchylostomiasis, simply because they passed a certain number of these worms after thymol! If the term is to be used in this sense, three-quarters of the healthy people of India suffer from "anchylostomiasis," which is absurd. Moreover, I have proved that small numbers of this worm have no effect in reducing the hæmoglobin, while one of the strongest advocates of the extreme harmfulness of this parasite acknowledges that 50 of them is a number altogether too small to have any deleterious effect.

It is evident then that the presence of the anchylostoma in many of Dr. Galgey's cases was purely accidental. I would not be understood to wish in any way to disparage Dr. Galgey's work, as his discovery of the frequency of this parasite in the West Indies is most important, and has led to the saving of life, and doubtless many of his cases benefited by the thymol. The abuse of the term "anchylostomiasis" might be thought to be of more theoretical than practical importance, but the following instance of the disastrous results of looking on the presence of a few anchylostoma as evidence of anchylostomiasis, and the consequent abuse of thymol, will be suffice to show that this is not the case. In a certain dispensary in Assam, a very experienced hospital assistant counted the anchylostoma passed after thymol in a series of 72 cases of anæmia which he treated with this drug. An analysis of the notes shows that in 58 of them the spleen was enlarged, reaching three or more fingers' breadths below the ribs in 40 of them, while in 13 cases it extended to eight fingers' breadths. In 17 of them there was no dropsy, and in 17 more only slight œdema of the feet, and in the rest more extensive dropsy. The great majority of these cases were then obviously suffering from malarial cachexia with anæmia, while in some of them anchylostomiasis might fairly have been suspected. All were treated with thymol, and many of them repeatedly, with the following result. In no single case were more than 50 anchylostoma passed after the thymol, and in 57 of them less than 20 came away, yet there were 17 deaths, no less than eight of which took place within six days of the last dose of thymol. The largest number of anchylostoma passed after any of these last doses was ten, while the average was five. The removal of such small numbers could have done the patients absolutely no good whatever, while there can be little doubt that the use of such a drastic measure in cases of extreme malarial cachexia (all 17 fatal cases had enlarged spleens, and 13 diarrhœa or dysentery) must have hastened the end of some who might very possibly have recovered under milder and more rational treatment.

We see then on the one hand that if about 500 anchylostoma be present for from six months to a year, they will produce anchylostomiasis, while on the other hand in all districts infected by this worm a large number of both healthy people and those affected with all kinds of diseases, will harbour the parasite in smaller numbers without any harm resulting, for the drain caused by them is repaired by the system. Intermediate between these extremes is a class of cases in which some 100 to 300 of the worms are present, the action of which might be withstood for a very long time in a healthy person, but if at the same time some other debilitating or anæmia-producing disease is also at work, such as malaria, dysentery, syphilis or bad feeding (especially a deficiency of nitrogenous foods), then such a number of the worms will play an important part in producing anæmia. These cases are not, however, pure anchylostomiasis, but malaria, &c., complicated by the action of the anchylostomum, and should be so classed. Such cases are, in my experience, much more commonly met with on tea gardens in Assam than pure uncomplicated anchylostomiasis, and thymol will be a necessary pre-

liminary in their treatment, that of the primary condition being afterwards attended to.

It is evident both from the Assam dispensary cases, and those of Dr. Galgey, that it is cases of malarial cachexia which are most frequently confused with anchylostomiasis, and as it is just in these cases of advanced malarial cachexia that thymol is so dangerous, so that the differential diagnosis of these two forms of anæmia becomes of great importance. The clinical differences are well known, and I do not propose to repeat them here, more especially as I have published a table of them elsewhere. I wish, however, to refer to a method of differentiating them by means of an examination of the blood. It occurred to me that as the anæmia of anchylostomiasis is caused by long continued small losses of blood from the intestinal mucous membrane, while that of malaria is brought about by the destruction of the red and white corpuscles of the blood by the plasmodium malaria, the hæmoglobin being retained in the system in the form of pigment, that the type of the anæmia in the two diseases might be expected to differ considerably. I therefore made a full examination of the blood in both diseases, with the result of finding that the differences were so great as to be of diagnostic value. As a full account of this research will appear elsewhere, I need only refer briefly to the main points established.

Firstly: in the anæmia of malaria the percentage of hæmoglobin and of the red corpuscles are equally or nearly equally reduced, so that the amount of colouring matter in each corpuscle, or the colour index, is about the normal (1·0). On the other hand, in anchylostomiasis the percentage of hæmoglobin is reduced about twice as much as the number of the red corpuscles, so that the colour index falls to one half of the normal (0·5). Secondly: in malarial anæmia the number of white corpuscles are proportionally more reduced than the red, so that only 1 white to 1,000 red, or even, in extreme cases, 1 to 2,000 are found. In anchylostomiasis, on the contrary, the white corpuscles are increased in numbers relatively to the red, 1 to 300 or 400 being commonly met with. Thirdly: the specific gravity of the blood is more reduced, relatively to the degree of anæmia, in anchylostomiasis than it is in malarial anæmia. These differences I have found to be so great, that the extreme figures met with in pure cases of the one disease did not overlap those of the other, so that they are of absolute diagnostic value. Moreover, I found that in cases where the two diseases complicated each other the blood changes were intermediate between those of the two primary conditions, so that in any case of malarial anæmia in which the colour index is unusually low, the presence of anchylostoma in harmful numbers may be rightly suspected. To give an example: in a case of chronic malarial fever in which I examined the blood, I found an unexpectedly low colour index. Thymol was given, and 159 of these worms were passed.

This difference of type of the anæmia is also of practical importance in furnishing a guide to treatment, for in malarial anæmia arsenic is indicated in order to increase the output of corpuscles by the bone marrow. It is worthy of note that I have found the marrow of the shafts of the long bones to be constantly converted into red marrow in malarial cachexia, just as it is in pernicious anæmia. Iron is of much less importance in the treatment of malarial anæmia, for there is plenty in the system in the form of pigment. In anchylostomiasis, on the other hand, iron is the essential drug, and can best be given in the form of dialysed iron or the sulphate of iron, which interfere least with the already weakened digestive powers.

It must also be borne in mind that in some advanced cases of undoubted anchylostomiasis very few or none of the worms may be present in the intestines at the time the patient comes under observation, owing to most of them having dropped off. In such cases thymol can do no good, while by irritating the already sorely tried intestinal mucous membrane, fatal diarrhœa may easily be set up by this powerful drug. An examination of the fæces should, therefore, never be omitted in advanced cases. The simplest method is to spread a small piece of fæces out in a drop or two of 1 in 20 carbolic acid (which removes all odour) under a $\frac{1}{8}$-inch coverslip, and to systematically examine the whole specimen under the microscope. I have found by experiment that in this way the ova of the worm will be easily detected if as many as twenty of the worms are present, while the number of ova found will serve as a rough test as to the probable number of worms present. If no ova are found by this method, if any worms are present they will be so few that they may safely be neglected. As these worms cannot increase in numbers within the body, but each must be introduced from without, it is obvious that there is no necessity to remove the very last worm by repeated doses of thymol. Much harm may be done in the attempt to do so, when the patient is in a very debilitated state, while no appreciable good can result from the removal of the few worms left after one or at most two efficient administrations of thymol. In some extreme cases it will indeed be advisable to treat the patient with strychnine and digitalis for a week or two before giving thymol at all.

Summary.

Firstly: it has been proved by Dobson and myself that from 60 to 80 per cent. of the inhabitants of Assam, Bengal, and many other parts of India, harbour the anchylostomum in numbers varying from a very few to 100 or even more. The mere accidental presence of such numbers of these worms does not, however, constitute anchylostomiasis, for I have shown that the loss of blood caused by them is fully repaired by the system, and no disease results.

Secondly; in anchylostomum-infected districts, in addition to the first class, cases will be met in which some debilitating or anæmia-producing diseases are complicated by a considerable number of these worms, say from 100 to 300, the drain caused by which might be withstood for a very long time by a healthy man, but which when added to such diseases will play an important part in the production of anæmia and cachexia. In my experience on tea-gardens in Assam, this class of cases is far more commonly met with than pure, uncomplicated cases of anchylostomiasis, and they should be returned under the head of the

primary disease, and stated to be complicated by the presence of the anchylostomum.

Thirdly: we have cases in which some 500 of the worms have been present for upwards of six months, and have by themselves produced distinct anæmia and other well-known symptoms. These constitute true anchylostomiasis.

In both the last two classes of cases thymol is indicated to remove the worms as a preliminary measure to the treatment of the anæmia, but it must be used with caution in advanced cases, and especially in those which are complicated with malaria or dysentery. In cases of other diseases in which these worms are accidentally present, thymol may also be given if not otherwise contra-indicated, but such cases should not be classed as anchylostomiasis.

Lastly: the mere presence of anæmia in a tropical climate should not be considered as an indication for dosing the patient with thymol, without first examining for the ova of the worm, and studying the type of anæmia met with, and all possible causes of the anæmia other than anchylostomiasis. Such a rule of treatment is both unscientific in its inception, and, as I have shown above, may also prove disastrous in practice.

I had intended to have discussed the way in which this worm causes disease, in the light of some microscopical work that I have done on this part of the subject, but as this paper is already a long one, I must leave this part of the subject for another communication.

LEPROSY IN THE CANARY ISLES.

By Stanford Harris, M.D., M.R.C.S.

The inhabited islands of the Canary group are:—Teneriffe, Grand Canary, La Palma, La Gomera, Hierro, Lanzarotte, and Fuerteventura. They lie in the North Atlantic between 27° 4′ and 29° 3′ N. lat. and 13° 3′ and 18° 2′ W. long. The most easterly—Fuerteventura—is 50 miles distant from the coast of Africa. Politically they form a sub-province of Andalusia, in Spain. In Las Palmas, the capital of Grand Canary, there is an asylum for lepers, which admits sufferers, as voluntary patients, from the seven islands. These are free to come and go at will, the inhabitants having no fear of catching the disease. The remark made by Dr. William Robinson in his interesting address on Consumption and the Means to Avoid Contagion (see *British Medical Journal*, July 23, 1898), to the effect that "there is no need to treat the sufferer as a leper," would have to be transposed to express the Spanish feeling in the matter, into such a statement as this: "There is no need to shun the leper as a consumptive." Here the fear of phthisis is far greater than of leprosy. All articles which can be burnt are so destroyed which have been used by a tuberculous patient, and the rooms which have been occupied by him are disinfected. This view as to the slightness of the danger of becoming infected by leprosy appears to be justified in these islands. There is no record of any nurse or attendant at the hospital having contracted the disease, although certain histories of patients point to the likelihood of their having acquired the disease by very close intimacy, such as eating and sleeping with those affected.

I propose to give an epitome of 15 selected cases, now in the asylum in Las Palmas, and to briefly comment upon their histories afterwards.

I may premise these by the statement that the register of admissions to the hospital dates from the year 1845, since when 544 lepers have been admitted, making an average of a little more than 10 per year. Since July, 1890, the register has been kept under a rather different system, which enables me to give the following details:—From 1890 (July) to 1898 (July) 42 males were admitted and 35 females.

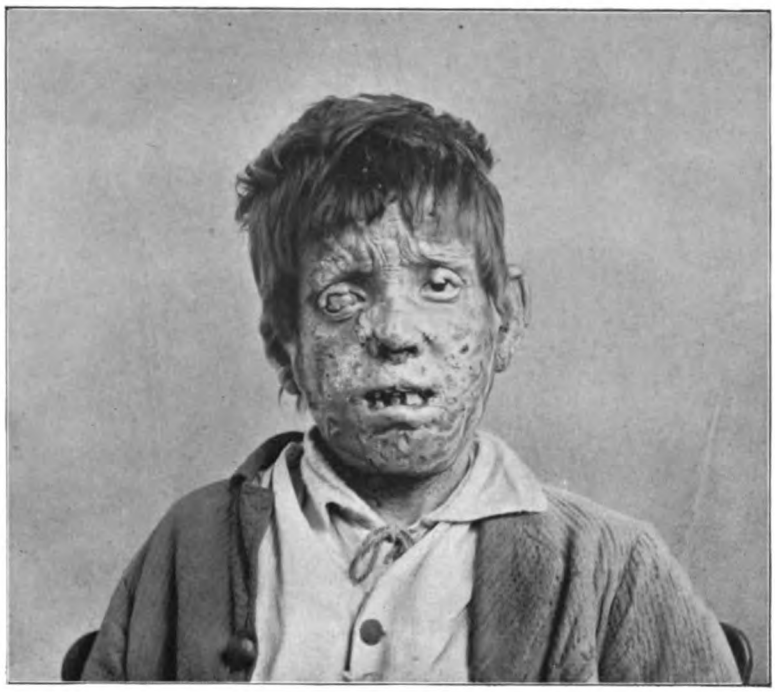
The 42 men are accounted for as follows:—13 have died, 18 are still in hospital, and 11 have left the asylum. Of the 35 women, 11 have died, 15 are still in hospital, and 9 have left.

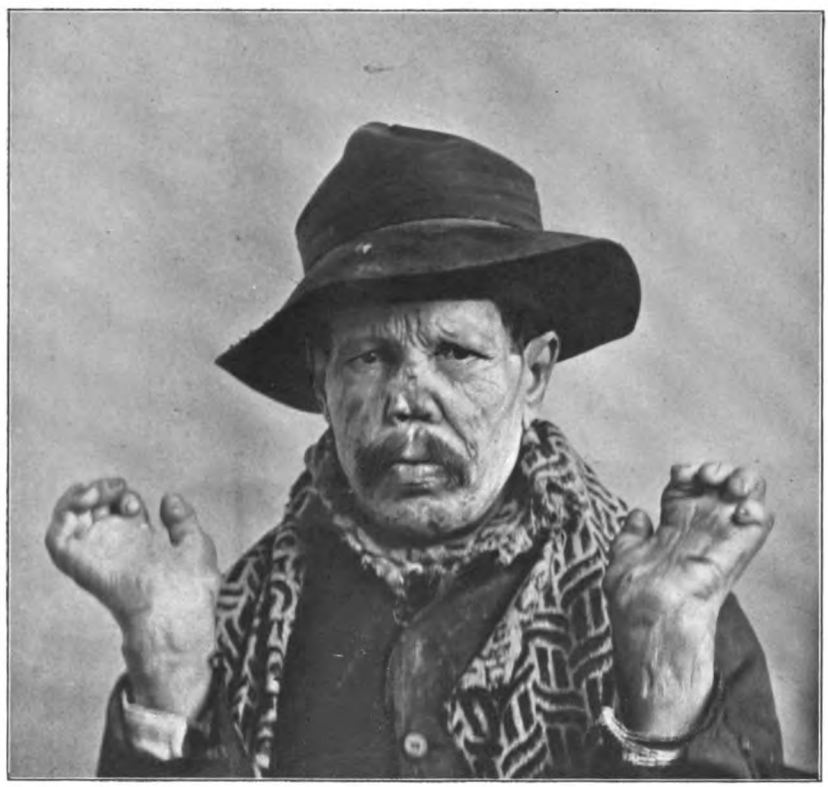
The islands supplying the 42 males are as here given:—Teneriffe, 15; Grand Canary, 12; La Palma, 7; La Gomera, 6; Fuerteventura, 1; Hierro, 1; Lanzarotte, 0. Of these males one was married, one a widower, and the rest unmarried.

I. — José Amador, aged 22, native of Fuerteventura, now in the hospital of San Lazaro, Las Palmas. Parents alive and well. Has had 12 brothers and sisters. No relations have ever been affected by leprosy. (In all cases the relations are asked about as far back as the grand-parents, and collaterally as to uncles, aunts, and cousins.) He never lived in association with lepers, but there were three lepers in his native village, an inland one. His food was gofio (a native meal, made by grinding and roasting maize, to which a little salt is added), potatoes, fish (fresh and salt). Parents' food had been the same, except that, having lived nearer to the sea for most of their lives, they had eaten more fresh fish.

The disease commenced, as well as he can remember, seven years since. He slightly burnt the calf of his left leg over a brazier (there is no cicatrix now visible), and after that a swelling followed, which he says has remained the same ever since (it is a soft, slightly discoloured swelling about the size of a large cherry). Other swellings followed on both lower limbs, and he lost sensation over the right elbow and adjacent parts.

Present Condition.—His face is covered by tubercles (in size averaging a large cherry). Some of them are broken—he scratches them, owing to their itching a great deal. There are translucent swellings on the outer sides of each cornea, at its junction with the sclerotic. The lower limbs have numerous tubercles, the original one on the left calf not being more advanced than the rest. There are patches of anæsthesia here and there on the lower limbs and over the right elbow. The hands are almost completely covered by tubercles. Beyond the itching there is little discomfort or pain. A bit of tubercle cut from the face, and examined under the microscope with a $\frac{1}{12}$ Zeiss oil immersion and with Abbe's condenser, showed bacilli about two-thirds the size of the tuberculous bacillus of Koch, lying within the large





LEPROSY IN THE CANARY ISLES.

By STANFORD HARRIS, M.D., M.R.C.S.

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cells which have been described by Virchow. Each cell contained on an average four bacilli.

II. — Domingo Hermandez, aged 16, native of Santa Cruz, Teneriffe, now in hospital of San Lazaro. No relations, as far as he knows, have ever been affected by leprosy. He has one sister. He has suffered from the disease for three years. It commenced by loss of sensation ("sleeping") over the cheek-bones on both sides.

Present Condition.—Over the malar bones there are slightly raised, faintly purplish, cushion-like swellings, which are completely insensitive. These are the only evidences of the disease. They slightly increase in size every year, so he states. His food has been the usual food of the poor, as in Case I., except that until he came into hospital he ate pork almost every day. He drank water, seldom wine or coffee.

III.—Antonio Rodriquez Melina, aged 34, native of La Palma—up country, now in the hospital of San Lazaro. Relations all healthy. None ever suffered from leprosy. Has two brothers. Five years ago his knees "went to sleep"—(he is still insensitive in those parts)—and they ache a great deal.

Present Condition.—Has tubercles all over the face, smaller than those in Case I. A few are broken by scratching. They itch very much, especially when he is warm at night. Seventeen years ago he went to Cuba, and returned four years ago, one year after the commencement of the disease. His food consisted of the usual gofio, vegetables, salt and fresh fish, while in his native island, with the substitution of rice for gofio while in Cuba. He has eaten pork "every few days" of his life, but it has always been first boiled for several hours.

IV.—Hermano Consales, aged 31, native of Port Ovotava, Teneriffe, now in the hospital of San Lazaro. A paternal aunt died of leprosy in Teneriffe, and a cousin died in this hospital of the same malady, No other relations have been affected, including two brothers and one sister. He has been a sufferer twenty-one years. For six or seven years before the disease commenced, that is, before the age of 10, he lived with a leper (who is still living). He ate with him, and was with him daily, but never slept with him. "White" patches appeared on his face at the commencement, and tubercles followed on the face, ears, eyes, and feet.

Present Condition.—There are numerous tubercles on the face, and there are extensive scars of cicatrical tissue. Tubercles are present at the outer corneal margins of both eyes, the hands (which he keeps in bandages) are swollen and ulcerated. His food has been gofio, potatoes, green vegetables, vegetable soup, and "when they killed a pig" fresh pork and sausages. He has always been fond of wine, and has drunk a great deal (three or four pints of native wine, "when he could get it.") This, it may be noted, is not a usual failing among the Spaniards. He also ate salt fish from Africa. He has talked with people on the Spanish-African coast, who told him that there were lepers there, where the fish was salted. He was a sailor (conscript) for one year, but was discharged on account of the leprosy. He was principally in Venezuelan waters while at sea.

V.—José Gregorio Sacramento, aged 41, native of La Gomera, now in the hospital of San Lazaro, Twenty-two years ago the disease commenced, so he says, at the tip of the nose (there is no mark or anæsthesia discoverable there now). No relations affected, but he knows nothing of a brother who is away in Cuba. Has never associated with lepers till he came into hospital.

Present Condition.—The cartilage of the nose has completely disappeared, the fore part of the nose has collapsed in consequence, but the skin over it appears sound. His face is very anæmic (not the rule here). He keeps a band round his forehead, but the skin is only slightly reddened beneath it. His fingers are shrunken and contracted into the palm of his hand (see photograph, which has been taken to illustrate the condition thus spoken of by Dr. Hillsbrand: "Simple anæsthesia of particular nervous provinces in the forearm, particularly the ulnar, with contraction of one or more fingers, but without any ulceration of the affected skin, I have occasionally observed also, but their leprous character was not fully acknowledged. Since I visited China and India, however, all doubts on that point have disappeared from my mind.") There are ulcers on the ankles and feet which do not itch. His food has been gofio, vegetables, fresh fish, and fish salted in his native island of La Gomera. He has eaten very little pork. No parts of his body are anæsthetic. Bacilli were obtained from the ulcers on the ankle.

VI.—Hernandez de la Concepcion, aged 23, native of La Palma, now in the hospital of San Lazaro. His father died a year ago of leprosy, and had contracted fingers as exhibited by his son. A brother and a sister are also affected, both younger than he is, and still in La Palma. He knows of no other relation ever having been affected. The disease commenced about eight years ago by loss of sensation in the feet (always described by the patients as "sleeping" in the part affected). Now he has contracted fingers, but less marked and less atrophious than in the preceding case. There are ulcers on the ankles and small tubercles on the body generally. Scratching—they itch very much—has produced a scabies-like rash. There are translucent tubercles on the outer margin of the left cornea. There are areas of anæsthesia throughout the body. The diet has been gofio, yams, vegetables, salt fish (from Africa), and salted pork. A little wine occasionally.

VII. — Domingo Pacheca, aged 13, native of Villa Orotava, Teneriffe, now in hospital of San Lazaro. No relative, he believes, has ever been affected. Commenced five years ago. His mother attributed it to a dog-bite in the thigh, but there is no mark there (it is perhaps unnecessary to remark that leprosy is unknown in the lower animals). His face is covered by tubercles, the general appearance as to colour, &c., viewed at a little distance, suggests a case of erysipelas. His food was the usual food of his class—gofio, salt fish, &c.

VIII.—Hernero Garcillo, aged 17, native of Villa Orotava, Teneriffe, now in hospital. His maternal grandfather died of leprosy, the only relation affected. He has four brothers and four sisters, healthy. He spent his childhood almost entirely in his grandfather's house, but never slept with him. Has suffered

four or five years; the elbow was first affected, but whether by insensibility or tubercle he does not remember.

Present Condition.—There is one tubercle on the right eyebrow and a small one on the chin, small warty tubercles on the elbows. His food, taken usually in company with his affected grandparent, was the usual diet already described.

IX.—Manuel Maison, aged 19, native of Las Palmas. (This is the case photographed—No. 2—to show a severe case, with complete destruction of one eye.) His mother died in hospital here about six years since. His maternal grandmother had contracted fingers, but died of some other complaint. A maternal aunt died of leprosy. His food has been similar to the rest, but he has taken very little pork.

Present Condition.—Face, as shown in photograph, is very much disfigured, the right eye is converted into a fleshy mass, the tip of the nose is destroyed, the left eyebrow eroded. The whole of the face presents an eczematous appearance. His lower limbs and hands are tuberculous and ulcerated. The trunk is free from any appearances of disease. The disease commenced by "sleeping" in the elbow about seven years since.

X.—Philipo Cartero, aged 44, native of La Gomera, now in hospital of San Lazaro. A male cousin died of leprosy eight years ago. No other relation has had leprosy. Never associated with lepers. First attacked fourteen years ago, while he was in Cuba, where he lived for ten years. His food there was rice, meat (not pork) and vegetables. He took a good deal of wine there. In La Gomera he took the ordinary food of the peasant, gofio, &c. The disease first showed itself by "sleeping" in the elbow, followed by tubercles on the forehead. Now, the nose is depressed at the junction of the cartilage and bone. There are tubercles scattered over the face, some of them broken. There is a translucent patch at the outer margin of the cornea. The hands are covered by tubercles and old scars. The lower limbs, below the knees, are similarly affected. The body is free. There is no pain, but itching is troublesome.

XI.—Salvador Noda, aged 30, native of La Gomera, now in hospital (for the second time). No relations affected. Has three brothers and two sisters. Never associated with lepers before the commencement of the disease. The leprosy first showed itself in Cuba, where he lived seven years, by "sleeping" in the elbow. The nose is slightly swollen across the junction of cartilage and bone. The arms present a few slightly raised purplish patches. This is a very slight case as regards appearances. His food in Cuba was rice, milk (which is taken in Cuba much more by peasants than is customary in the Canaries), and some flesh. In Gomera, the usual diet, but little fish.

XII.—Antonio Sanchez Manté, aged 16, native of Grand Canary (up country), now employed in the hospital, but lives in the town of Las Palmas. Has been affected one year. His mother has leprosy, and one sister. They live in the town. A brother also is affected, and he slept with him for some time before he himself was attacked. The disease commenced by "sleeping" in the heel, followed by ulceration on the hands. All these ulcers are now healed, except one on the little finger. There are a few scattered ulcers on the lower limbs, downwards from the lower third of the thigh, to the heels, and from the middle of the arm of the upper limbs. The cicatrices on the hands are smooth and uncontracted. The trunk and face are unaffected. His food, the usual diet of gofio, salt fish, &c., occasionally pork. Has never taken wine.

XIII.—Hilo Imesa, aged, 14, born in La Gomera, now in hospital. Affected seven years. No other member of his family has been a leper. Has three brothers and four sisters. Never associated with lepers, but he has had lepers as neighbours and occasionally has been in their houses. Ulcers in the heel were the first symptoms. The eyes were early affected. Never had any loss of sensation.

Present Condition.—The right eyelid is swollen, the outer margin of the cornea is opaque, and the corneal vessels are injected. The left eye is in a state of conjunctivitis. Tubercles are scattered over the face. Some look cheesy. The arms, below the elbows, have ulcers and cicatrices, the legs and ankles are similarly affected. There is itching. His food has been the usual food of the poor (gofio, &c.), except that he has never taken pork, except once "to taste it."

XIV.—Tomas Mendez, aged 44, native of Villa Orotava, Teneriffe. Affected eight years, in hospital here two years. No relation ever affected. Was eighteen years in Cuba, having returned four years ago. Has four brothers and three sisters. Has never lived in the slightest association with lepers till he came into hospital. His first symptom was a foot "going to sleep"; the next, ulceration in the neck.

Present Condition.—Except the face, this patient is completely covered from the neck to the tips of his toes with a psoriasis-like rash with a dark purple basis; there are a few tubercles, some open, on the fingers. There is considerable loss of sensation, with a sense of "heat" in the lower limbs. In Orotava he took the usual diet; in Cuba rice, pork, butter and vegetables.

XV.—Diez Castrero, aged 26, native of La Palma, now in the hospital of San Lazaro. One brother, aged 26, died in this hospital of leprosy eight years ago. No other relations have suffered from the disease. As soon as the brother mentioned showed signs of the disease his father sent him "further into the country," as he believed it was contagious. This patient states that this is the general belief in the island of La Palma. The disease first showed itself by "sleeping" in the knee, afterwards nodules appeared on the face.

Present Condition.—He is in bed and speaks very hoarsely, owing to both hard and soft palate being affected. He is covered by a dark purplish rash which does not disappear on pressure. He says the blotches, now purple, were originally white in colour. Scattered at intervals over the blotches are nodules and ulcers. On the limbs there is an eczematous eruption, extending in the case of the lower limbs to the tips of the toes. There is a small pannus on the outer side of the left cornea. His food was that already described, except that he always ate a great deal of pork, both fresh and salted. He took freely the native (Vino Tinto) wine, but not to excess. He

has smoked cigarettes largely, but cannot smoke any more, owing to the condition of the mouth.

Since it has been occasionally suggested that leprosy is probably introduced into the system by the food, the question of diet has always been inquired into. All the sufferers here have been eaters of the gofio, and almost all have taken largely of the salted fish, in most cases imported, in a state of imperfect preservation. The imported fish come from infected countries. The history of many cases shows a family tendency to the disease, while others appear to have never been exposed to contagion. As far as I know the well-to-do are never victims of leprosy here.

Papers read at the Annual Meeting of the British Medical Association.

PELLAGRA IN EGYPT.

By F. M. Sandwith, M.D., M.R.C.P.

Physician to and Lecturer on Medicine at Kasr el Ainy Hospital, Cairo.

In 1890 I began to study the anæmia of Egypt, which seemed to fill the hospitals with peasants and soldiers living an outdoor and apparently very healthy life, and it was some time before I stumbled on the fact that all this was due to anchylostomiasis,[1] and later on I learnt that Griesinger in 1851 had proved that the anæmia was due to *anchylostomum duodenale*. But although his work was done in the same Cairo hospital, the remembrance of his researches had entirely died out, and a new generation of native doctors had arisen who had never heard of Griesinger or Pruner, and the discovery had to be made a second time. Eight years ago there was no English literature and no great interest in tropical disease, and I went myself to Egypt quite ignorant of the diseases of that country. In 1893, while collecting a few records for publication, I became aware that a large number of my anchylostomiasis patients showed a symmetrical eruption which sunburn, chapping and dirt could not explain. I eventually decided that some of them had pellagra in addition to their anæmia, and though there was no one in Egypt who could give any information on the subject, I was confirmed in my suspicion by Italian physicians visiting my hospital wards.

In 1894 I first returned some hospital patients as pellagra, and the following year I spent part of my holiday in Italy, where I saw more than 100 cases of pellagra in lunatic asylums, pellagra retreats, and the insane wards of general hospitals.

Since 1893 I have had more than 500 cases of pellagra in my hospital wards, besides many others seen in other sections of the hospital and at the lunatic asylum. This high number is partly due to the fact that the peasants themselves have discovered that interest is now taken in this disease, and they tell each other to go to Cairo for treatment in gradually increasing numbers.

[1] "Four hundred cases of Anchylostomiasis," in *Transactions of Eleventh International Medical Congress*, 1894.

History in Egypt.

Dr. F. Pruner, under the heading of "Leproses," writes as follows[1]:—"Pellagra is sporadic in Egypt and such as we have studied it in Milan. We have seen three cases of it among the peasants, one of whom presents to-day, twelve years after our first seeing him, paresis of the upper limbs, with retraction and muscular atrophy." This is almost the only serious mention of the disease, though one or two writers have referred to a pseudo-pellagra, which may have been the real malady. Dr. A. Figari Bey writes of venereal disease appearing in Lower Egypt, with "a kind of leprous pellagra."[2]

Pellagra has been known in Europe since 1735, when it was described in Oviedo, in the north of Spain (Hirsch).

Geography.

Until now the disease was believed to be confined to countries between 46° and 42° N. latitude, but I can vouch for its being present as far south as Assouan or the Tropic of Cancer, latitude 24° N.

The geographical range depends on the food supply and the poverty of the inhabitants. I imagine pellagra can be found in any part of the world where bad maize is the chief diet of the poorest classes.

For the purposes of this paper I have analysed all the cases admitted under my care in the year 1897. These were altogether 178, but it is only in 164 of these that their homes could be accurately determined.

Lower Egypt.		Upper Egypt.	
Galioubiyeh ...	47	Ghizeh	24
Menoufiyeh ...	29	Beni Souef ...	2
Dakahlieh ...	18	Minieh	1
Gharbiyeh ...	14	Fayoum	—
Charkiyeh ...	10	Assiout	7
Behera	—	Girgheh	1
Cairo	9	Keneh	1
		Sohag	1
	127		37

These figures show the great preponderance of the disease in Lower Egypt, where maize is much grown and eaten. If we except Ghizeh, which is the province adjoining Cairo, we see that the disease is rare in Upper Egypt, where the peasants live chiefly on millet (Sorghun vulgare), and not on maize.

The greatest number of cases naturally come from the provinces nearest to Cairo, and it is probably the long distance from the hospital which accounts for the absence of patients from Behera. It must be remembered that the poorest patients come scores of miles on foot to the hospital, begging their way. Pellagra is said to be unknown in the Fayoum, where the natives eat no maize. Old students have assured me that it is rare in the neighbourhood of Luxor for the same reason, but I saw several cases there last February, both in hospital and among beggars.

In 1891, after a famine, Surgeon-Captain Myles, fresh from seeing my patients, found four cases of pellagra among 6,000 starving Arabs at Tokar, on the Red Sea. My figures show very plainly that it is a

[1] "Topographie Médicale du Caire," Munich, 1847, p. 67.
[2] "Studii Scientifici sull' Egitto," Lucca, 1865, part v., p. 334.

country-bred disease, not attacking townsfolk, who have a more varied diet, and are not so much exposed to the sun as the peasants. The Cairenes admitted for the disease are generally beggars. In Alexandria, Port Said, and Suez, the disease is very rare.

Nomenclature.

As the disease is only now beginning to be recognised by Egyptian doctors, it is hardly to be expected that the peasants should distinguish it. But they have two names for the eruption. *Qushuf*, often pronounced *'ushuf*, means chapping, and the peasant believes that his skin is affected by the action of cold wind and water. They more rarely make use of the synonym *gofar*, which is also used for an eruption which attacks camels, and sometimes horses.

Not even the sick, after they are cured, can be made to believe that the eruption is an outward and visible sign of a cerebro-spinal disease.

The nomenclature drawn up by the Royal College of Physicians, London, in 1896, classes pellagra among general diseases, but as lately as 1884 they included it in diseases of the skin. It is no more a skin disease only, than are elephantiasis, scurvy, syphilis or leprosy.

Many derivations have been suggested for the word "pellagra." The simplest seems to me the best, for *pell' agra* in Italian means rough skin.

Age.

The bulk of the cases are men in the prime of life, who ought to be working for their country and their families. The following figures are taken from the hospital statistics for the three years 1895-6-7. In no year were there any cases under the age of five, but it is quite common to have boys about the age of puberty. Patients above fifty are rare, probably because they die of concurrent diseases or drift into secondary dementia.

	5-20	20-40	40-60	Above 60	Total
Pellagra and Anchylostomiasis	41	225	15	2	283
Pellagra, Anchylostomiasis, and Bilharziosis	32	119	3	—	154
	73	344	18	2	437

Every case during these three years has been complicated by the presence of anchylostoma worms in the intestine, and about one-third of the patients have been troubled in addition with changes in the urinary passages, or in the lower bowel due to distoma hœmatobium.

Sex.

In the three years out of 437 cases only eight have been females. This is not entirely due to the fact that Mussulmans don't like sending their women to hospitals, because out of a total of 4,723 patients at Kasr el Ainy in 1897, no less than 1,516, or nearly one-third, were women and children under six years.

If the whole number of 1,516 were analysed, and all children, Magdalenes and town dwellers were excluded, a relatively small proportion of country folk would remain, and these, as I shall show, are the potential pellagra class. The fact that men seem to suffer more than women in Egypt is the converse of experience in Italy, where the chief sufferers seem to be peasant women. This is probably due to the fact that in Egypt women are much less employed as agriculturists, while in Italy it is they who do most of the field work.

However, if one had time to go into the country districts and search for pellagra, I imagine many cases would be found among the women, who by reason of poverty or etiquette cannot get as far as a Cairo hospital.

Nationality.

All cases were Egyptian, not Nubians or Soudanese, who seldom lead the same life as the Egyptian fellah, though I daresay some have exactly the same food. Some of the predisposing causes, such as dirt, favus and anchylostomiasis are rare among the Nubians and Soudanese.

Occupation.

Excluding some boys, three women and men, too ill to have any known occupation, there remain 156 patients admitted in 1897. Of these, 137, or 88 per cent., were peasants, and the remainder were—mason's labourers, 6; readers of the Koran, who are practically beggars, 4; boatmen, 3; policemen, 2; brickmakers, 2; potter, 1; and servant, 1.

They were invariably men of the poorest class, who had been out of work, and therefore out of food for some time. These figures again intensify the fact that pellagra is a country bred malady, seldom affecting townsfolk. The bulk of the Egyptian population outside towns are the fellaheen, and by the census of 1897, the number of land cultivators is calculated at about 2,050,000, or two thirds of the total male population.

Causes.

Italian writers[1] believe a good deal in the hereditary taint from pellagrous parents, but I have not been able to satisfy myself that such cases exist in Egypt, for boys yield more easily to treatment than youths and adults, and the disease is certainly not common in children under ten. The question of heredity may, however, be similar to that of alcohol. In July, 1895, I had the pleasure of visiting the *Pellagrosario* at Mogliano, near Venice. Here Dr. Enrico Locatelli kindly devoted a morning to me and showed me a hundred pellagrins of both sexes, sane and insane. Among them were several boys aged about 12, and some of these were called cases of hereditary pellagra. I could not, however, discover that there were any special characteristics, though many of them had heads, faces and teeth suggestive of rickets and syphilis. One baby a year old was at the breast of a pellagrous woman, it had no teeth, and its anterior fontanelle was larger than a shilling. It is obvious that such a child will start badly equipped in life, and that diseased maize will cause it to fall a victim to pellagra earlier than a healthy child would. Also it

[1] Lombroso, "Trattato profilattico e clinico della Pellagra." Turin, 1892. "Relazione Sanitaria for 1893," *Instituto Pellagrosi*, in Mogliano. Treviso, 1894.

might be more liable to relapses as is the case with "hereditary pellagra."

But if it be true, as is asserted,[1] that even children at the breast are fed partly on polenta in Italy, it must be difficult to prove that any given case is hereditary and not acquired.

The essential causes of pellagra, besides concurrent diseases, are bad maize, poverty, and exposure to the sun. Of these three, maize is the most important, and it is necessary that it be diseased and also that it be one of the chief articles of diet. Healthy maize varied with other food will never produce the disease, as seen by its absence among all but the poorest classes, and also among town dwellers, thousands of whom eat good maize regularly as one article of diet. Lombroso claims that he has found the fungus of bad maize, and by making a tincture of it has produced pellagra in dogs and poultry.

Clifford Allbutt[2] goes so far as to say that the fungus is *reticularia ustilago*, but he does not quote his authority. This question, and many other doubtful ones with regard to pellagra, will, I hope, now be worked up in Cairo.

The maize is either cut before it is ripe, gathered on rainy days (especially in Italy), stored away undried in damp, dark cupboards, perhaps still sheathed, or it is grown from diseased seed. Any of these causes tend to produce a toxin in the grain, which forms the pellagra poison in man. Also, it must be stated, that the poorer peasants sell the best of their maize at the market and keep the worst to eat at home.

In Italy the chief predisposing cause is alcohol among the poorest peasants; but in Egypt the peasants do not drink alcohol, and are very seldom victims of opium and hasheesh. Untreated syphilis and malaria are very common, and every patient is the host of one or two parasitic ailments, such as anchylostomiasis, bilbarziosis of bladder or rectum, favus and intestinal worms, such as ascaris and oxyuris. Sometimes an unhappy creature is actually suffering from all these parasites at the same time, which effectually prevent his earning his bread. Quite a limited amount of maize toxin would probably be sufficient in such a case to produce pellagra.

Kinds of Maize.

There are in Egypt two kinds of *durra* eaten, but as they are often confused, I must devote a few lines to first describing the one which has apparently nothing to do with the causation of pellagra—*durra beledy*, called also D. himegy and D. saify, is *sorghum vulgare*, which is known as great millet or Indian or Guinea corn. It is an annual cane-like corn grass, with a dense head of spikelets, bearing numerous small grains.[3] This kind of durra is eaten in Lower Egypt, and is the chief cereal of Upper Egypt.

Durra shamy, unfortunately named as if it was the Syrian variety of the above-mentioned, is *zea mays*, well known to the English as maize or Indian corn. It is an annual cane-like grass, which bears a dense head of closely packed grains as large as peas, enclosed in a sheath called the cob. It is a native of South America, and excepting rice, no other cereal is so widely cultivated in the world. It was introduced into Egypt from Syria about sixty years ago.

There are many different kinds of maize. I find my patients live chiefly on 14 varieties, and one-third of them eat one sort called camel's tooth, in addition sometimes to other kinds of maize, millet, and wheat.

Maize sown in Lower Egypt in July is ripe in November and December.

Storing of Maize.

It is obviously of importance to know how the fellaheen keep their maize after it is picked. I found by taking notes of 160 patients that 37 of the poorest bought it in the market as occasion arose, while 78 kept it without the sheath, 38 with the sheath, 5 as loose grains, and 2 as ground flour. The greatest number, therefore, store it as heads of grain, with or without the enveloping sheath. To keep it inside the sheath seems the easiest way of courting damp and disaster, and this is considered bad practice by the peasants themselves. It is also known to be wrong to store the heads (kooz) before they have been thoroughly exposed to the sun to get dried. But whether they keep the heads with or without sheaths, they have very primitive methods of storing the maize during the early months of the year, when rain may come and dew is always present.[1] The heads are either kept in small, dark, mud cupboards on the ground, with no sunlight and no protection from the mud floor, or in small attics on the roof, equally dark but not equally damp. Both these methods are warranted to cultivate fungi.

Preparing Maize.

I have also taken notes of how pellagrous patients eat their durra. Two-thirds of them eat it in the form of bread, while others have it roasted, and all eat it at times raw in the green state. This last is probably not dangerous if the parasite has had no time to form, and it must be a matter of future investigation to see what heat in cooking is necessary to destroy the poison. The habit of making Italian *polenta* does not seem to have found favour in Egypt.

Symptoms.

Where patients are very stupid and often weak-minded, and talk nothing but Arabic patois, it is difficult from them or from their friends to get a clear account of early symptoms, but I think one has a fairly clear idea of what the premonitory signs are. First of all a digestive group, constipation or diarrhœa, thirst, pains in epigastrium or abdomen, and all the signs of dyspepsia. Then come headache, vertigo, noises in the ears, lassitude, pains in the back and limbs, sleeplessness, and depression of spirits, besides anæmia, and palpitation on exertion, which two latter are probably caused by anchylostomiasis rather than pellagra.

[1] Hirsch, "Geographical and Historical Pathology," ii., p. 244.
[2] "System of Medicine," 1897, ii., p. 801.
[3] This is believed to have furnished the reed of St. Matthew and the hyssop of St. John, so has probably been a native of Syria and Egypt for hundreds of years.

[1] Though the annual rainfall at Cairo is only 1·2 inch, in parts of Lower Egypt it is about nine inches.

The symptoms of individuals arriving at the hospital, whether as out-patients or in-patients, may be conveniently described in this order, digestive, cutaneous, spinal and cerebral, which is more or less chronological. Early cases will probably show no mental change; late cases, while quite mad, will perhaps exhibit no evident skin lesions (pellagra *sine* pellagra).

The *tongue* is a very important member, and will sometimes help to decide a doubtful diagnosis. In 163 patients of whom I have notes, one half on admission had the tip and edges denuded of epithelium, another quarter had "bald tongue," the whole of the organ having lost its epthelium, while in the worst cases the palate also was bare. The remaining patients showed five coated tongues, and 37 which were normal except for anæmic pallor. The characteristic change in the tongue is therefore preternatural cleanliness in whole or in part. Of the 121 patients whose tongues were wholly or partly denuded of epithelium on admission, I have notes of 83 tongues on leaving the hospital—45 were returned as normal, while 38 were still somewhat denuded after treatment. This is, therefore, one of the symptoms which improves, and in recent cases can be cured, while in very chronic cases the improvement is only slight.

Parotid Gland.

I found in a few cases enlargement without pain of both parotid glands, and therefore took notes of this. Out of 151 patients, 145 were returned as normal, while only six had some enlargement on admission, which diminished or disappeared while in hospital. I have not seen this symptom mentioned in any book. It is associated with "bald tongue," and is common in children who have a good deal of eruption about the face, neck, and ears.

Teeth.

In Europe and America dyspepsia is so often occasioned by deficient teeth, that I thought it would be well to eliminate that possible cause. In examining 166 patients, I found the teeth of 120 were quite sound, 24 had lost one or two teeth, 21 had lost from three to eight teeth, and one old man had parted with all his teeth. On the rare occasions when a tooth was seen to be carious it was entered as lost. These figures show that 87 per cent. of the patients had perfect powers of mastication. The Egyptians are known to have excellent teeth, like other people unaccustomed to modern cooking and the use of knives and forks.

Bleeding Gums

Are occasionally seen among the insane cases, perhaps from lack of vegetables.

Bowels.

I have already said that constipation or diarrhœa may occur among the early symptoms, but they are not a necessary accompaniment of pellagra until its advanced stages. I have again notes of 166 patients in hospital, of whom 103 were normal, 9 had slight constipation, one of them being a fatal case, 46 suffered from slight diarrhœa, and 8 had excessive diarrhœa. One-third of the cases thus had much or little looseness of the bowels, partly due to pellagra, but partly due also to intestinal parasites and their thymol and purge treatment.

The eight cases of great diarrhœa were all pellagrous, and far too ill to be treated by thymol, and no less than five of them died. Dysentery may occur in the last stage of pellagra, but is not a symptom.

Urine.

Hirsch[1] says that the urine is often alkaline, and of low specific gravity, but I have not found this the case. In 167 cases the house physicians found it alkaline in only three individuals.

Weight.

Chronic pellagra invariably causes loss of flesh, and eventually the emaciation ends in the person being dried up like a monkey. I have notes of 156 cases who spent more than one week in hospital. Nineteen left the hospital with their weight stationary; these were mostly patients who remained but a short time. One hundred and one gained, and their average increase was 5·4 pounds; this is a satisfactory proof that two-thirds of the patients improve under treatment. Eleven of these only gained one pound, while three others gained 20, 24, and 31 pounds weight. Boys and youths gain weight out of all proportion to middle-aged men.

Thirty-six of my year's cases lost weight, the average loss being four pounds. Eight of them lost only one pound each, but the greatest emaciation was shown by a fatal case which lost 19 pounds before death. One melancholic, with chronic diarrhœa, scaled 13 pounds less, and another lost 11 pounds, chiefly because he refused food and indulged in phases of religious excitement lasting seventy-two hours at a time. One weak-minded boy of 18, who tore his clothes and tried to throw them out of window because his skin felt burning, lost four pounds weight. But then he had nine times the thymol cure during sixty-six days in hospital. The causes of loss of weight under treatment were diarrhœa, dementia or melancholia, and the thymol cure, which includes purging and liquid diet, for twenty-four hours.

I now come to the cutaneous symptoms, which being most apparent, have claimed so far the largest share of attention, and have, indeed, given the name to the disease.

Eruption.

Eruption begins as an erythema, not unlike a severe sunburn, and being confined to the parts of the body which are exposed to the solar rays, it was at first thought by some authorities that there was no such general disease as pellagra, and that the skin changes were all due to solar action. But the disease is never to be diagnosed by eruption alone, and is far too serious to be relegated to the dermatologists. The regular symmetry of the eruption is what strikes the observer most; the erythema or icthyotic mottling extends up the forearms and legs to exactly the same level, and finishes off in the same crescentic way.

The affected skin on the limbs is almost entirely confined to the extensor surfaces, though in advanced

[1] Vol. ii., p. 218.

cases there is roughness on flexor side of legs and on flexor surface of wrists.

The primary erythema disappears by desquamation, and leaves behind it patches of slight roughness on some of the extensor surfaces, such as elbows, insteps, knees, hands, and to a less extent, legs. These rough patches get caked with dirt, and look black or dirty grey. I originally took the appearance for dirt alone, and several of my colleagues in Egypt tell me that they made a similar error. It is this error and the fact that the patients are all country folk and the best European doctors townsfolk, which has prevented pellagra being recognised until now.

If anyone will take the trouble to clean thoroughly with turpentine the dark-coloured patches which the patient calls "chapping," and thinks of no consequence, he will find that there is a definite roughness on the site of the former eruption.

Given a continuance of the same conditions of food and life, the following year a second attack of erythema will occur, leaving at its subsidence a more decided roughness. This continues every year, and though the roughness becomes more marked every time, and perhaps invades the chest, neck and face, the original hypertrophy of the skin, due to inflammation, disappears, and a gradually increasing atrophy of affected skin takes its place.

As it is only the skin exposed to sun and wind which becomes affected, it is obvious that the affected sites must depend upon the clothes worn by the peasant. In Italy the women dress like other Europeans, and I have never seen the pellagrous skin changes excepting on hands, forearms, neck and face.

In Egypt the fellah wears less clothing, and the eruption varies with his costume. If he works in the fields almost naked, the skin of almost his whole body may be attacked, and this, though rare, I have seen in boys. If he wears a sort of night-gown, open at the chest, fastened only by a girdle and with loose sleeves, and nothing else but a skull cap, it is easy for the sun to burn his face, neck, chest, arms, hands, knees, legs and feet. The only covered part of the body which becomes hard and rough is the skin over the coccyx and great trochanters, but there are pressure spots due to the extra sitting and lying which the sick man is reduced to.

One hundred and sixty-five patients on admission had eruptions, which I noted as follows:—78 had only slight roughness, as I have described, while 75 others had much roughness on hands and lower half of forearms, backs of feet and insteps, knees, lower half or whole of legs, neck and nape, front of chest, face, ears and lips. The extensor surface of legs was in an early icthyotic state. Three patients were slightly exfoliating from the affected parts, and eight others were exfoliating very markedly, leaving patches of new skin beneath. The remaining patient had a thick, scaly, dirty grey eruption over the exposed parts, like psoriasis. It is thus seen that all the 165 cases of whom I have notes had more or less eruption on admission, though at least half of them would not be recognised as pellagrous by physicians unacquainted with the disease.

The eruption, whatever its form, is greatly modified by stay in hospital. Slight cases entirely disappear and severe cases lapse into slight roughness only. Out of 135 cases, whose state on departure was noted, 36 had clear skins without any sign of roughness, and 99 went out with more or less roughness still remaining. About one month of treatment will produce a great difference in a man who is desquamating extensively with brown, rough, scaly flakes of epidermis falling off his chest, face, neck, arms, and legs. I have not seen cases of the nails peeling off, which is mentioned by Italian writers.

Site of Primary Eruption.

The peasant is so stupid, and so little acquainted with pellagra, that it is difficult to get him to determine which part of his body was first affected by the "chapping." But on examining about 100 cases, I find that the parts are earliest affected in this order: Hands and forearms, elbows, feet and insteps, legs and knees, upper part of chest, and, last of all, shoulders, neck, and face.

Time of Primary Eruption.

In order to try and get at the truth of this, I have had notes taken of 153 patients by means of the Coptic months, which are used by agriculturists. The eruption appeared for the first time, according to months, as follows:—November, 13; December, 14; January, 43; February, 17; March, 13; April, 5; May, 6; June, 15; July, 7; August, 8; September, 8; October, 4.

This seems to bring out the fact that two-thirds of the cases begin between November and March, being the five months when the maize is most plentiful and the weather most damp. During the seven summer months there are comparatively few new cases. Some of the most intelligent have told me that if the eruption begins with them in February it leaves in May, if they continue to work, but will leave much earlier if they rest.

Itching and Burning of Skin.

Erythema causes a swollen, burning sensation, which may give place to itching when the acute symptoms have passed off. By questioning 164 people on admission, we found that 71 complained of itching sensations, 3 of burning pains, and 90 were troubled with neither.

Atrophy of Skin.

The skin of 161 cases was examined, and 100 were found to be fairly normal, but 58 patients under 40 years had more or less atrophy of neck, back of hands and feet, and more rarely other parts. One boy had a wrinkled face, which made him look very like a monkey, and all had prematurely old necks and hands. Three of the cases all being primary attacks, had some hypertrophy of the neck and hands, and I have no doubt this is quite common in cases before they ever come to the hospital. One man complained of two patches of deep anæsthesia (beneath the skin) on the extensor surfaces of right wrist and left elbow. Besides the loss of elastic and subcutaneous tissue, there is a gradually increasing deposit of pigment, which helps to turn youth into premature old age.

The chief spinal symptoms are changes in the reflexes and pain or tenderness in the back.

Knee Reflex.

The knee-jerk of 165 cases was noted on admission, and showed only three as normal, leaving 48 slightly exaggerated, 76 very brisk indeed, 15 feeble, and 23 absent. It is difficult to be certain whether the knee-jerk is normal or brisker than normal, but, in any case, it is clear that at least half of the cases are more lively than necessary.[1] Early cases show exaggeration as a sign of nervous activity, and it is only the last stages in which no reflex can be obtained. I have notes of 129 patients on leaving the hospital, and they show 16 per cent. normal, 54 per cent. slightly exaggerated, 10 per cent. very exaggerated, 12 per cent. diminished, and 8 per cent. absent.

This shows an improvement, and it is quite common for feeble and for very brisk jerks to become in hospital slightly exaggerated or normal. Ankle clonus was often looked for, but both it and wrist clonus are quite rare, and occurs only in some incurable cases. Skin reflexes, such as the epigastric and abdominal, follow the course of the knee-jerk when it is distinctly too brisk, but their absence cannot, I think, be necessarily taken as a sign of disease. Like other workers, I found many cases of asymmetrical knee-jerk, some of which persisted on leaving the hospital, and were associated with unequal pain in the back.

Pain in the Back.

This is an important symptom, for the knowledge of which I am indebted to an afternoon spent with Dr. Gonzalez, at his asylum at Mombello, near Milan. He has had thirty-five years' experience of pellagra among Italian lunatics. Some patients will tell you that they used to have pain in the back,[2] but in most cases you must discover the tenderness for yourself, by pressing a knuckle at the sides of the dorsal and lumbar vertebræ, where the spinal nerves appear.

On this subject I have notes of all the 178 cases admitted during 1897, as I wanted to try and discover which of the nerves seemed most affected. In 59 cases there was no pain or tenderness on pressure, while in 42 cases the whole back was complained of, and I imagine that the pain was caused by rheumatism as well as pellagra. Twelve patients had tenderness between the 1st and 4th dorsal vertebræ, 35 flinched between the 5th and 8th, 17 between the 9th and 12th, and 13 had pain only near the lumbar vertebra—19 of all the cases had marked asymmetry of tenderness.

The figures are enough to show that although back pain is a symptom in only one-third of the cases, it is chiefly the mid-dorsal region where it is likely to be present. This is corroborated by the native treatment, for in 13 cases there were cautery or seton marks near the 6th dorsal vertebra. Back pain is one of the symptoms which is almost certain to disappear if the patient is willing to stay long enough in hospital. Of my cases, 85 per cent. had no pain when they left hospital, and 15 per cent. still had a little tenderness or flinched at the firm pressure of a knuckle. One boy, aged 13, was cured, though he had a history of being obliged to walk with his head bowed down, because of the pain in his back. On admission, he had great tenderness on the right side at the 10th dorsal, and on the left, from the 7th to 11th dorsal. He had cautery marks as large as a penny over the 2nd dorsal and to the left of the 9th and 10th dorsal vertebræ.

Temperature.

Pellagra is essentially a non-febrile disease, for among 158 observations I have notes of only one that had a little fever. This was a fatal case, and his temperature was about 100°, evidently caused by complications.

I have omitted to count all cases where a few days' fever was produced by malaria or other accompaniment.

Sleep.

I have already mentioned sleeplessness among the prodromata, and it is a symptom commonly present in acute or advanced cases.

Among notes of 150 patients on admission I find that 40 per cent. slept normally, say, from 8 p.m. to 4 a.m., with an occasional short sleep by day; 23 per cent. slept only about six hours; 19 per cent. slept for about four hours; and 18 per cent. slept for less than three hours in the twenty-four. Insomnia to some extent was therefore present in more than half the cases. With the exception of a few incurable patients, everyone sleeps all right after three or four weeks' rest in hospital.

Tuczek[1] mentions several nervous phenomena which are to be seen in Italy, but which so far I find rare in Egypt. Among them are tonic and clonic convulsions, epileptic attacks, spastic paralysis, atrophy and paralysis of muscles of lower limbs, unilateral ptosis, and some other affections of eye and spinal cord. Here we must remember the very different type of temperament.

The Italian peasant is addicted to alcohol, subject to irritability, frenzied excitement and diseases of brain and spinal cord, and therefore is more likely to exhibit nervous and mental symptoms than the Egyptian fellah, who is phlegmatic, stupid and sheep-like, with an undeveloped brain and a happy immunity from much nerve disease except that occasioned by syphilis.[2]

One of my Egyptians, however, walked very badly with staggering gait, had head tremors and hardly

[1] My figures may be compared with Professor Raggi's, who found, in 36 cases, 59 per cent. with lively knee-jerk, 5 per cent. exaggerated, 22 per cent. weak, and 14 per cent. absent.—"Reflessi tendinei nei pellagrosi," being a reprint from the "Rivista Clinica," 1883.

[2] Some of the Italians I saw had a history of cramp-like convulsions, causing them to arch their backs. There are seven varieties of Italian pellagra, and two of them are "those that go backwards" and "those that are doubled up."

[1] Pellagra article in Hack Tuke's "Dictionary of Psychological Medicine," London, 1892. I myself saw three cases of spastic paraplegia in Mogliano.

[2] Dr. Warnock noticed among his 45 pellagrous patients in 1897 at the Cairo Lunatic Asylum, wrist drop, paraplegia, general tremors, ataxy, epileptic seizures, retention of urine and herpes zoster. (Annual Report.)

slept at all at night. He became no better in hospital and lost ten pounds weight.

Mental Symptoms.

In Italy 10 per cent. of the pellagrous drift into lunatic asylums, but the proportion of insane in Egypt is so far smaller. Among the early symptoms are great mental depression, thinking is an effort and the patient becomes gradually excitable, stupid, and morose. A settled gloom is present on the faces of most of those admitted, and they have lost all power of smiling and laughing, and refuse even to try to do so. In advanced cases, there are deep, transverse furrows of melancholy across their foreheads, and their eyes are quite wanting in expression.

Among 166 notes, I find that 39 per cent. on entering the hospital were thought to be of normal mental condition; 24 per cent. were sad and stupid, and had to be roused to take an intelligent interest in anything beyond food and sleep; 31 per cent. were distinctly melancholic, discontented, insistent craving for food and cigarettes at all times, hungry eaters of extra food, yet often emaciating, 3 per cent. had early secondary dementia, with melancholia, loss of memory, and dirty habits.

These figures show that mental changes are very common when searched for, and sometimes are sufficiently serious to compel one to send the patient to the lunatic asylum.

I have notes of the 66 patients who were distinctly melancholic or demented on admission:—16 were cured, 30 left better, 18 left with no improvement,[1] and 2 died. One man, aged 30 on admission, was very resistive, with delusions of persecution and possession, refusing food because milk dried up his body, weeping much when talking, and refusing to show his tongue. Eventually the possessing devil told him he might show his tongue to me only under the protection of a sheet.

The delusions, such as poisoning, burning, possession, persecution, and refusal of food, hallucinations of taste, and attempted suicide by drowning, are all insane renderings of the subjective symptoms of pellagra. Dr. Warnock, who has been much interested in pellagrous lunacy since his arrival in Cairo, classifies his 45 cases as 15 melancholic, 5 maniacal, 8 mania of persecution, and 17 secondary dementia.

Diagnosis.

This now seems to me easy, but beginners must eliminate sunburn, dirt, leprosy, and psoriasis, while considering an eruption, and psychologists must think of other causes of melancholia and dementia. The presence of denuded tongue, altered knee-jerk and tenderness or pressure near the dorsal vertebra, will help to decide doubtful cases. In pellagra there is a history of a fairly exclusive diet of maize, and more or less skin eruption recurring at the beginning of the year, besides signs of general poverty and peasant life.

Duration of illness before entry.

In 162 cases the average time that the individual stated he had been ill was two years. In only 36 cases had the illness lasted less than one year, and in nine patients it had been of five years' duration or more before the man came to hospital. Speaking generally, the fellah does not make up his mind to journey to Cairo for treatment until pellagra has become chronic, and he is too exhausted by that and other troubles to earn his bread.

Stay in hospital.

Among 173 patients the average number of days in hospital was thirty-six. Two men only remained seven days before they were passed on to the asylum; three others were only four, seven, and sixteen days in the wards before they died. Four of the weak-minded melancholics were kept in hospital for as long as four months to see the utmost good which energetic treatment could produce. One gained twenty pounds weight, and learnt to laugh and talk, but all the results were disappointing.

Concurrent Diseases.

Besides the constant presence of anchylostomata, my notes mention bilharzia of bladder, 45 times in 1897; bilharzia of rectum, 24; favus of head, 7; ascarides, 2; rhabdonema, 1; oxyurides, many times; strongylus subtilis, 3; bronchitis, 6; malarial liver and spleen, 19; general tuberculosis, 2; mitral disease, 2; subacute rheumatism, 2; chronic kidney, 4; syphilis, many times; and two cases of suppurative cellulitis of leg, one of which caused death. Entozoa and malaria, succeeded by chronic pellagra, produce a condition within a measurable distance of death.

Prognosis and Result.

Pellagra may last ten years or more. The oldest case I have so far seen was one of twenty years' duration, though I have seen deaths after five years. Chronic diarrhœa, absence of patellar reflex, obstinate sleeplessness, and melancholia, are all very bad signs.

This is the result of my cases during the three years 1895-6-7:—

	Cured.	Relieved.	Not relieved.	Died.	Remaining.	Total.
Pellagra and Anchylostomiasis ...	50	184	18	8	23	283
Pellagra, Anchylost. and Bilharziosis...	19	99	3	9	24	154
Total	69	283	21	17	47	437

This gives a general death rate of 4·3 per cent., if we deduct the 47 cases remaining in hospital, being 3 per cent. only for the first category, and 6·9 per cent. for the patients attacked by all the three diseases. The extra presence of bilharzia disease therefore doubles the danger of the unfortunate victim.

Pathology and Post-mortem Appearances.

The great feature of fatal cases is general emaciation, with consequent brown atrophy of heart, fatty degeneration and atrophy of liver and spleen, and sometimes also of kidneys. The intestines show atrophy of muscular coats, and some hyperæmia, but this may be modified in the small intestine by the ravages of the anchylostoma and, perhaps, other entozoa, if the thy-

[1] This includes those sent to Asylum.

mol cure has not been practised. In bilharzia cases that worm will be found in the portal blood, the liver will show bilharzia changes, and the colon and rectum will probably have papillomatous growths. Dysentery is occasionally the final messenger of death, and in that case specific ulcers will be present, but I doubt if ulcers are caused by pellagra alone.[1] The skin shows chiefly atrophy and a deposit of pigment. There is also usually pigment in the cortical cells of the brain, in the heart muscle, and in the liver cells and spleen. The brains I have seen show no evident naked eye changes.

Tuczek[2] has carefully analysed eight autopsies at which he assisted in Italy. His most important results refer to the spinal cord, where he found symmetrical sclerosis of the posterior columns, and specially of Goll's columns, also in the pyramidal tract, but he found the posterior roots intact.

The spinal cords which I have seen show no naked eye changes, and though some have been preserved for future examination, I cannot now pretend to verify Tuczek's conclusions.

In 1897 I had seven pellagra deaths, and in two of them no autopsy could be made. Of the remaining five, two died of tubercle. Before death, patients remain for a few days in a condition of great exhaustion and stupor, which the Italians have named "tifo pellagroso."

Treatment.

The native is generally taken by his friends to a neighbouring wise man or wise woman, who is sometimes also the village barber and vaccinator. Any painful part of his body, such as his head, epigastrium, or back is burnt with red-hot irons. If that does no good, setons are tried, and a journey is made to a mosque, or sheikh's tomb, to pray for recovery. Last of all, he applies for relief to the Government hospital, or is brought there by the police, starving and homeless, and unable to answer questions intelligently.

My hospital treatment does not vary much. I believe rest is the most important thing. Ordinary mixed diet is given, unless diarrhœa be present, but I have adopted in practice the theory of some of my Italian teachers, that even good maize is bad for those poisoned by diseased maize. Therefore, pellagra patients are debarred from this article of the hospital diet, and with this rule they are quite content. If necessary, they are given extra bread, for which many of them clamour, and for loss of weight cod liver oil is added. Lately, I have given fresh bone marrow in tabloids to many of the worst cases, with a surprising gain in weight. The skin is rubbed daily with zinc ointment, partly to assist desquamation and partly as a mild stimulant, and the patients have a tepid Turkish bath once a week. All tonics are useful, especially strychnia, for the advanced and prematurely old cases. But as there is no specific drug treatment for pellagra, and every individual is an early or late case of anchylostomiasis, the thymol and iron cure is obviously indicated, and the pellagra and anæmia are benefited at the same time. All but the worst cases get, therefore, 8 grains of sulphate of iron three times a day. They get plenty of fresh air, for the windows are never shut, and twice a day they go on to balconies or into the garden. Diarrhœa requires careful dieting with peptonized foods, and drugs, of which opium is one of the most useful, if kidney disease is not present. The natives, unfortunately, believe that milk acts as a laxative. Weak-minded patients will want, in addition to the above, salt baths (much used at Milan), massage, and electricity. Excited cases want surfeeding and sedatives to insure sleep.

Prevention.

The Egyptian Government should now imitate that of Italy, which, besides teaching cleanliness and hygiene among the affected peasants, has for some years had soup kitchens and retreats for the poor, and has also encouraged the people to sow better maize, to dry it more thoroughly, and to keep it as dry as possible.

[1] Scheube gives ulcers as a p.m. sign. "Die Krankheiten der Warmen länder," Jena, 1896, p. 245.

[2] "Klinische und Anatomische studien über die Pellagra," Berlin, 1893.

BLACKWATER FEVER.

By L. Westenra Sambon, M.D. (Naples).
London.

The opening up of the Dark Continent as a new field of enterprise for the white man brings very forcibly before us the question of blackwater fever. This dreaded disease, so prevalent throughout tropical Africa, is causing appalling mortality among the pioneers of civilisation and the missionaries of Christianity.

Of late years there has been much discussion on blackwater fever, but the almost universal opinion is still in favour of the old idea that it is merely an aggravated manifestation of the ordinary malarial paroxysm.

I do not pretend to state definitely what blackwater fever is, but I believe we have a large body of fact to show what it is not; and, surely, the explosion of errors and misconceptions is of no small importance in scientific investigation.

Before entering on the discussion of this disease, it is necessary to note that authors have often confounded hæmoglobinuria with hæmaturia, a very different condition, which is distinguished by the presence of blood corpuscles in the urine, and may be due to injury or to some toxic substance, or be symptomatic of disease of the urinary tract, such as calculus, cancer, nephritis, or of some general disease such as purpura or scurvy.

Of the many names given to the disease under consideration, I have chosen that of "blackwater fever" (Febris melanurica) because it involves no preconceived idea as to its pathogenesis, and has the further merit of being the one most widely used in medical literature and common language. We should never commit ourselves to names expressing opinions which may turn out to be erroneous, and would therefore entail a frequent and confusing change of nomenclature. The name blackwater fever simply denotes

the most striking and characteristic symptom of the disease—the presence of dark pigment in the urine.

Causes of Melanuria in General.

In studying the blackwater fever of Africa and other tropical countries we naturally inquire into all such diseases and conditions as produce melanuria. Thus in several parts of Europe and in North America we find a disease of comparatively rare occurrence which resembles the blackwater fever of tropical regions in the most striking manner. Authors have called it paroxysmal hæmoglobinuria, and, being unable to agree on its etiology, have ascribed it to cold, that *deus ex machinâ* of most maladies. Then there is the so-called infantile hæmoglobinuria, or epidemic hæmoglobinuria, an outbreak of which was described by Winckel as it occurred in the wards of a lying-in hospital at Dresden in the spring of 1879. It is probably no other than paroxysmal hæmoglobinuria. Further, we know that the absorption of certain poisons will cause melanuria. Among such are carbolic acid, arseniuretted hydrogen, chlorate of potash and others. Transfusion of blood may also cause melanuria, especially when the blood of an animal of one species is introduced into the circulation of an animal of another species, the red corpuscles of the transfused blood being rapidly destroyed during their passage through the blood vessels of the animal into the circulation of which they are received.

Of paroxysmal hæmoglobinuria I shall speak presently. As to toxic melanuria we know very little. All I can say is that the symptoms induced by some drugs—potassium chlorate, for instance—resemble a paroxysm of blackwater fever, just as acute arsenical poisoning may resemble an attack of cholera.

Theories of Blackwater Fever.

The ideas now generally prevalent on blackwater fever are:

(1) That it is an unusually severe paroxysm of the ordinary malarial fever.

(2) That it is a peculiar condition caused by the administration of quinine in malarious patients.

It is these erroneous conceptions that I desire to disprove in this paper.

Natural History of the Disease.

I will first give a short sketch of the disease, and call attention to some very important points in its natural history, which have hitherto been neglected.

Geographical Distribution.—This, one of the most important elements in the study of a disease, is unfortunately, in the case of blackwater fever, for various reasons, most indefinite. The disease was first described in the small island of Nossi-Bé, off the north-west coast of Madagascar. It is prevalent throughout tropical Africa, especially along its great water-ways. It is found all along the West Coast from the Senegal to the Coanza, but especially on the Congo and on the deltas of the Niger and Gambia rivers. On the East Coast it is also widely spread, especially along the Zambesi and on the shores of Lake Nyassa. In America it extends over the southern States of the Union, especially in Florida, Georgia, and the Mississippi bottoms. It is generally prevalent in the plains of Venezuela and in the island of Cuba. It has appeared now and again in Java and New Guinea. Several cases have been reported from India, Assam, and Cochin-China. In Europe its occurrence has been established for Sicily, Sardinia, the Roman Campagna, and the Greek archipelago.

Endemic Prevalence.—Although blackwater fever has a wide distribution, it is limited in its endemicity to low grounds and lands that are occasionally overflowed with water. Several authors have reported its occurrence at high elevations, but we must not forget that in blackwater fever, as in malarial fevers and other diseases, relapses may occur at long intervals and far from the place in which the disease was contracted. Moreover, the swamps at the foot of mountains are the very habitat of blackwater fever, and we know also that the disease may have a long period of incubation.

Seasonal Prevalence.—In the Southern States of the American Union blackwater fever is reported to be especially frequent in late summer and in autumn. On the West Coast of Africa it prevails after the rainy season is over, when the atmosphere is hot and moist. It is therefore apparent that, like many other diseases, it requires peculiar conditions of heat and moisture. Relapses of the disease may occur at any season.

Epidemics.—Blackwater fever at times assumes an epidemic form. It may not be seen for years in a district, and then numbers of cases may occur within a short time. It was epidemic among the labourers employed in making the canal through the Isthmus of Corinth. In 1885 there was an outbreak of the disease in Castiadas (Sardinia), when twenty were attacked in one prison. Of late years blackwater fever has become far more prevalent in some regions, while in others it seems to have been only recently introduced.

Generally follows Malaria.—It is of the highest importance to note that blackwater fever is seldom a primary disease. Authors, from all parts, are unanimous in stating that the liability to infection is constantly associated with the occurrence of malaria. On the West Coast it has been observed that the disease seldom comes on within the first year of residence. Another fact worthy of notice is that strangers are far more liable to the disease than the natives of the place.

Symptoms.

General Course.—The onset of blackwater fever is usually abrupt. A violent rigor invariably ushers in the attack, while the temperature rapidly rises to 103° or 104° F., and may later reach 106°. In most cases it only lasts a few hours, and then, with profuse sweating, falls to normal. The patient complains of pain in his head, back, and loins; he is excessively thirsty, and experiences a sensation of distress about his epigastrium. From the outset there is profuse bilious vomiting and sometimes diarrhœa; the scleræ and the skin over the entire surface of the body become of a deep yellow colour, which is characteristic; but the most striking symptom is that of the urine, which becomes of a dark red colour, and some-

times almost black. The patient at first may show great mental anxiety and apprehension, but soon passes into a state of prostration. In severe cases the breathing becomes very laboured and deep owing to the increasing poorness of the blood. Sometimes the urine becomes exceedingly scanty or entirely suppressed; the patient falls into a state of collapse and dies. The duration of the attack varies greatly; it usually lasts from four to six days. In severe forms death may ensue on the second or third day. The attack may consist of two, three, or more paroxysms, corresponding to successive broods of the parasite. If the patient recover he is liable to relapse. The interval between the relapses may be considerable in duration; weeks, months, nay, even years may elapse. Relapses are usually milder than first attacks. Re-infection may take place.

Urine.—The urine in blackwater fever is generally strongly acid. Its specific gravity varies inversely to the amount passed; it is usually above normal. On shaking it produces a greenish-yellow foam. On standing it deposits a thick reddish-brown sediment, which is chiefly composed of an amorphous yellow pigment, aggregated into rounded masses or forming regular casts of the renal tubules. By gradually raising the urine to the boiling temperature a reddish-brown coagulum is formed, which consists mostly of cell globulin. Occasionally a very few corpuscles may be detected in the sediment as the result of renal congestion, but, as a rule, they are totally absent. Of course, nephritis may be associated with blackwater fever, and then hæmaturia may complicate or follow hæmoglobinuria and the urine will contain blood corpuscles and serum albumen. Formerly the colour of the urine was attributed to the presence of bile, but now we know that bile is either entirely absent from the urine, or is present in insignificant amount. The spectroscopical examination exhibits the two absorption bands of oxyhæmoglobin.

Blood.—If the blood be examined during an attack of blackwater fever, the red corpuscles are found to be undergoing rapid destruction; their number may fall in less than twenty-four hours to a third of the normal. At a certain moment of the paroxysm there does not seem to be one sound corpuscle in the patient's body. But the destruction, however intense, seems to be almost immediately replaced by a rapid regeneration, then blood corpuscles of every size, nucleated corpuscles, and large quantities of blood plates will be found under the microscope. At the same time will be witnessed a marked leucocytosis, which begins with the paroxysm and persists for a certain length of time thereafter; it is in every way similar to that which meets the invasion of the spirillum Obermeiri in relapsing fever. If freshly drawn blood be allowed to stand the serum will be found to present a very dark red colour, indicating the presence of much colouring matter in solution.

Pathology.

Parasitology.—The parasitology of blackwater fever is far from being settled. Several authors claim to have found the parasites of æstivo-autumnal fever, and more especially crescentic bodies. This fact is considered by the majority as a final confirmation of the idea that the disease is malaria. F. Plehn described a separate variety of parasite, much smaller and ovoid in form, which does not become pigmented. Fisch also described a distinct organism; and Manson, though believing melanuric fever to be a malarial disease, is of opinion that its organism is specifically different from the malaria parasites already identified. Albert Plehn, who studied forty-three cases of melanuric fever in Cameroon, considers it possible that the disease may be caused by a separate type of organism, but he could detect no morphological difference between the parasites which he found in these cases and those occurring in æstivo-autumnal fever. Yersin found a cocco-bacillus in the urine of his patients, and claimed it to be the cause of melanuric fever. Breaudat found the same organism in several cases, and recognised it to be bacillus coli communis.

Morbid Anatomy.—The *post-mortem* appearances in cases of blackwater fever are usually complicated with those produced by malaria. When these are eliminated and when death occurs early during an attack, and the necropsy is made soon after death, the following special conditions are found: The liver, spleen, and kidneys are enlarged. The liver is of a yellowish-brown colour on section; it shows marked cloudy swelling of the cells, which contain granules of yellow pigment. The gall bladder always contains much bile, and is sometimes greatly distended. The spleen is congested, its capsule is thick, and the parenchyma is black and soft. Masses of dark yellow pigment are to be found both between and within the cells. The kidneys show intense hyperæmia. The cortical substance on section is found to be of a pale yellowish colour, the pyramids showing brown streaks, especially towards their apices. There is marked infiltration of leucocytes in the interstitial tissue, and the epithelium of the secreting tubules shows cloudy swelling. The convoluted tubules are filled with granular pigment, which is partly free in their lumen, partly contained within the lining cells. This pigment, together with shed epithelium, forms regular casts within the straight tubules.

Is Melanuria a Symptom of Malaria?

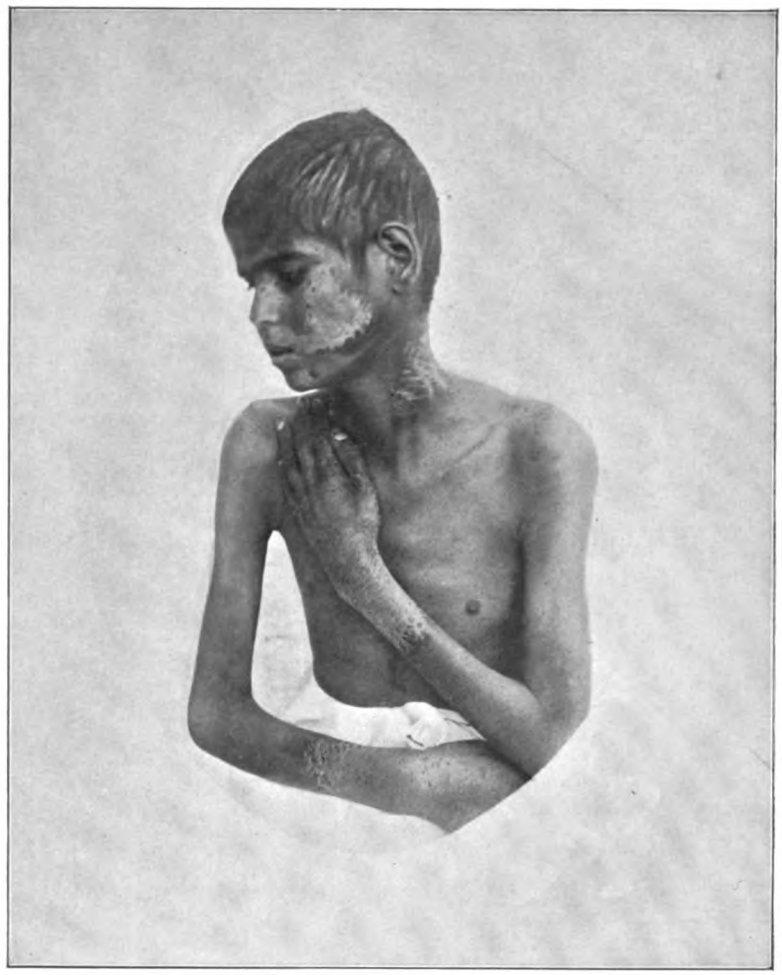
Now let us examine on what grounds authors have believed blackwater fever to be a manifestation of malaria.

(1) The disease is met with in malarious countries.

(2) It is usually preceded by attacks of the ordinary malarial fever.

(3) The malarial parasite or pigmented cells have been found in the blood and organs of those suffering from the disease.

At first sight these facts seem to be very strong evidence; but they soon crumble on accurate investigation. Laveran still holds the opinion that the various types of fevers and their peculiar organisms are but the modifications of one type under varying conditions; but opinion is now almost universally agreed on the plurality of types. With which type of malaria is blackwater fever associated? Possibly with any; but in regions in which it is most prevalent we find it associated with that form of malaria which has been called summer-autumn fever. On the West Coast of Africa, where so many cases of blackwater fever occur, summer-autumn fever is exceedingly prevalent, while



PELLAGRA IN EGYPT.

By F. M. SANDWITH, M.D., M.R.C.P.

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tertian and quartan fevers seem to be totally absent. On the other hand, we find blackwater fever to be unknown in regions highly infected by the summer-autumn fever. Thus we begin by noting the fact that the geographical distribution of blackwater fever does not coincide with that of malaria. Then, again, the relative prevalence of blackwater fever and malaria varies greatly in the different regions in which both diseases occur; and moreover, though the same conditions of soil, temperature, and moisture seem to favour both diseases, their seasonal maxima bear no relation to each other.

The summer-autumn type of malaria is characterised by the fact that its paroxysms do not present the chill which is so striking a feature to tertian and quartan fevers, but blackwater fever always begins with a severe chill; it is, therefore, in this respect, in marked contrast to the form of malaria of which it is supposed to be a paroxysm. Another distinguishing feature is the marked leucocytosis of blackwater fever, which is absent in uncomplicated malaria.

Even in the seventeenth century Torti of Modena succeeded in distinguishing malarious fevers from the chaos of diseases with which they were confounded by the fact that they yielded to quinine. The discovery of the parasites of malarial fevers has made us forget the diagnostic value of quinine, although its specific action in the treatment of these diseases has been fully confirmed. In blackwater fever, on the contrary, quinine is not only useless, but possibly injurious.

In all forms of malaria there is certainly an extensive destruction of blood corpuscles. In severe æstivo-autumnal infection, the destruction in a single paroxysm may amount to a third of the entire number, and yet no hæmoglobinuria occurs. But in malaria we know that the parasite transforms the corpuscular hæmoglobin into black pigment, which is only very gradually eliminated. In blackwater fever a very different process is at work. No black pigment is formed, but the infected red corpuscles are rapidly disintegrated and eliminated, possibly in the same way as the corpuscles of transfused blood from an animal of different species.

To explain why blackwater fever occurs in some of the regions in which malaria prevails and not in others the usual and comfortable explanation of "special climatic influences" has been adopted, but in the same locality we usually find blackwater fever and tropical malaria prevailing at the same time, and besides, the geographical distribution of blackwater fever shows that the disease can prevail under the most varied climatic conditions. Then again, it is well known that people who have suffered from blackwater fever in tropical Africa may have relapses of the disease in England many months after their return, probably in midwinter.

As already stated, the parasite of summer-autumn fever has been at times demonstrated in patients suffering from blackwater fever, but this is no proof that it is the cause of the disease. We know that melanuric fever usually attacks those suffering from æstivo-autumnal fever, and we also know that the crescentic bodies associated with this malady may be found in the peripheral circulation even months after the patient has apparently entirely recovered. No wonder, therefore, that these non-febrile forms should be found in the subject of blackwater fever. But observations have demonstrated that, when present, the parasites of malignant tertian diminish considerably in number during the hæmoglobinuric paroxysm, and besides, in quite a number of cases, no such parasites could be demonstrated.

Some have asserted that blackwater fever is always preceded by several paroxysms of the ordinary malarial disease, but we know of many cases now in which melanuric fever was contracted within a few weeks of landing, and without any previous attack of malaria.

Finding that malaria alone could not account for blackwater fever, and that climatic conditions were not sufficient to explain its occurrence, authors looked for some other agent, and quinine was incriminated.

Is it Quinine Poisoning?

The idea that quinine, the great specific remedy for malaria, could produce blackwater fever in patients suffering from ordinary summer-autumn fever originated with Tomaselli, of Catania. It is a misinterpretation of the fact that the administration of quinine, even in small doses, may excite a paroxysm of blackwater fever in a patient who has previously contracted the disease and still bears it in a latent form. But quinine, even in large doses, never produces blackwater fever in healthy people, nor in malarial patients who have not previously suffered from the disease. Besides, blackwater fever has frequently spontaneously appeared in patients who had never taken quinine, and moreover several authors claimed to have cured blackwater fever with large doses of quinine. Then the idea of a special idiosyncrasy was put forward to account for this peculiar action of quinine in malarious patients, but the explanation is not admissible, seeing that in regions in which blackwater fever prevails quinine may excite a paroxysm in quite a number of those who have contracted the disease, but in the regions from which blackwater fever is absent quinine never produces it in anyone. In examining the hundreds of cases of so-called quinine hæmoglobinuria we find invariably this to be their history: A patient suffered repeated attacks of malaria, which were satisfactorily treated by quinine in the usual doses. One day, suddenly, instead of the usual paroxysm, blackwater fever manifests itself and the administration of quinine seems to aggravate its symptoms and excite new paroxysms whenever administered. Later the blackwater fever disappears and the administration of quinine, even in large doses, never causes it again. But quinine is not the only agent that will excite a paroxysm of blackwater fever; just as often relapses are caused by fatigue and chill. Thus we come to the conclusion that blackwater fever is not malignant tertian infection modified by special climatic conditions or the influence of quinine. What is it, then?

Blackwater Fever and Texas Fever.

I will say first of all that I believe it to be very closely related to the paroxysmal hæmoglobinuria of temperate regions. I do not mean to say by this that all cases of hæmoglobinuria should be considered as cases of blackwater fever, but that such are most cases

of so-called paroxysmal hæmoglobinuria. Several authors have pointed to the fact that the two diseases are clinically indistinguishable, and Dr. Wheaton proved that in their morbid anatomy they are also identical. The only difference is that while blackwater fever has a very high mortality, paroxysmal hæmoglobinuria seldom proves fatal; but we must not forget that the mortality of blackwater fever varies greatly in different regions, and I believe its deadliness on the West Coast of Africa and in other regions can be fully explained by the fact that in those regions it attacks the wrecks of severe tropical malaria.

The recognition that blackwater fever is the same as paroxysmal hæmoglobinuria gives us no clue to its pathogenesis, because practically nothing is known regarding the latter.

Far more light is shed on our subject by the hæmoglobinuria of cattle which has been so thoroughly and successfully investigated by Babes in Roumania, Smith and Kilborne in the United States, Krogius and O. von Hellens in Finland, Sanfelice and Loi in Sardinia, Celli and Dionisi in the Roman Campagna, and Koch in East Africa. These authors have found that the hæmoglobinuria of cattle is caused by an hæmatozoon which Babes classed between bacteria and protozoa.

Starcovici believes that the hæmoglobinuria of cattle is not caused everywhere by the same parasite, but by various types which differ somewhat morphologically from the pyrosoma bigeminum described by Smith. However this may be, it is reasonable to conjecture that the parasite of blackwater fever, if not identical with that which causes Texas fever, very probably belongs to the same group. The descriptions which F. Plehn and others give of the parasite found in blackwater fever seem to confirm my view.

The hæmoglobinuria of cattle resembles very closely that of man. It prevails under the same climatic and seasonal conditions; it gives rise to the same symptoms, and its *post-mortem* appearances are identical.

EXPERIMENTS WITH DR. UNNA'S NEW METHOD OF TREATING LEPROSY.

By Dr. J. A. Voorthuis (*Apeldoorn*).

Whilst practising in Deli East-coast of Sumatra in 1894 several cases of Leprosy amongst Europeans came under my notice. I then heard of Dr. Unna's new method of treatment and found occasion to ask this eminent dermatologist to enable me to make some experiments with his method. As in Deli Lepra, often in very desperate forms, was at the time rather frequent amongst chinese coolies on tobacco plantations I had a good opportunity for testing this new method.

Dr. Unna by extensive pathological investigations had found out that one substance exists in the human body provided with complete immunity against the Lepra-bacillus, namely the muscle substance. He therefore thought it of great importance to treat Lepers with a preparation principally consisting of muscle substance. He found a very suitable solution in *Valentine's meat juice*. In the beginning he applied this fluid by subcutaneous injections; only a marked influence on the tubercles was observed but they had no practical result.

He then resolved to introduce the substance directly in the blood by intraveinous injections, by which proceeding the effect on the leprous tubercles proved more distinct.

The meat juice is a completely sterile preparation. After opening the bottle it can for a very long time be preserved, provided the substance be covered with a small layer of a 1 per cent. oil solution of chloretum hydrargyricum.

For diluting the meat juice we used an artificial serum consisting of chloret. natr. 0.5 natr. phosphor. bas. 0.1 aq. destill. 100 which serum can easily be sterilised by boiling just before use.

The skin being carefully cleansed, one of the bigger veins at the elbow is made to swell by finger-pressure and the fine needle of a Pravaz-syringe being easily introduced, the fluid is very slowly injected.

We commenced with 0.2 c.c. M. meatjuice diluted with the same quantity of the artificial serum. The sequel of such an injection is that the patient directly afterwards feels a distressing sense of dizziness, gets chilly and has shivering fits, frequently accompanied by chattering of the teeth.

The temperature of the body is raised to 39° or 40° C., which elevation lasts for an hour or two, where after the former state of health returns. Every second day an injection was repeated and the quantity of meatjuice injected, increased; within a few weeks this quantity could be raised to 1 c.c. M. at once.

I could never observe the slightest disturbance of the general state of health in the four cases I treated, which were all chinese coolies on tobacco plantations. One of them acquired Beri-Beri in the course of the treatment and we were obliged to stop. He died a few weeks afterwards.

In the other cases distinct amelioration of the general state was obvious; in all cases a direct effect of the medication on the tubercles was easily noticed; this effect only appeared after four or five injections had been made. The nodules and tubercles became reddened and swollen; some of them weakened and a few were within a few weeks totally resorbed; others again could be emptied by incision. On the contrary there were also tubercles which only became reddened and swollen without afterwards being weakened or resorbed. Although no durable cure can be mentioned, the remarkable amelioration of the general condition of the patients and the active influence on the nodules and tubercles stated in my four cases, the complete account of which will shortly appear in a paper edited by Dr. Broes Van Dort, seem to indicate urgently the continuation of this experiment and the study of the influence of other muscle extracts on Leprosy.

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BLACKWATER FEVER.

BLACKWATER fever is undoubtedly the greatest scourge to the European in tropical Africa. Alarmed at its increasing spread and appalling mortality, the Foreign Missions Committee of the Church of Scotland, the Livingstonia Committee of the Free Church of Scotland, and the African Lakes Trading Corporation, sent a deputation to the Foreign Office to urge the desirability of sending properly qualified men to investigate the nature, cause, prevention and cure of this disease. Again at the Edinburgh meeting of the British Medical Association, when Dr. Sambon had finished reading his paper on blackwater fever, which we publish in another column, a resolution was passed that the Council should bring pressure to bear upon the Government to take the matter in hand. Government, however, had already appointed a commission consisting of Dr. Daniels, Dr. Christopher, and Dr. Stephens, which will proceed shortly to British Central Africa. Meanwhile it will not be amiss to discuss the conflicting literature which has recently appeared on blackwater fever. The most important papers are (1) Dr. Manson's Chapter on Hæmoglobinuria in his admirable "Manual of Tropical Diseases," 1898; (2) Dr. A. Woldert's article on "The Use of Quinine in Hæmoglobinuria" in the *New York Medical News*, April, 1898; (3) Dr. Sambon's paper on "Blackwater Fever," read at the Edinburgh meeting of the British Medical Association; (4) Dr. Koch's paper on "Blackwater Fever," in his "Reise Berichte," Berlin, 1898; (5) Dr. Connolly's article on "African Hæmoglobinuric Fever," *British Medical Journal*, September 24, 1898; (6) Dr. Crosse's paper on "The Treatment of Blackwater Fever," read at a meeting of the Pupils' Physical Society, Guy's Hospital.

Dr. Manson in his manual, and Dr. Sambon in his paper, give concise but excellent descriptions of the symptoms, pathology, and morbid anatomy of the disease, and very little could be added to their lucid accounts. Mr. Connolly, in his article, distinguishes three varieties: the sthenic, the insidious, and the pernicious, but we see no reason for such a division. The disease may appear insidious when following or superseding a paroxysm of tropical malaria as often happens, and of course its attacks may vary greatly in intensity, but such conditions do not warrant the distinction that Mr. Connolly suggests.

The geographical distribution of the disease in Africa and in other countries, as far as it is known, is carefully stated in Dr. Sambon's paper, and the writer points to the fact that the disease, though widely distributed, is limited in its endemicity to low grounds, and especially to the valleys of rivers that overflow their banks. This at first sight seems a mistake, because people have been attacked very frequently at high altitudes, but Dr. Sambon explains this apparent contradiction by stating that the swampy regions at the foot of mountains are especially prolific of the disease, that the period of incubation may be exceedingly long, and that relapses frequently occur in malaria and other diseases.

Several authors have stated that blackwater fever generally appears ten or twelve days after residence in the low coast districts. The natives of the coast seem to enjoy a certain immunity from blackwater fever, but those of the interior contract it easily when they come to the coast.

Authors have frequently attributed blackwater fever to chill, "that *Deus ex machinâ* of most maladies," as Dr. Sambon calls it. Dr. Crosse, in his recent address, states the case of a doctor who was attacked after walking out in the rain the night before, and explains his own attack by the very opposite condition, because he says it was caused by exposure of his back to the sunshine a few hours before. Such statements are manifestly illogical, not only because of their conflicting nature, but because we know already enough of the life history of protozoa to understand that a much longer period of incubation is necessary.

But is blackwater fever a protozoal disease? For a long time physicians in tropical countries believed it to be merely a more severe form of the ordinary malaria, and the presence of malarial parasites in patients of blackwater fever seemed to confirm this view. Lately several authors have advanced the idea that, though belonging to the same group of diseases, its parasite must be of a distinct variety. Such is the opinion of Dr. Manson, and such is also the belief of F. Plehn, who distinctly describes a peculiar variety. Prout (*Lancet*, August 1, 1891), Fisch (*Deutsche Med. Ztg.*, Berlin, 1896), Woldert (*New York Medical Journal*, 1895), and others, also describe a special parasite, but notwithstanding that all their descriptions agree on its being a small, ovoid, and unpigmented parasite, yet they are mostly of so uncertain a nature as to leave us in doubt whether the observer saw parasites or simply vacuoles. Dr. Crosse goes back to the old belief and states that blackwater fever is not a separate disease, but only a pernicious form of the ordinary malaria. Dr. Sambon, on the other hand, adduces a strong body of evidence to prove that it is not malignant tertian infection or any other identified form of malaria. The disease has a peculiar geographical distribution quite distinct from that of tropical malaria, its seasonal maxima do not coincide with those of malaria; it often prevails epidemically, it differs widely in symptoms, pathology, and morbid anatomy. Dr. Sambon therefore endorses strongly the opinion expressed by Dr. Manson and F. Plehn, but he goes even further, and basing himself on the somewhat uncertain bacteriological observations and on the strong clinical analogy, states blackwater fever to be due to a parasite similar, if not identical, with that which causes Texas fever in cattle.

Several years ago Professor Tomaselli, of Catania, expressed the idea that blackwater fever was nothing more than quinine poisoning; this absurd idea spread like wild-fire. The Greek physicians took it up in good faith, and so did other medical men in Europe and America. Some physicians in Sicily and Sardinia, noticing that the disease prevailed in the season in which beans ripen, attributed it to this vegetable, but their idea did not become popular. Several authors showed how untenable was the idea that quinine was the causative agent of blackwater fever, and no one better than Dr. Woldert in the *Medical News*. F. Plehn, who had at first accepted this etiology, judiciously discarded it in his later writings. The last flash of the quinine theory seemed to be the recent new edition of Tomaselli's own work, when Koch, speaking before the German Colonial Association in Berlin announced, among other equally wonderful discoveries, that he had found blackwater fever to be nothing more or less than quinine poisoning, and he added, "I must, unfortunately, refrain from telling you all my reasons for this assertion, but you may rely upon it that they are absolutely conclusive." This oraculous sentence ran on the wings of the press in a moment all the world over.

Dr. Moffat, Principal Medical Officer, Uganda Protectorate, in a letter to the *British Medical Journal* (September 24, 1898) states that patients in East Africa will no longer take quinine, and fears that Koch's assertion will cause much harm.

The question of quinine, not as a cause, but as a remedy, in blackwater fever, has also been greatly discussed, especially by American authors, and extreme views have been disputed with equal vehemence. What we gather from a conscientious perusal of the literature on the subject, is that quinine has no specific action on the germ of blackwater fever. While several authors claim to

have cured the disease with large doses of the drug, others state that it did not alter its course, and others that it aggravated the symptoms and probably helped in bringing on a fatal termination. The truth may be this :—

(1) That in many cases the quinine was in no way responsible for the improvement or aggravation.

(2) That when blackwater fever developed, while the patient was suffering from a spell of malarial activity, the quinine destroyed the malarial infection, and thereby improved the patient's condition.

(3) That in severe cases of blackwater fever the quinine acted as an additional poison, and helped to weaken organic resistance.

Certainly we cannot deny the fact that quinine, even in small doses, can excite a paroxysm of blackwater fever in patients who have previously contracted its germ, and it is this fact, repeatedly observed, that gave rise to the erroneous conception, formulated a quarter of a century ago by Tomaselli, that the disease might be due to quinine poisoning.

PLAGUE IN INDIA.

THE change of front of the Bombay Government in their dealing with plague was to have been expected. It was impossible for the high pressure of the last two years to be kept up permanently, and even if it had been possible, it is doubtful whether it would have been wise. The time for stamping out an epidemic disease is not when that disease has gained a strong hold on the community. Any attempt in that direction is but following the lead of Canute when he commanded the sea to go no further, and the result is likely to be as equally hopeless. The error of the Bombay Government, with regard to plague, lies not in want of energy when once it began its stamping-out policy, but in its persistent indifference when plague was first suspected, and when repressive action was likely to have been useful. The history of the plague of 1896, in Bombay, is the old story of a controllable smouldering fire having been allowed to burst into a conflagration, which no efforts could extinguish. The mistake has undoubtedly been atoned for, but at a cost financially, political, and socially, which is likely to leave its traces for a long time to come.

The policy of land quarantine will be given up, and all the harassments attendant on this system will be swept away. It seems ludicrous that such a system should have prevailed in Bombay, when the whole of the Bombay presidency was infected. But because this system is not required, and is harmful in an infected locality, we trust the Authorities will not fall into the error of an opposite character and remove the restrictions in healthy provinces against the infected Presidency. Quarantine, like everything else, has to be used with discretion, and because it is useless in an infected locality, it does not follow that the same can be said of it in a healthy province.

SANITARY PROGRESS IN INDIA.

SIR JOSEPH FAYRER in his able address to the Sanitary Congress held in Birmingham last month, gave some striking illustrations of the results of sanitary work in India during the last half century. The ordinary death-rate of the British soldier, which had stood from the years 1800 to 1830 at 84·6 per 1,000, and from 1830 to 1856 at 56·70 per 1,000, has now fallen to less than 15 per 1000. The death-rate of the Native Army has fallen to below 10 per 1,000, and the death-rate of prisoners to 27 per 1,000. As regards the vast civil population, epidemics, famine, and long-established modes of living which obstruct improvement, had not only to be dealt with, but also ignorance, prejudice, and religious scruples which tend to a dogged resistance of all measures taken for the amelioration of their condition. The people persist in their ancestral modes of life, resist all changes, and object to the well-meant measures devised by Authority for saving them from plague, cholera and pestilence. Notwithstanding these obstacles, Sir Joseph shows that there has been a gradual amelioration in the health of the population.

TROPICAL PATIENTS AND STRATHPEFFER SPA.

ANGLO-INDIANS and others returning to England suffering from the effects of the climatic conditions to which they have been long exposed, have not infrequently to visit some of the Continental spas for the purpose of regaining their health. Certain places have, in this respect, enjoyed a special reputation, and not undeservedly. But now that more attention is being paid to the medicinal properties of the spas in Britain, it may be asked, Are there not health resorts in Britain which would equally prove beneficial to the patient from the tropics? If so, there are doubtless many who would infinitely prefer a visit to a health resort in the British Isles than to one on the Continent, and particularly is this likely to be the case when the time before returning to the tropics is somewhat limited.

For very many years past the highlands of Scotland have been considered to be one of the best regions where patients, debilitated by malaria and other diseases contracted in warm climates, are likely to recover their strength. The purity of the atmosphere and the bracing nature of the climate are important factors in these cases, which factors were as much appreciated by Sir Andrew Clark, who sent many of his patients there, as by Sir Ranald Martin, who first brought the highlands into notice. By the advice of these two medical men the highlands of Scotland have become a favourite resort for those in search of health and amusement. The Northern country, moreover, possesses an additional advantage, which is not so well known. In Ross-shire there are the mineral springs of the Strathpeffer Spa which are excellently suited for tropical patients, and which, year by year, attract an increasing number of visitors. Situated in a valley and surrounded by beautiful scenery, comprising hill and dale, woodland and water, pines, heather and bracken, it is an excellent place for a spa. There are two kinds of springs, sulphur and chalybeate, varying in strength. The weakest sulphur spring contains a small quantity of iron, while the two strongest, each of them containing the largest quantity of sulphuretted hydrogen in any known spring in Great Britain, are free of iron.

An excellent pump-room has been erected, where the waters from the several wells are given out. Adjoining the pump-room are very extensive buildings, in which the most modern arrangements have been made for sulphur baths, douche baths, pine baths, peat or mud baths, Russian baths, and hot air baths. The attendance is good. There is a pavilion with reading room and a band which discourses music at the hours when the waters are drunk, which are from 7 to 9 in the morning, and again from 12 to 2 in the day. At these hours the pump room and neighbourhood presents an animated scene, friends and acquaintances meeting and discussing their ailments or the topics of the day while they drink the waters. There are plenty of interesting excursions near, so that three weeks or a month can be pleasantly spent at the spa without *ennui*. For golfers there is a good golf course. For the gouty, the anæmic, and the neurasthenic, the waters are specially useful, and we believe that Strathpeffer is bound to become a favourite spa for tropical patients.

Articles for Discussion.

BATHS AND BATHING IN TROPICAL COUNTRIES.

THE daily bath has come to be an essential factor in the life of European residents in the tropics. So much is this the case that to them is ascribed the re-introduction of the habit to Western Europe. In Grecian and in Roman epochs the bath was a feature of their civilisation, but with the decline of their prescience bodily cleanliness, as represented by bathing, also disappeared. It is to the return of old Orientals to Europe, and more especially to Britain, that we owe the resuscitation of the daily bath in modern times. So universal has become the custom that most people in Britain, of all but the lowest classes, are ashamed to say that they do not take a daily bath. In the tropics the bath becomes a necessity for purposes of cleanliness alone. The

skin, of Europeans at all events, is kept in so constantly active a condition that, to neglect its use places the individual at a disadvantage, as far as personal health is concerned. There can be no doubt of the efficacy, as regards health, in thus freeing the skin of its own secretion. But the taking of baths has far-reaching effects besides that of cleanliness, and it is on this point I would invite discussion. Many persons in tropical climates take cold baths, whilst others indulge in lukewarm, tepid, warm, or hot baths. If cold water bathing is beneficial from its therapeutic effects, then are warm baths deleterious, or *vice versâ*? If warm baths are advisable, are cold baths harmful? For the purposes of curtailing the scope of the argument, it will be convenient to divide baths into those at the temperature of the body and those considerably below as represented by cold, lukewarm and tepid baths. Moreover, it is only morning or evening baths that are being considered—"tubs," as they are called—not sea-bathing, Turkish baths, &c., &c.

Bathing in water considerably below the temperature of the body causes a physiological effect which is summed up in the well-worn expression that by it "the blood is driven from the skin to the internal organs." In other words, a considerable tonic action is induced. The blood seeks refuge in the central organs of the body, and the great abdominal blood receptacles, the liver, spleen and portal system generally, are engorged. This is the consequence of immersion in cold water in temperate climates, or in any climate; but in the tropics the abdominal organs are always in a more or less plethoric state, and the consequence of cold water bathing will be to critically engorge the liver, distend the spleen and portal vessels, and flush the kidney to such an extent that the urine may be charged with albumen. In proportion as the water of the bath approaches the body temperature, so are these effects lessened; but when the temperature of the water is only 20° below that of the body, some such sequelæ are met with. The hot bath, on the other hand, determines the blood to the skin, and instead of congestion we have relief of internal organs from a fulness of blood.

It will be naturally asked, what do the natives in tropical countries do as regards baths? Shortly, it may be stated they do not take baths in the sense that they get *into* water either hot or cold. The natives of India, for the most part, elect to stand erect, and pour water over themselves.

Douching and rubbing the skin dry with a cloth is their method of freeing the skin from its excretion. The Mahommedans, in conformity with religious tenets, make a palaver of washing frequently, but it is a mere "schoolboy dip" of the finger tips and the mouth. The Chinese wash more thoroughly, if not so frequently. They usually raise a lather of coarse native soap, and after washing, rub themselves dry with a cloth.

These baths are mostly, if not wholly, for cleanliness' sake, and except perhaps the Hindoo douche, have no therapeutic action tonic, depressant or otherwise. Immersion, however, in water, for say five minutes, is quite another matter, and it is this form which Europeans in the tropics affect. They do not follow the native custom of bathing; the bath, or morning "tub," is quite an innovation, and its wholesomeness or otherwise is the question before us. To sit in cold water for, say, five minutes, on first getting out of bed in a tropical country, must be attended by very decided physical effects. The shock is relatively greater than in temperate countries, the abstraction of heat during the process can be but ill afforded, and the depression of bodily temperature is difficult to recover from. Neither the circulatory nor respiratory system respond to the stimulus, and the digestive organs may be congested to a dangerous extent. Especially is this the case with the feeble and in those "getting on" in years. But even in the case of young men who have resided, say, five years consecutively in the tropics, the inevitable anæmia, the hepatic congestion, and the cardiac and muscular relaxation are such, that but few can take a morning bath in water at a temperature below 60° without exposing themselves to a depression of temperature which may end in chill, and lay the door open to any one of the many ailments ever ready to seize upon those who are, from whatever cause, in any way below the standard of health.

The opponents of the warm or hot baths declare them to be relaxing, and betoken effeminacy. This is neither true in its practical nor therapeutic aspects. After fatigue, nothing is more stimulating or refreshing than a hot bath. The good effect is probably that heat is restored to the exhausted economy, and vigour thereby re-induced. Cold water would still further tend to depress the temperature consequent upon fatigue, and instead of stimulating, still further depress, the system. There can be, therefore, but little doubt that a bath of cold water taken in the tropics after violent exertion, such as a tennis match, a long bout at racquets, or on returning from a long march or shooting expedition, is contra-indicated, and positively harmful. But for the early morning "tub," what are the indications? The question does not seem difficult to answer; why lose the body heat by immersing in cold water, unless tonic effects are assured. The whole question is, Are the tonic and re-actionary effects sufficient compensation for the loss of heat? The loss of heat to the body is not a mere expression or sensation, it is the induction of a physiological state which requires consideration, as reaction is not established in the tropics with the readiness it is in temperate countries. One has to weigh the two items in the scale and consider, Can the body afford this loss of heat? In my opinion, in the case of tropical residents of over, say, five years' standing, the body cannot afford it. For the young, vigorous and newly-arrived youth in the tropics, the cold bath may be indulged in with impunity, and it may be with benefit; but as years lapse, the tropical resident calls for bath water warmer, and still warmer, until he finds he has best health only when the water of his bath is not below the temperature of his body.

It is a curious fact that Canadians, even when the temperature of the air is considerably below zero, do not employ hot water to even wash in. They ascribe to immersion of the face and hands in hot water many local ailments, and similarly deny themselves frequent general hot water baths, experience having taught them its pernicious effects. This is a curious outcome of practical experience, that British folk in regions at times arctic in their temperature, should have come to avoid washing in hot water, and that the same race in the tropics have worked out the lesson that bathing in hot water is alone salutary.

J. C.

Recent Literature on Tropical Medicine.

OPHTHALMOLOGY IN ITS TROPICAL BEARINGS.

Fundus Changes in Leprosy.—Dr. Trantas, ophthalmic surgeon to the Greek Hospital, Constantinople, in a paper just published in the *Recueil d'Ophthalmologie*, records a case of ocular leprosy of great interest. A woman of 45, mother of two healthy children, presented herself at the Greek Hospital for an affection of the left eye of a year's standing. On examining the eye, the following conditions were found: diffuse infiltration of the lower third of the cornea: several tiny superficial ulcerations in the same situation: complete insensibility of the entire cornea: and an iritis with numerous posterior synechiæ. In the right eye, of which the patient made no complaint, insensibility of the cornea was also found, but not quite complete, and a commencing keratitis similar to that in the left. These lesions caused Dr. Trantas to suspect leprosy, and on making a careful general examination, unmistakable evidences of the disease were found. Under treatment the keratitis in the right eye improved so much that it became possible to obtain a clear view of the fundus, with the result that a distinct choroido-retinitis was discovered, the following being the main features of the ophthalmoscopic picture:—A large patch of choroidal pigment a little above and outside the macula: several tiny round spots in the immediate neighbourhood of the macula, believed to be retinal exudates: some pigmentary disturbance on the temporal side of the macula: towards the periphery one or two yellow patches of fresh choroiditis. Central vision was almost normal in this eye, but the field was contracted. The urine was normal, and there was no history of syphilis or other diathesis save leprosy.

Dr. Trantas draws special attention to (1) the insensibility of the cornea: (2) the relation between the degree of insensibility and the amount of infiltration: (3) the possibility of ocular lesions existing without attracting the attention of the patient—hence the utility of ophthalmoscopic examination in all cases of leprosy: (4) the close resemblance of leprous lesions of the fundus to analogous syphilitic lesions; and (5) the existence of a distinct leprous choroido-retinitis.

The main interest of the paper centres in the last point, as I believe I am correct in stating that this is the first case in which distinct fundus changes of leprous origin have been described. Lopez (*Arch. f. Augenh.* xxii., 1891) examined forty-five cases of ocular leprosy without finding ophthalmic lesions: Berger (*Les maladies des yeux dans leurs rapports avec la pathologie générale*) states that retinal lesions have never been found by the ophthalmoscope: while Panas, in his monograph, makes no allusion to fundus changes.

Experimental Quinine Amaurosis.—Dr. W. A. Holden, continuing the work initiated some years ago by Brunner, Barabaschew, and De Schweinitz, has recently published the results of his experiments in the induction of quinine amaurosis in dogs (*Knapp's Archives*). The dogs were killed after a period varying from two hours to seven weeks after the administration of toxic doses of quinine, and the

eyes, optic nerves, brains and spinal cords examined—by the Nissl methylene blue method for cell-changes, and the Marchi osmic acid method for nerve-fibre changes. Retinas examined three days after several toxic doses revealed vacuolation, paleness, and breaking down of the cell-body in a few ganglion cells; while large globules of a myelin-like character were found in the nerve-fibre layer. On the ninth and seventeenth days more ganglion-cells were affected, and more myelin-globules present. On the seventeenth day the first changes in the optic-nerve were noticed—breaking down of the medullary sheaths of many fibres. On the forty-second and forty-seventh days the ganglion-cell layer and nerve-fibre layer of the retina were almost gone, leaving large cavities; many of the fibres of the optic-nerve were broken down, and the degeneration of the nerve could be traced up to the termination of its fibres in the external geniculate body and pulvinar. No signs of degeneration elsewhere in the brain, or in the spinal cord, could be detected.

These experiments are of great value, as they demonstrate clearly that the changes in the optic nerves, tracts and chiasms described by previous observers do not constitute the primary lesion, but are secondary to changes in the retinæ. Apparently the first effect of a toxic dose of quinine is to cause spasm of the retinal vessels: then as a result of the lessened blood-supply the less resistant elements of the inner layers—the ganglion-cells and nerve-fibres—break down; an ascending degeneration of the optic-nerve fibres follows the retinal changes.

Azotate of Cocain.—Nitrate of silver—valuable as it is in the treatment of trachoma and certain conjunctival catarrhs common in tropical practice—has one serious drawback, viz., the severe pain, sometimes lasting for hours, which so frequently follows its application in solid form or strong solution. Probably many, like the writer, have endeavoured to obviate this by the instillation of hydrochlorate of cocain, and have been equally surprised and disappointed at the negative results; the fact being that an insoluble chloride of silver is formed which renders the cocain useless. Dr. Neuschuler (*Rec. d'Ophtal.*), assistant in Galezowski's clinic, has tried azotate of cocain (previously employed by Saafeld to anæsthetise the urethra) for this purpose with complete success; this salt forms no precipitate with nitrate of silver and a few drops of a 1 in 20 solution instilled in the conjunctival sac before the application of the caustic renders the little operation painless.

Sub-Retinal Cysticercus.—A. Lutkevitch (Wratch. No. 12, Moscow) describes three cases of sub-retinal cysticercus observed in the Moscow Eye Clinic. In two of the cases extraction was attempted, without success; the third case was not operated on. Cases of ocular cysticercus are rare in Russia; only thirty-seven cases in all have been recorded.

M. T. Yarr,
Major, R.A.M.C.

UNITED STATES OF AMERICA.

The Pernicious Malarial Fever of the Tropics.

In the *Medical News* for July, 1898, Dr. J. E. Stubbert, of New York, after remarking upon the small death-rate from tropical diseases during the making of the Nicaragua Canal, owing to the medical officers in charge being allowed a free hand, deals with the treatment of malarial fever as follows:—

"Remittent malarial fevers are far more common than the intermittent form; they are more intense in their onset than in temperate zones and require more energetic and prompt treatment, but if intelligently handled, in the vast majority of cases, will respond readily to treatment. The most important point for our surgeons to insist upon is the early recognition and treatment of malarial fevers in their various forms. In the Nicaragua Canal hospitals the average duration of cases of remittent malarial fever was five and one-half days; had these patients been neglected in the incipiency of their attacks they would probably have been ill twelve or fourteen days and many deaths would have occurred.

"In the tropics a physician does not depend upon small doses of calomel and quinine, nor can he wait for the action of a cholagogue before administering the last-named drug. An active sudorific, followed at half-hour intervals by three 10-grain doses of quinine, which in turn should be followed within two or three hours by a large dose of calomel, rhubarb, and soda, will nip in the bud most cases of remittent fever, so that a daily dose of from 20 to 30 grains of quinine for three days thereafter will be followed by convalescence.

"Far more dangerous to the patient than yellow fever, is pernicious malarial fever, especially in the form known as malarial hematuria. This undoubtedly is the disease known as the 'black-water' fever in Africa, and a careful perusal of its history will, I believe, show that the administration of quinine, contrary to the report of Koch on his recent trip to Africa, has nothing to do with its production. Those familiar with malarial hematuria in our Southern States and Central America know that generally the hematuria antedates administration of the drug, and disappears when the patient is thoroughly cinchonized. If the milder forms of remittent fever are early recognized and promptly treated, the pernicious forms will not be encountered, for it seldom develops as a primary form; there is always a history of recurring, and generally neglected, malarial attacks, and seldom does it develop in a new arrival in the tropics.

"The statement that 'malaria, even untreated, in any single attack is a self-limited disease, and successive attacks are milder and milder,' unfortunately will not prove true in the West Indies. On the contrary, immigrants from northern climes if properly treated during their first, second, and third attacks of malarial fever, show greater resisting power during the second, and seldom have more than the three attacks. If neglected, each successive illness becomes more severe, and finally they develop the dreaded pernicious type.

"Pernicious malaria occurs only in those persons who have been a long time subjected to slow malarial infection, without the proper administration of quinine. So closely does the appearance of the patient resemble that of one with yellow fever that superficial observers have named the disease 'high-land yellow fever.' The distressing symptoms are hematuria, intense nausea and black vomit, extreme thirst, frequently repeated chills of a congestive character, and, at times, sinking turns amounting almost to syncope. The remaining symptoms are those common to other cases of remittent fever; like yellow fever it has an algid, icteric, and hæmorrhagic variety. In the algid form there is a marked difference between the rectal and sublingual temperature, amounting at times to 3° F. The treatment of this disease must again be different from that of the mild forms of malarial fever. Promptness, energy, and unremitting watchfulness are desiderata without which the patient will be lost.

"Much discussion has lately taken place as to the advisability of administering quinine in these conditions, but I believe the preponderance of evidence to be in favour of immediately thoroughly cinchonizing the patient by hypodermatic doses of quinine and establishing free diuresis and catharsis. In a majority of these patients thorough cinchonism will be evidenced by fall of temperature and amelioration of other symptoms.

"Prophylaxis against tropical malarial fever in our camps should consist of changing the clothes before retiring at night (unfortunately often impossible), avoidance of constipation, and a daily ration of quinine, to which whisky should be added when the subject has been exposed to rain. Sleeping in shacks or in tents with the sides open, and as far as possible selecting for camps high sites exposed to wind and sunshine should be encouraged. Hammocks should be swung at least three feet from the ground; in more permanent locations beds constructed of split limbs of trees are better than hammocks. Mosquito nettings should always be used, and as the mosquito of the tropics is often smaller than his fellow of the North, a very fine mesh is indispensable. Add to these precautions a careful, systematic medical supervision of water supplies, kitchens and diet, a daily inspection of each company by a medical officer, and finally, rejection at recruiting-stations of men with positive histories of malarial infection, and the invaliding home of all patients who respond only temporarily to treatment and this bogey of the tropics will be robbed of half its terrors."

Autopsy Finding in Yellow Fever.

The *Boston Medical and Surgical Journal*, September 15, 1898, in referring to an article which appears in the *Medical News* for September 3, by Dr. Eugene Wasdin, of Marine Service, on "The Value of the Findings on Subjects Dead from Suspected Yellow Fever," observes that the matter is of much importance in a campaign such as we have just gone through, and it is altogether desirable to render mistakes as few as possible.

Dr. Wasdin says: "The yellow fever cadaver has assuredly a most characteristic appearance. All subjects dead from the disease bear a close resemblance to each other; indeed it would not be difficult to make a diagnosis, other facts being favourable thereto, from the cadaver alone. The body is usually quite rigid, this change in the muscles coming on early and persisting. The colour is invariably more or less intensely yellow, and is due to a mixed hepatogenous and hematogenous jaundice. The entire skin is tinted, the scalp usually presenting a startling contrast to the parted hair, and the whites of the eyes are yellow, the change taking place in the conjunctivæ early in the disease, the scleræ becoming tinted later. This yellow tint is always contrasted with the deep purple discolorations from hypostasis, which quickly appear in the skin of the dependent portions of the body after death, and which frequently appear before. It is at the edges of these hypostatic areas that the mixture of biliary and blood jaundice is particularly noticed as a muddy, thick, greyish-yellow tone. Hypostasis is common to all cadavers, but particularly is it prominent in this disease. It is not confined to the lower portions of the body, as the buttocks, loins and shoulders, but invades the neck, chest, ears and face; the genitals, as a rule, and the finger and toe-nails are of a deep purple colour. This discoloration occurs quickly, and is prominent within an hour after death. The pupils are usually dilated, the tongue foul, and the gums bloody; the anterior nares are caked with blood. This is a picture of a typical yellow fever cadaver, and the appearances may be accentuated or softened, but the characteristic *ensemble* will be present in all."

The resemblance externally of the yellow fever cadaver to that of a person who has died of malarial fever is often very striking. In such cases dependence must be placed upon conditions found in the internal organs. There is in yellow fever a general yellow tinting on section, and the spleen in uncomplicated cases is of normal size and appearance. The kidneys are always congested and swollen, and on section show fatty areas, particularly about the base of the pyramids within the cortex. The liver, the author has usually found normal in size and showing marked fatty changes. The vessels of the stomach walls are congested, the mucosa thickened and presenting numerous patches of extravasation and shallow erosions. The lungs are congested and yellow; the pleuræ and pericardium present numerous ecchymoses, as well as the heart muscle.

Histologically the fatty changes are best seen in the liver and kidneys. The liver cells are more granular, stain more faintly and contain numerous fat droplets; those of the kidneys lining the tubules, especially of the cortex, are also filled with drops of fat. The so frequent importance of an immediate diagnosis often renders the demonstration of the finer histological changes impossible for practical purposes.

The writer discusses briefly the bacteriology of the disease, and some personal experiments in which he was able in many cases to isolate a bacillus which gave rise on inoculation to characteristic lesions. This experience has been that of many other observers. These organisms elaborate a toxin which exerts a characteristic and powerful influence upon the vasomotor system, as evidenced in the intense engorgement of the vessels, the gaseous, toneless pulse, and the full diastolic heart, prior to and *post-mortem;* also, in that rapid metamorphosis of the albuminoids of the highly organised cells of the liver and kidneys. The intention is not to contrast these organisms, but to accentuate the fact that there is no constant sign at these autopsies by which we can name the particular one which has produced the disease.

Dr. Wasdin quotes an interesting case occurring recently at Key West, in which, after careful examination, he concurred in the conclusion that the patient had died of yellow fever, but was later convinced that it was simply an intoxication from auto-infection with some member of the colon group of organisms.

"Finally," he says, "I would suggest that typical autopsic findings should always be regarded with suspicion, and demand immediate investigation of their cause. Should there be, or not, any local centre of auto-infection, there should be an investigation of the immediately preceding history of the patient, and

should there prove to be evidence of exposure to infection of yellow fever, and should the clinical charts and history present the evidences of the disease which we have learned to recognise, then and only then can the information gained *post-mortem* be of decisive value to the diagnostician."

Diagnosis of Yellow Fever.

In *The Medical News* of July, 1898, Dr. T. E. Stubbert, of New York, Lecturer on Tropical Diseases, University Bellevue Hospital Medical College, states that yellow fever is most likely to be confounded with pernicious malaria, and presents the differentiation of the two diseases in tabulated form :—

YELLOW FEVER.	PERNICIOUS MALARIA.
Headache bilateral - frontal, and post-orbital.	Headache generally unilateral-frontal, and temporal.
Temperature and pulse divergent; temperature rarely higher than 104° F.	Temperature and pulse correlative; temperature generally 105° to 107° F.
Albumin present in large quantities early in the disease.	Albumin rarely present.
Quinine has no effect on the progress of the disease.	Quinine has a specific effect if given hypodermically and early.
Stage of remission on third or fourth day.	Remission not present.
Attacks new arrivals.	Generally history of chronic malarial infection.
Always history of exposure to infection.	No history of exposure to infection.
Black vomit appears on third or fourth day.	Black vomit appears within thirty-six hours.
Hematuria very rarely present.	Hematuria a marked symptom.
Liver unchanged.	Liver enlarged and tender.

Dr. Stubbert goes on to say that dengue or break-bone fever is a disease having its habitat in warm climates, and has been confounded with mild cases of yellow fever. The reason is not apparent, for their clinical histories are distinct. The following table shows a comparison of the more prominent symptoms common to both diseases :—

YELLOW FEVER.	DENGUE FEVER.
Cephalgia and nephralgia are the characteristic pains, and are constant.	Pain most severe in joints and muscles, and is *paroxysmal.*
Pulse and temperature divergent.	Pulse and temperature correlative.
The slowing of the pulse begins early in the disease.	The slowing of the pulse occurs late in the disease.
Congestion of face early in the disease. No edema.	Rash on face, followed quickly by edema.
Albuminuria.	Albuminuria absent.
Icterus.	Icterus absent.
Black vomit.	Black vomit absent.
No eruption.	Polymorphous eruption, followed by desquamation.

Leprous Ulcer of the Lip.

Dr. Henry L. Wagner, of San Francisco, reports in the *Boston Medical and Surgical Journal*, September 8th, 1898, a case of Leprous Ulcer of the Lip. This condition is extremely rare, only one case having been reported. At the International Congress it was stated that the primary seat of this contagious disease was in the mucous membrane of the upper air passages. Dr. Wagner's case was that of a middle-aged white man who had lived in China. He had a broken-down ulcer with nodules on the lower lip, that had existed for some months with but little pain. It appeared to be syphilitic, but the nose and throat showed nothing abnormal. A portion of the ulcer submitted to examination showed the presence of lepra bacilli and innumerable suppurative bacilli. The lepra bacilli were lying about among the staphylococcus and streptococcus of suppuration. These bacilli were found partly within and partly without the cells. The patient did not return for further treatment. This case showed the lepra tuberosa form of the disease, and also demonstrated a channel by which it could be communicated.

ITALY.

G. Bisio. *La Campagna della R. Nave "Amerigo Vespucci" nel mare delle Autille.* "Annali di Medicina Navale," Aug., 1898.

The author publishes a few cursory notes on the sanitary conditions, medical institutions and most prevalent diseases of the West Indies, which he took during a cruise of the Royal training ship *Amerigo Vespucci* in January, 1898.

A. Bignami. *Sulla questione della malaria congenita.* Supplements to the Policlinico, No. 30).

Dr. Bignami states, on the strength of his own observations and of those of Bastianelli, Caccini, and Tayer, that no parasites and no malarious morbid conditions are to be found in the fœtus of a mother that suffered from an attack of pernicious malaria or from a number of relapses during pregnancy. Cases of congenital malaria have been published by authors, but they must be held as doubtful until the presence of parasites in the new born child of a malarious mother will have been thoroughly ascertained.

The author is inclined to believe that the passage of malarial parasites from mother to fœtus does not occur because the infected cells do not extravasate as easily as the normal, and in support of this idea he states that in cases of inflammation with hæmorrhagic exudation he never found plasmodia in the extruded corpuscles. But possibly the non-infection is due to the fact that the fœtus' blood is not appropriate to their development. In favour of this hypothesis he states that young cells in the adult are not attacked, and that the nucleated corpuscles of cattle are not infected by the pyrosoma bigeminum as Smith, Kilborne and Dionisi have demonstrated in Texas fever.

Dr. Bignami points to another fact which, if not constant, is certainly not rare. It is that though the mother be in a condition of grave anæmia from a late malarious infection, the fœtus may present a healthy condition of the blood, and none of the signs of degeneration in the liver which may be strikingly marked in the mother. This leads the author to express, with due reserve, the idea that the destruction of the red cells may not be caused by parasitic toxines as stated by most writers, but by the plasmodia themselves. If a toxine were the cause of anæmia it would certainly pass into the fœtal circulation, and induce in it a like condition.

Id. *Febbri tropicali e febbri estivo-autunnali dei climi temperati.* (Annali di Medicina Navale, Aug., 1898.)

Dr. Bignami discusses in this paper Koch's statements on malaria, and proves that the German Professor has made no new discovery. The type of fever described by Koch was described eight years ago by Marchiafava and Bignami in Rome, and its parasite in patients who contracted the disease in the Roman Campagna, the east coast of Africa, or the swamps of Texas was fully described by Marchiafava, Bignami, Bastianelli, Dionisi, Gravitz, Dock, and others. Dr. Bignami illustrates his paper with temperature curves—some taken by Koch in East Africa, others by himself in Rome, and shows that they are exactly alike. As to the parasite, Koch's description coincides in every way with those given by authors in other parts of the world. Koch's tropical malaria is nothing more nor less than the disease that

the Italian physicians named summer-autumn fever. As to blackwater fever, Dr. Bignami contends Professor Koch's views. Blackwater fever is not merely quinine poisoning, but a malarial disease.

G. Rem-Picci. *Sulle lesioni renali nella infezione malarica.* (Il Policlinico, Vol. v., M., 1898.)

The author states that opinion is conflicting on the connection between kidney disease and malaria. Some authors deny any connection whatsoever, others greatly exaggerate its frequency; and certainly, says the writer, it is difficult to establish in every case whether the renal trouble be a direct consequence of the malarial infection or not.

Dr. Rem-Picci studied the subject in the Hospital of St. Spirito in Rome from 1891 to 1898. He bases his statements on 80 cases, in which he found renal complications. Considering that only in one year (1895) 6,184 malarial patients were treated in that hospital it is evident that renal complications are not frequent in malaria.

The author, examining methodically the urine in a large number of patients, was able in many cases to detect the symptoms of renal mischief from their earliest manifestation, and he ascribed to malaria all such cases as became apparent during malarial infection, and after the patient had been in hospital for some days. The renal complications found by the author in such patients cover the whole field of renal diseases. He found slight albuminuria, acute Bright's disease, chronic parenchymatous nephritis, chronic interstitial nephritis, amyloid disease, and hæmoglobinuria.

We will not deny a transient albuminuria as a possible complication, but we cannot agree with the author in considering the various renal diseases as features of the malarial infection, simply because they happen to arise or develop during an attack of malaria. We all know how frequently one infection will give opportunity to others by enfeebling resistance, and how often a latent disease has suddenly developed under the influence of an acute fever. Malaria is one of those diseases which most frequently lead to other infirmities. The frequency of pneumonia, of typhoid, of dysentery, of blackwater fever in malarious patients is well known, and these diseases were at one time considered to be of malarious nature when complicating malaria. Renal diseases are no more malarial than pneumonia or typhoid.

A Society for the Study of Malaria.

Guistino Fortunato and Leopoldo Franchetti, both members of the Italian Parliament, have proposed the foundation of a Society for the study of malaria. In an interesting circular published widely through the press to obtain public support, they state that malaria in Italy prevents the cultivation of 2,000,000 ettari of land, prevails more or less in 63 provinces, 2,823 municipalities, infects every year 2,000,000 of inhabitants and kills 15,000 people. A grant of £20 will procure the title of foundation fellow. Ordinary fellows will have to pay thirty shillings yearly. The Society will encourage in every possible way studies on malaria, and will publish a yearly report of its work.

F. Rho. *Malattie predominanti nei Paesi caldi e temperati.* Torino, 1897.

We have received a copy of this admirable treatise on tropical diseases, and we shall review it extensively in a future issue.

GREECE.

Methyl-blue in Malaria.

J. P. Cardamatis, of Athens, reports that he has administered methyl-blue in 275 cases of malarial fever during the last three years, and has been amazed at its prompt and effective action. He considers its therapeutic value in malarial fever far superior to that of quinine, and "cannot praise it enough." Its efficacy was especially noticeable in persons so saturated with quinine that they had ceased to react, and in those in which quinine was counter-indicated. The results were undeniably successful in 93 per cent. of the total cases of acute and chronic malarial fever treated, and it even confers immunity; only 15 per cent. of the persons continuing to live in extremely malarial districts had relapses within a year. The result was negative in 7 per cent. The only inconveniences were the staining of the mouth and clothing when the liquid form was used (infants). He gives four tablets a day, one every two hours. The daily dose for adults is 10 to 12 grains; for older children, 8 grains; for younger children, 6 grains; infants, 1 to 2 grains. It is administered ten to fifteen days, and then suspended for two or three days and a purgative given, resuming for three or four, and then suspending for three to eight days, and resumed at intervals, ensuring a total of seventeen days in twenty-two for acute or chronic quotidian, in which the fever disappears in the first five days, sixteen days in forty-eight, when the fever reappears in the first five days, and twenty-eight in sixty days in the tertian and quaternian form.—*Deutsche Med. Woch.*, February 3.

SOUTH AFRICA.

An Epidemic of Ankylostomiasis in South Africa.

Dr. James Matthias gives in the *South African Medical Journal* the following account of an epidemic of ankylostomiasis among the underground European miners in the Kimberley and De Beers mines:—

"It was about ten months ago that the ova of the ankylostoma duodenale were first found in the fæces of a De Beers miner, and since I have seen them in 30 cases, besides which about 15 have been treated by other doctors for this disease. There can be no doubt but that a large number infected by this worm have not as yet been treated, their symptoms not being severe enough to incapacitate them from working. That a very large proportion of the miners who have worked for any considerable time, say six months, in the more frequented parts of the mine are affected is certain, and the opinion I have formed is that those whose work entails the soiling of their hands in the mud of the old tunnels, are without exception infected by the parasite. I think it can be shown that this epidemic of ankylostomiasis is not one of quite recent origin, but that it was prevalent in Kimberley at least as early as 1890, beyond which date our statistics do not go. It is quite likely that, did they do so, it would be shown that it was present soon after deep working was commenced in the mines. Thanks to the careful way in which the statistics of the De Beers Benefit Society have been kept, I have been able to get precise information as to the health history of my cases from as far back as 1890 to date, from which it is seen that one case has suffered, off and on, since 1890 from gastro-enteric troubles, and several others since 1891, 1892, and 1893. The names of these men only appear on the Benefit Society's books when they are absolutely unfit for work, and I know personally that many of them, while continuing at their work, were under treatment, at very short intervals, for disorders of the bowels and other complaints of the same character as those which are caused by ankylostomiasis; therefore I have no hesi-

tation in saying that the disease has been endemic here for the last seven or eight years. When these men formerly used to consult medical men, their explanation of their state of debility and anæmia almost always was, that they were suffering from the effects of the inhalation of the smoke of bad dynamite; indeed, this was so generally accepted a theory amongst them, that I was myself, as well as others, converted to it, which accounts for our not having made a correct diagnosis earlier. The details obtained from the Benefit Society's books plainly show that the effects, which this little worm produces on its host, are very serious, even though our miners are very well fed and naturally robust men. It incapacitates them for work, often for weeks and months together; many have left the country for from six months to a year at a time, only to return to the same condition within a few weeks of starting work; several I have seen in such a low condition that they could not walk without assistance, and seemed almost at the point of death; two miners died about a year ago whom I had attended, and from their symptoms I am now quite sure that they died from ankylostomiasis. From my own observations, therefore, I can but come to the conclusion that the effects of this parasite on the health are most pernicious. All the cases I have met with have been among European miners (I do not attend any of the Kafirs), who are employed underground, and a large majority of these were men who were platelayers, or timberers, and therefore worked in the older tunnels through which the trucks run, and which have been in use for years. The disease, if not entirely confined to the underground miners, is certainly far more common among them than among those employed on the surface. I have not come across a single employee working on the floors, who presents anything like the characteristic features of ankylostomiasis, nor have I succeeded in finding ova in the fæces of any of those employed above ground that I examined. The entire exemption from the disease of those who do not work in the mine itself, if it is the case, would be rather significant, as the dejecta of a couple of thousand men employed underground is daily, and with great care, spread out on the floors with the best blue ground from the mine; so the worm gets every chance of propagating itself, and if it does not do so on the surface, it must be that there are climatic or other influences there which are not favourable to its welfare.

"So far as I can learn, this is the first time that ankylostomiasis has been known to occur in South Africa in the form of a serious epidemic; but the probability is that the parasite is indigenous to this country, as it is now found in almost every locality within the temperate zones, and that, in this country, we may expect to have serious epidemics arising whenever and wherever the conditions necessary for its abundant multiplication are present, that is, where there is a favourable soil, moisture, and heat, and men to work in the ground and eat their food with unwashed hands. When I give it as my opinion that the ankylostoma is indigenous to this country, I do not mean that it is so to every locality in South Africa, but only to those where the conditions are favourable to it. For instance, it almost certainly is not so to this district, its presence here being due to introduction from some moister and warmer locality of South Africa. At the same time it is quite possible that the parasite was brought here from any far distant country, as we have miners who have worked in almost every country in the world. In our Kimberley mines all the conditions underground are favourable; the mud in the tunnels is always moist, the temperature in them averages 76° for eight months in the year and 70° during the others, and the white miners always eat one meal a day underground. On the contrary, in this district the ground on the surface, on account of the dryness of the atmosphere, is not probably a favourable soil for the increase of the parasite, so the disease is not likely to become a scourge to the inhabitants of the town or district of Kimberley. In the other mining centres, such as Johannesburg and Rhodesia, if the mines have sufficient moisture to make the ground in the tunnels muddy, the conditions will be as favourable to the ankylostomes, as in the mines here, and they will almost certainly be found there. One would expect that those who work in the gardens of Natal, and perhaps of Cape Town as well, would be likely to suffer from this disease.

"The symptoms which my patients have presented have been the same as are described by authorities as occurring in epidemics in other parts of the world, and are but what we would expect as the result of chronic internal hæmorrhage, combined with chronic intestinal irritation, namely, anæmia in all possible gradations, with resulting debility, shortness of breath, palpitation of the heart, &c., and pain in epigastrium on pressure, dyspepsia, not unfrequently diarrhœa, &c.

"There is no difficulty in making a correct diagnosis in a suspected case of this disease, as the fæces contain ova in such great numbers, and they are so easily seen and distinguished under the microscope. Indeed, I have only in one case failed to find ova in the first specimen put under the microscope, and since it has been known that ankylostomiasis was prevalent here, I have not examined the fæces of a suspected case without finding ova, his general aspect being so characteristic."

Reviews.

LECTURES ON THE MALARIAL FEVERS. By William Sydney Thayer, M.D., Associate Professor of Medicine in the Johns Hopkins University, Baltimore, U.S.A. London: Henry Kimpton, 1898. 326 pp.

This handsome volume contains nine lectures on the malarial fevers. The subject is handled in a comprehensive and masterly manner, and the book is one which all students of tropical medicine would do well to read carefully. In the first Chapter "The Pathogenic Agent of the Malarial Fevers" is dealt with from an historical standard; and it is shown that even a hundred years before the Christian era Varro assigned a parasitic origin to malarial fevers. The second lecture is devoted to the methods of examination of the blood, and to a description of the Hæmocytozoa of malaria. The student will find here an admirable guide to the methods of investigation, necessary for examination of the blood in malaria. Lecture III. deals chiefly with the conditions under which malaria prevails. The "Clinical Description of the Malarial Fevers" contained in Chapters IV. and V. is altogether classical, and supplies us with one, if not the best exposition of the kind in the

language. The temperature charts of the various fevers are standard types to go by, and should be carefully studied. Equally thorough are the subjects of the sequelæ and complications; the morbid anatomy in acute and chronic malarial infections discussed in Chapters VI. and VII. The last two Chapters are devoted to Phagocytosis and the Diagnosis, the Prognosis, Treatment and Prophylaxis of malarial fevers generally.

Charts of cases of malarial and other fevers, to the number of 19, are incorporated in the text, and, at the end of the volume, three excellent coloured plates, showing the blood cells and the various stages of development of the plasmodium malaria, are beautifully and exactly delineated.

As students of tropical medicine we thank Dr. Thayer for his valuable contribution, and have much pleasure in commending his "Lectures on the Malarial Fevers" as a work of a high order.

News and Notes.

THE QUEEN has appointed Sir William MacCormac, Bart., President of the Royal College of Surgeons, and Sir Francis Henry Laking, M.D., to be Knights Commanders of the Royal Victoria Order, and Mr. Alfred Downing Fripp and Fleet-Surgeon Alfred Gideon Delmege, M.D., to be Members of the Fourth Class of the same Order, in recognition of their services in connection with the recent accident met with by the Prince of Wales.

As evidence of the important part that members of the medical profession are playing in public life in Greater Britain, we note with pleasure that William Bisset Berry, M.D., a graduate of the University of Aberdeen, has been appointed Speaker of the new Parliament at the Cape of Good Hope.

WE are pleased to note that Calcutta is now officially declared free from plague. There has been no fresh case for ten days, and the last plague patient has left the hospital. We endorse the warning administered by the Lieutenant-Governor, Sir John Woodburn, that sanitary precautions against plague should be strictly adhered to and continued during the approach of the cold weather, when the possibility of a reappearance is to be entertained.

THE soldiers returning from Khartoum to Cairo seem to be suffering rather markedly from enteric. It is noticeable that whilst during the advance up the Nile to Omdurman the health of the army was exceptionally good, but since the crowning victory enteric seems to have claimed many victims. Possibly the strict discipline which was necessitated to reach Khartoum has been somewhat relaxed, and with the relaxation disease has followed.

IT has been arranged that the members of the Commission specially appointed to investigate malaria and blackwater fever in British Central Africa, as mentioned in our last issue, shall before proceeding to their destination, do some preliminary work in Rome and Calcutta. Dr. C. W. Daniels, of British Guiana, will visit Calcutta to study the work already done of the Indian Medical Service. Dr. J. W. W. Stephens, formerly Lawrence student in pathology and bacteriology at St. Bartholomew's Hospital, and Dr. R. S. Christopher, of University College, Liverpool, commence their investigations at Rome.

THE management of some of the hospitals in South and Western Australia seem in want of re-organisation. We have had the Adelaide Hospital disturbances before us for some time, and now several nurses have been dismissed from the Government Hospital in Coolgardie. The public, championed by the Mayor, are taking up the cause of the nurses.

A REUTER'S telegram, dated Simla, October 12, 1898, contains the following announcement:—

"The appointment has been sanctioned of a Special Commission, consisting of five members, for the purpose of making scientific inquiry regarding the plague. The Commission will investigate the origin of the various outbreaks and the manner in which the disease is spread, and will also consider the effect of curative serum and preventative inoculation. The Secretary of State for India appoints three scientists from home. Messrs. J. P. Sewett and A. Cumine, Indian civilians, have been nominated here as the two other members of the Commission."

THE LIVINGSTONIA MISSION.—Twenty-five years have passed away since David Livingstone died, near Lake Bangweolo. The Livingstonia Mission, advocated by Dr. Stewart, of Lovedale, Livingstone's fellow-traveller, was founded in 1874. The following year the *Ilala* steamed into Lake Nyasa, bearing a company of pioneer missionaries, headed by Dr. Laws, who, for a quarter of a century, has ably directed the Livingstonia Mission. The good work done in the Medical Department may be seen from the following summary of cases for 1896:—

STATION.	MEN.		WOMEN.		CHILDREN.		TOTAL.	
	MED.	SUR.	MED.	SUR.	MED.	SUR.	MED.	SUR.
Livingstonia ..	1148	2412	188	248	112	211	1448	2871
Ngoniland ..	157	113	175	217	645	283	977	613
Bandawe ..	..	..	..	..	..	..	6256	2325
Karonga* ..	..	..	..	..	..	..	7644	M. & S.

* For six months.

New Drugs, Instruments and Surgical Appliances.

THE "AMIRAL" SOAP.—A pamphlet issued by The "Amiral" Soap Syndicate on the Treatment of Obesity has been sent us, and with it numerous favourable opinions as to the merits of this method of reducing obesity. Lengthened experience can alone determine the efficacy of the Amiral Soap to dissolve surplus fat. The soap is prepared from "purified extract of amiral gall," and united with a basis of materials possessing "penetrating qualities." It is contended by this means that bile can be made to penetrate the skin as in the case of many other drugs. The method of em-

ploying the "cure" is at once convenient and simple. Twice a day the soap is to be rubbed on the part of election, for three or four minutes, by a piece of flannel or other convenient means, and the lather allowed to remain on the skin for three or four minutes longer. An immediate result is not claimed for the soap, but, after a fortnight's time, absorbent effects are to be expected. The soap, as prepared, readily forms a lather; it is in no way irritating or injurious, and being free of mineral compounds, it can be used by those endowed with even the most delicate constitutions. To the surgeon it will no doubt prove a useful adjunct to his armamentarium in the treatment of fatty tumours.

THE Thymo Tooth Paste, which has been brought under our notice, fulfils all the advantages claimed for it by its inventor. Being in the form of a paste, it is better suited for travellers and residents in Tropical countries than are dentrifice powders, which are now dry, now moist, and frequently rendered mouldy, owing to the alterations in the atmospheric state. The compressible tube in which the Thymo Tooth Paste is enclosed renders it capable of carriage in the portmanteau without the dread of breakage and the distribution of its contents over one's wearing apparel.

Mr. C. Baker's microscopes have been so well and so favourably before the public and the profession that it seems unnecessary to draw attention to them. We are pleased, however, to see that one of our most distinguished investigators in tropical scientific work, Surgeon-Major Ronald Ross, in Calcutta, has discarded microscopes of foreign manufacture, and done work of the highest order with the "Diagnostic Microscope," price £12 2s. 6d. complete, with powers ranging as high as $\frac{1}{12}$-inch oil immersion. In his catalogue Mr. C. Baker specifies microscopes of many varieties and of many prices, but for students of tropical medicine we can recommend the "Diagnostic Microscope" as being at once moderate in price, handy for clinical work and clear in its definition.

Correspondence.

THE CARE OF THE TEETH AT SEA.

To the Editors of "The Journal of Tropical Medicine."

DEAR SIRS,—Will you permit me, through your columns, to comment on the dental surgery practised by the surgeons attached to the Fleet of the Royal Navy, Her Majesty's Army, and more especially to the ocean-going steamers of the merchant service?

In the large merchant and passenger-carrying ships thousands of travellers voyage for weeks at a time without touching port, or, if they do, it is for the shortest possible time, just to embark passengers and mails. I think you will agree with me that where a passenger, say from London to China, takes a passage, for which he pays at a high rate, he naturally expects, and has a right to obtain, the correct surgical treatment that he may need during the time he is on board, but I venture to assert, and to prove if needs be, that in the matter of dental surgery, the treatment he gets from the surgeon on board is of the most meagre or radical kind. For instance, if he be suffering from odontalgia at the stage where it is changing into periosteal inflammation, the treatment he will get is, that carbolic acid will be placed in the tooth, or the tooth will be extracted, both treatments being in many conditions absolutely wrong.

I will mention a case recently under my notice. A lady consulted me on the eve of her departure for the Far East. She complained of pain in a lateral incisor. Finding on examination the nerve to be exposed, the treatment indicated was devitalization of the pulp performed by the application of an escharotic. But I was at once confronted with this difficulty: the dressing, after some days, should be removed, and in this case would have to be done by the surgeon on board, and the case carried on by him, so as to enable the patient to preserve the tooth in a healthy condition till she arrived at her destination, or at a place where skilled attention and treatment could be obtained, and so the case to be carried through in the ordinary conservative and almost painless way. Such initial treatment by me was impossible, as a ship's surgeon is practically ignorant of the work of nerve killing, and even if he were not, he is quite powerless, as he does not carry in his equipment the necessary instruments for removing the nerve after it is devitalized. Failing this correct treatment being carried on in such a case as this, periostitis may occur or a condition of collateral hyperæmia may be set up, difficult at times to subdue, and so from existing circumstances, being debarred from dealing with the case in the proper way, I was forced to immediately remove the exposed and live pulp. The exigencies of the situation compelled me to put the patient to a certain amount of pain, which I maintain is a position of things that should not be possible to occur, and could not, if the steamship companies arranged that the gentlemen whom they employ as surgeons were instructed to some extent in the practice of dental surgery before taking up their appointment, thus enabling them to receive a case from a dental surgeon in London and hand it over to another in the Colonies. This is simply one case, which I can multiply in my own practice, and no doubt could be added to by other dental surgeons.

As to the Royal Navy, the only dental instruments a staff surgeon or a fleet surgeon is equipped with are those he would use for extraction, and I assert with a sense of surprise and regret that among this set of instruments, as supplied by Maw, Son and Thompson, is the obsolete key, which if used now, and any injury to the maxillæ resulting therefrom, the operator would render himself liable to a charge of negligent dental practice. So much for the instruments. Now, for a moment, let me refer to the perfectly helpless condition of the patients who require dental aid on any of our war ships. The Fleets cruise for months together, sometimes touching at ports where there are no skilled dental surgeons, so they can get no help there, and so what happens is this: that these poor fellows, officers and men being all alike where pain is concerned, are *nolens volens*, compelled to lose their teeth or bear the pain, which is sometimes continuous. There is no need for me to point out to you, Sir, the conditions and dangers consequent on such a state of things.

With regard to the Army, the arguments are the same. In some parts of Her Majesty's dominions our men are a thousand miles from a large town, and in all their bodily ills they have to rely on the surgeon; and surely, under these circumstances, he ought to possess and be able to use all that scientific knowledge which is at his command.

Now for the remedy. It is simple. The General Medical Council should ordain that a curriculum be formed for a post-graduate course of dental surgery for men taking appointments in merchant ships, the Army and the Navy, and then for the War Office, the Admiralty and the Mercantile Marine to order all their surgeons to pass it.

The course need not be a long one, and should comprise instruction in—

(1) Dental Anatomy (Human), 1 course 12 lectures.
(2) Dental Surgery, 1 course 12 lectures.
(3) Practical work at a dental school for three months.

There would not be any great need for any mechanical instruction in this post-graduate course, and it should be

distinctly laid down by the General Medical Council that if the surgeon should at any future time desire to practise dental surgery in any town he would be required to go through a course of instruction in mechanical work and a full course of practical work at a dental school, and take the L.D.S.

I am, dear Sir,
Faithfully Yours,
GORDON HOOPER, L.D.S., R.C.S.Eng.
44, Devonshire Street,
Harley Street, W.

Communications, Letters, &c., have been received from :—

B.—Dr. Henry A. Bodeker (Kikuya); Dr. Bonnafy (Paris); Dr. Brown (Penang); Surg.-Capt. W. J. Buchanan (Dacca).

C.—Dr. Cunhu (Portuguese India).

D.—Dr. R. J. Drummond (Talawakelle); Major T. A. Dixon, R.A.M.C. (Peshawar); Dr. Chas. Dennehy (St. Lucia); Dr. Dumat (Edinburgh).

E.—Dr. Eder (Columbia); Dr. Chas. Dennehy (St. Lucia.)

F.—Dr. Hunter Finlay (Queensland); Dr. Farmer (Mysore).

G.—Dr. Douglas Gray (Zomba); Dr. Gahne (British Honduras); Dr. Otho Galgey (Jamaica).

H.—Dr. Stanford Harris (Las Palmas); Capt. Holt (Deblali); Major Hendley (Simla); Surg.-Lieut. C. B. Harrison (Shinawari).

J.—Dr. H. N. Joyat (Fiji); Dr. W. Johnston (Cape Town).

K.—Surg.-Lieut. W. H. Kenrick (Bombay); Dr. K. Chander Bose (Calcutta).

M.—Dr. Mease (Cassel): Major Moffett (Brechin); Lieut.-Col. Allan May (India); Dr. Maovicar (British Central Africa); Surg.-Col. McGann (Bangalore); Major Melville, R.A.M.C. (Quetta); Mr. C. H. Massiah (Demerara); Dr. R. Chas. MacWatt (Bombay); Dr. T. C. Mugliston (Singapore); Dr. Moorhead (Tong Shan).

N.—Dr. W. D. Neish (Jamaica); Surg.-Col. T. H. Newman (Punjab).

O.—Dr. O'Donnell (Oorgaum).

P.—Rev. Dr. Palmer (Westminster); Fleet.-Surg. E. R. H. Pollard (West Indies); Major Porter (Nasirabad). Dr. A. Pechell (Nowgong); Capt. C. E. Pollock, R.A.M.C; (Ranikhet); Dr. Wm. F. Pen-davis (Chile).

R.—Dr. Rho (Rome).

S.—Dr. Saunders (Perth, W.A.); Dr. F. C. Sutherland, Mr. James Stalker (Gold Coast); Dr. Shekleton (Niger Coast Protectorate); Dr. Scheube (Greiz); Dr. Sonsino (Pisa); Dr. E. J. Stubbert (New York); Dr. Max. Simon (Singapore); Mr. A. H. Spurrier (Zanzibar).

T.—Major C. R. Tyrrell, R.A.M.C. (Punjab).

W.—Mr. H. J. White (Persia); Mr. Watts (Bogra); Lieut.-Col. H. R. Whitehead (Rawal Pindi).

Papers Promised.

(1) Tetanus Neonatorum in the Tropics; (2) On some recent Queensland Epidemics; (3) Arsenic in the Treatment of Tropical Fevers; (4) The Abuse and Proper Use of Quinine. By Hunter Finlay, M.D.

Some Remarks on Ankylostomiasis. By Max. F. Simon, M.D., L.R.C.P., M.R.C.S.

(1) Lightning Stroke; (2) Clinical Notes on various Tropical Disorders. By W. D. Neish, M.D., L.R.C.P., &c.

Points in connection with the Outbreak of Plague in Calcutta. By Kailas Chander Bose, Rai Bahadru, L.M.S., &c.

Dysentery as a Factor in Liver Abscess. By Surg.-Capt. W. J. Buchanan, B.A., M.B., &c.

Births, Marriages and Deaths.

The charge for inserting announcements of Births, Marriages and Deaths, is 3s. The notice should be accompanied by a remittance.

BIRTHS.

DAUBENY—On 3rd inst., at Kucking, Sarawak, the wife of Charles Wm. Daubeny, Esq., of a son.

DENT.—On 10th inst., at Summerhill, Dumfries, N.B., the wife of Captain Edgar J. Dent, the King's Own Scottish Borderers, of a daughter.

DEATHS.

BRISLEY.—On 10th inst., at his residence, The Pines, Umzimkulu, South Africa, George C. Brisley, J.P.

LUCAS.—On October 9, at his residence, Endsleigh, Knole-road, Bournemouth, Benjamin Lucas, formerly of High Wycombe, and of Albury, N.S.W., aged 73. Australian papers please copy.

MUNRO.—On August 30, at Brisbane, Australia, Charles James Maillard, aged 38, second son of the late Surgeon-General William Munro, C.B., LL.D.

OTTLEY.—On October 10, at 39, Ladbroke Square, W., Richard Byam Ottley, formerly of Coolootai, New South Wales, fourth son of the late George Weatherell Ottley, of Antigua, in his 83rd year. West Indian and N.S.W. papers please copy.

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2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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THE CONDITIONS FAVOURING EXFLAGELLATION OF THE MALARIA PARASITE.

By F. KENNETH WILSON, M.B., B.S.LOND., M.R.C.S., L.R.C.P.

Senior House Surgeon, Seamen's Hospital, Albert Docks.

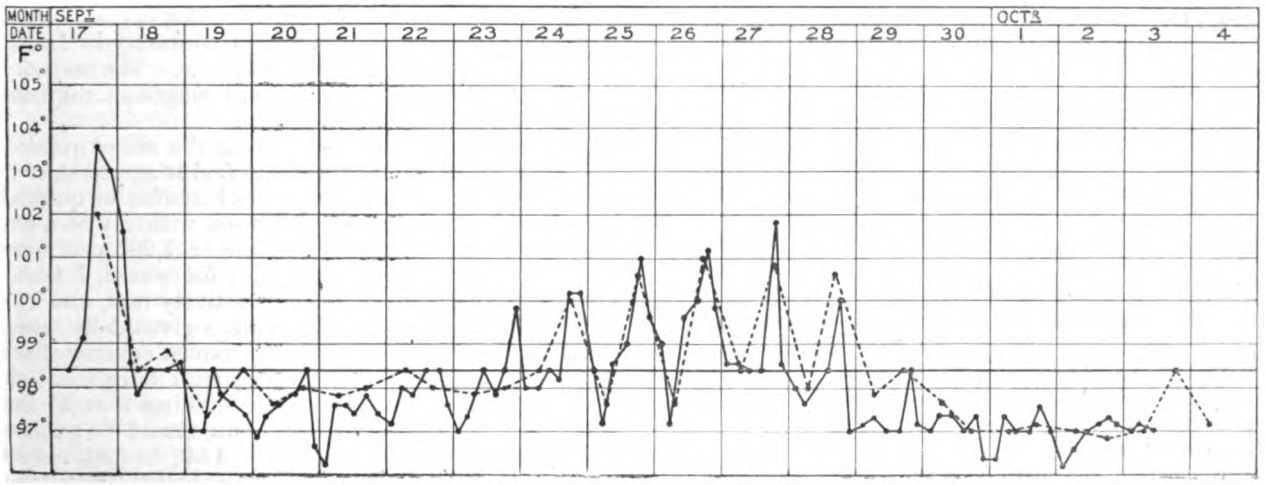
SURGEON-MAJOR ROSS, in the *British Medical Journal*, Jan. 30th, 1897, discusses the physical conditions favouring the evolution of the flagellated body of malarial infection from the crescent body. He describes an experiment on the transformation of the crescent body, and points out that this is facilitated by exposure of the blood to the atmosphere for a certain time. His experiments I have lately had an opportunity of repeating and confirming.

Johan T., a Russian, aged 38, ship's carpenter, had his first attack of malaria on 28th August, 1898, when he was in Mobile, Alabama. From that date he was ill nearly continuously with a daily attack of fever, the symptoms on alternate days being more severe. There were many on board his ship who had malaria, and the captain dosed them all with quinine. The Russian had his last dose of quinine six days before his admission to the Seamen's Hospital, Albert Docks, on September 17th, 1898. He was then cachectic: his spleen and liver were considerably enlarged; his tongue was large and teeth indented, and he had slight diarrhœa: there were no abnormal physical signs in his chest. His temperature rose on the day of admission to 103·5°. By the next day it had fallen to 98°, and remained slightly subnormal until September 23rd. On September 19th the parasites found in his blood were those of the malignant tertian type (æstivo-autumnal). Small forms were fairly numerous: on one occasion as many as three parasites were seen in one corpuscle. The crescent form of the parasite was found in large numbers; once twin crescents were seen. From September 23rd until the 26th there was an evening rise of temperature to about 101°; on each of these occasions profuse sweating occurred, but no rigor. On the 27th treatment with quinine—five grains in solution every six hours—was commenced. The doses given on that day evidently took effect on the rise of temperature due on the 29th, but were not in time to prevent the parasites from maturing on the 28th.

After the quinine had taken effect on the fever forms crescents were the only form of parasites found, and they were in rather smaller number than before. The quinine was continued until his discharge on October 4th. He was seen four days later and still had a very few crescents in his blood.

I.—(*a*) Finger pricked; cover glass preparation made immediately; ringed with vaseline and examined after fifteen minutes. Eight fields showed five

Black line indicates night and morning temperatures; dotted line four hourly temperatures.



TEMPERATURE CHART—JOHAN T.

crescents and three spheres. Another eight fields showed eight crescents — one field contained two crescents and one sphere. (*b*) Similar experiment. Examined after twenty minutes. Eight fields showed five crescents and three spheres. Another eight fields showed eight crescents. (*c*) Similar experiment. Examined after forty minutes. Eight fields showed four crescents and five spheres—one field contained one crescent and one sphere. Another eight fields showed seven crescents and one sphere.

II.—(*a*) Similar experiment, but the drop of blood was exposed on the finger for half a minute before the preparation was made. Examined at once. Eight fields showed two ovals, four spheres and two flagellated bodies. Another eight fields showed three spheres, two ovals and four crescents. (*b*) Similar experiment with one minute's exposure. Examined at once. Eight fields showed six spheres, one oval and three crescents—one field contained one oval and one crescent; another field two spheres. Another eight fields showed seven spheres and two crescents. (*c*) Similar experiment, with two minutes' exposure. Examined after thirty minutes. Eight fields showed six spheres, one flagellated body and one sphere in a leucocyte. Another eight fields showed eight spheres.

III.—(*a*) Similar experiment with one minute's exposure and breathing on the slide. Eight fields showed one crescent and seven spheres. Another eight fields showed three crescents, one oval, four spheres, and one flagellated body. (*b*) Similar experiment with two minutes' exposure and breathing on the slide. Eight fields showed one crescent and seven spheres. Another eight fields showed four crescents, three spheres and one oval.

IV.—Finger pricked through vaseline, the blood squeezed out under the vaseline and left thus exposed for an hour and then examined immediately, showed seven crescents and no altered forms.

The general conclusion we may draw from these experiments is that protection from the atmosphere and moisture delays the transformation of the crescent body, while exposure to these accelerates the change; thereby confirming Ross's observation.

METHODS OF USING QUININE AND STRAY NOTES ON MALARIAL FEVERS.

By Surgeon-Captain W. J. Buchanan, B.A., M.B., Dip. St. Med.

Superintendent Central Jail, Midnapore, Bengal.

In the course of an attempt at collective investigation on the use of quinine as a prophylactic, I was able to collect the views of fifty-one medical officers in civil employ in India, as to their methods of using quinine.

I find that the usual dose given is from 5 to 15 grs. Often 5 grs. three or four times a day, or more frequently 15 grs. each morning. In my own experience I find it best to give 15 to 25 grs. in the early morning, and usually consider it takes 1 drachm to prevent a recurrence of the paroxysm.

Regarding the period of the attack at which it is best to give quinine, I find there are practically three methods. Some give it at any time during the attack, regardless of a rising or falling temperature; others, and the majority, give it as soon as the sweating stage comes on and the temperature is falling. Others again wait till the temperature has fallen, and give it with a view of preventing a second paroxysm. When the fever is undoubtedly malarial, proved by blood examination or other evidence, it appears best to give the quinine at once. It will not cut short the actually present attack, but it can thus attack the hæmatozoa during sporulation, and so make sure of preventing a second paroxysm. On the other hand there is an undoubted prejudice among many men, and especially among native practitioners (and among patients), against giving quinine with a rising temperature. It gives headache they say, increases any tendency towards delirium, and besides it is very frequently rejected by the stomach. In hospital practice one usually gives quinine in the early morning, when the fever is very low or absent, and I usually order it to be repeated once after three hours. If malarial fevers behaved with text-book regularity, it would be easier to lay down rules, but as will be mentioned below, such is not the case in the fevers usually met with in the plains of Northern India. As a matter of fact, therefore, quinine is usually given in the fever-free state. In irregular fever attacks, or in the more rare regular remittants, when one is sure of one's diagnosis (as to a malarial origin), the quinine must be given in moderate doses (grs. 5 to 10) three or four times daily, and persevered in for several days. In some such cases I have given 30 grs. daily for over a week before I got the fever subdued.

Another question, about which some difference of opinion exists, is the necessity for a preliminary purge. The native patients, among which our practice largely lies, generally need this purgation.

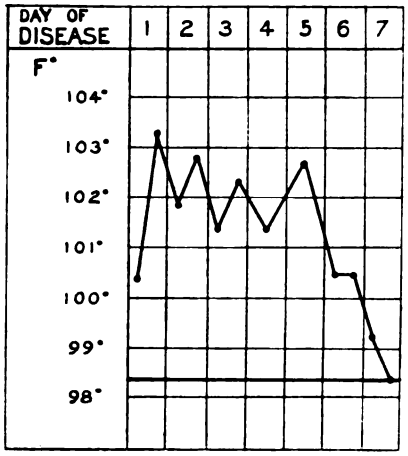
Their tongues are generally foul, and I usually find the quinine useless or rejected till the bowels are freely moved. When possible I give calomel (grs. 5 or 6) the evening before, or an early morning saline, or both, and followed by quinine in the morning. Of the fifty-one medical officers who answered my queries on this point I find the large majority are in favour of the preliminary purge. Others say "where indicated," "if constipation exist." But as a rule it appears to be usually considered necessary in India. Opinion is divided as to the best purge. The majority are for calomel, but castor oil and magnesia sulphate run it close.

As regards cinchonism following the use of quinine or other cinchona preparation, I find it agreed that it is very rare after the use of cinchonidine or quinine in prophylactic doses. This agrees with my own experience. Even when I was giving 1,200 prisoners 15 grs. of cinchonidine every day for weeks, I found symptoms of cinchonism comparatively rare, and extremely rare when 5-gr. doses were given daily, even for three or four months. The personal equation here comes in, but as a rule I agree with Surg.-Lt.-Col. A. Crombie (late of Calcutta) in thinking that in the treatment of malarial fever quinine should be pushed till these symptoms are complained of; in fact, unless these symptoms occur in my own personal experience

of fever attacks, I never feel sure that I have got rid of the attack.

As regards the type of fever, it is a much more difficult point to be decided than appears at first sight, especially as quinine is usually administered at once; the type of the fever is thereby necessarily altered, or, at least, does not always answer to ordinary described types. Moreover, in the crowds of patients one usually has during the malarial season it is seldom possible, with the staff at one's command, to do more than take the temperature twice a day, morning and evening. This naturally leads to an appearance of irregularity.

As regards intermittents, one certainly finds the quotidian type by far the most common, say 90 per cent., tertian 10 per cent.; and the quartans so rare as to be curiosities; but the majority of medical officers in India will say that irregular attacks in the autumn are the most common of all. Such a chart as the following is certainly a very common sight in an Indian hospital:—



Many of these are what the French writers call "subintrant," that is, the second and other paroxysms have begun before the temperature, due to the previous attack, has fallen to normal (anticipating), or there may have been a fall to normal during sleep, or at some period when the patient was not under observation (we have no nurses in native hospitals, and our sick attendants cannot be relied on for temperature taking). But while one must attempt to classify cases of malarial fever, it must never be forgotten that the clinical manifestations of paludism are manifold and types of fever blend into each other, and there exist many intermediate forms, and cases which begin as quotidian change to tertian and *vice versâ*, a fact which seems to me to be against the theory of special hæmatozoa for these types. At least, I have never been able to satisfy myself that the hæmatozoa found belong to either of the special varieties; further experience may lead to this skill, however, and it is possible that the cases I have observed of this kind may have been due to the fact that I gave quinine enough only to suppress one or more paroxysms, but not enough to break the fever.

Another point I have paid special attention to during the æstivo-autumnal fever season of 1897 is that the initial vigour is very frequently absent in the very hot weather, while it is very common in the attacks met with in November when the cold weather has begun. Epistaxis is, I find, a rare complication of malarial fever. I noted it only twice in 700 cases last year.

In native hospitals, and in jails in India, we are dealing with patients who have for years, at times, suffered from malarial attacks. In our well-drained, clean and comfortable jails, malarial fevers do not originate, but in the fever season slight causes light up the malaria latent in the system; that is, attacks are more in the nature of relapses than re-infections. This is understandable, now that we know the hæmatozoa can remain latent in the organism for long periods.

The vexed question of "remittent fever" is one on which I fear to tread. Cases of continuous malarial fever, lasting five, eight, ten, or eleven days, we often meet with, and can cut short by quinine; but that there is another not yet differentiated continuous fever, not malarial, not enteric, not undulant (Malta), I firmly believe, but I am not yet in a position to publish my notes and charts on this subject.

Pernicious Attacks of Malarial Fevers.

These are certainly rare in India at the present day. Of the fifty-one replies I received I find only three officers say they met with such. Twenty years ago such pernicious attacks, much resembling cholera, were not uncommon in many places, Peshawur, for instance. It is, however, possible that some cases of rapid unconsciousness, with high fever (temperature 107° F. 109°), which are treated as heat-stroke, may be really pernicious malarial attacks; one recent case I had in camp (temperature 107°). Patient had fever for two days before, temperature rapidly rose to 107°. I deluged him with cold water, after three hours' pouring water on him his temperature remained at 100°. I gave him 30 grs. quinine by the mouth, next afternoon temperature again rose to 105°. I again douched him and gave 30 more grs. quinine, and repeated the dose on next morning. Temperature remained normal. In this case patient was quite unconscious from the high fever.

Moreover, the personal equation must always be borne in mind; one man would call a case pernicious, when another would call it a very severe attack only.

Malarial hæmoglobinuria is certainly extremely rare in India. Of the fifty-one medical officers who answered my queries only two had seen cases; one was doubtful if his case was malarial. The only genuine case, therefore, in the experience of fifty-one medical men, is that furnished me by Dr. A. Crombie. This was in a prisoner in Dacca Central Jail. Considering the great opportunities enjoyed by Dr. Crombie in large cities like Calcutta and Dacca, the fact that he only met one case is proof of its extreme rarity.

MALARIAL PERIPHERAL NEURITIS.

By H. Campbell Highet, C.M., M.D.
Bangkok, Siam,

The publication of two cases of so-called "Malarial Neuritis" by Dr. Alexander James in the *British Medical Journal* of May 8, 1897, has induced me to forward the following notes of ten cases which I lately had the opportunity of treating in Singapore. Malarial neuritis is, besides, an affection about which very little seems to have been written heretofore, although the opportunities for its study must be innumerable. I trust, therefore, that, considering the scarcity of any literature on the subject, the length of these notes will be pardoned.

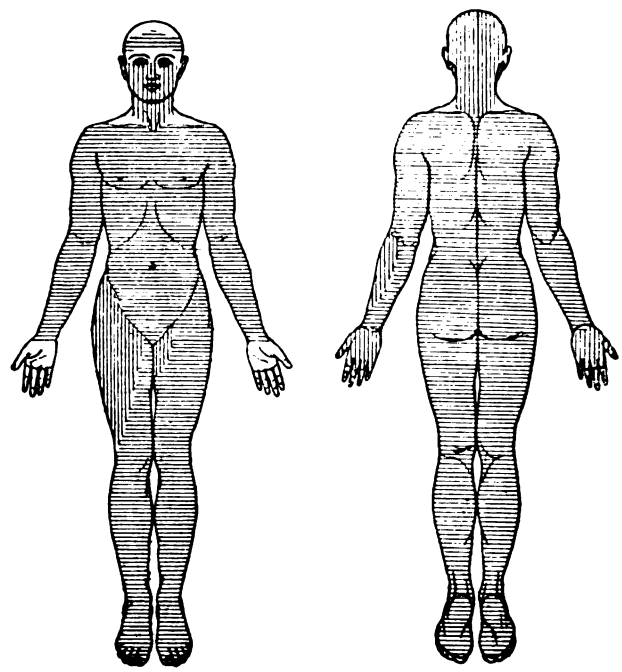
General Symptomatology.—The ordinary course of events in these ten cases has been that intermittent fever, or more commonly very frequent attacks of remittent fever, have been present for some months before any complaint is made with regard to the limbs. Sooner or later, however, pains in the legs and knees are complained of, not at first when at work, but on reaching home at sundown. During the night they are worse, and only pass off towards morning. Cramps too are often present. Later on, the knees are felt to be becoming weak; they often seem about to give way, they are painful in the daytime as well as at night now, and pain may also be complained of in the thighs, back and arms. The pain is continuous and boring in severe cases, and is increased at intervals by short, stabbing spasms. Tingling in the legs, numbness, cold feet, and a feeling of weight in the feet are also common symptoms. Fever still occurs at frequent intervals, or may be almost a daily occurrence, and by-and-bye the nocturnal pains become so severe that sleep is greatly disturbed. Weakness of the limbs amounting to paresis or even paralysis may ensue. It is noteworthy that the paresis may take on a markedly remittent character in its severity. In several of my cases, and in one especially, the weakness of the limbs was always much greater during the paroxysms of fever. In this case the patient stated that unless he "hobbled" upstairs before sunset he could not go up unaided at a later hour, as he then had lost almost all power in the legs. In one case hemeralopia was present.

Heart symptoms, such as tachycardia, arhythmia, and dilatation, are frequent, and as might be expected, enlargement of the spleen is the rule.

Before discussing the symptoms in detail, the following two cases may be given as typical examples of the affection as it has appeared to me in Singapore:—

I.—G. W. M., Eurasian, male, states that he has been a clerk in a saw-mill for the last twelve months, and that during that time he has had repeated attacks of fever. Five months ago he had a very severe attack of fever, and since then he has not had a normal temperature for more than two or three days consecutively. Three months ago, he began to complain of numbness and of a feeling of coldness in the feet and legs and of pain in the legs, thighs, back and arms. Soon the legs felt so weak that he had considerable difficulty in getting about towards evening. Severe cramps in the muscles of the legs and back became frequent, and were always worse after sunset. Latterly, the pain and cramps have been so severe at night-time that sleep has been greatly interfered with. For the last month he has had great difficulty in finding his way about at night, as at sundown the sight becomes very dim.

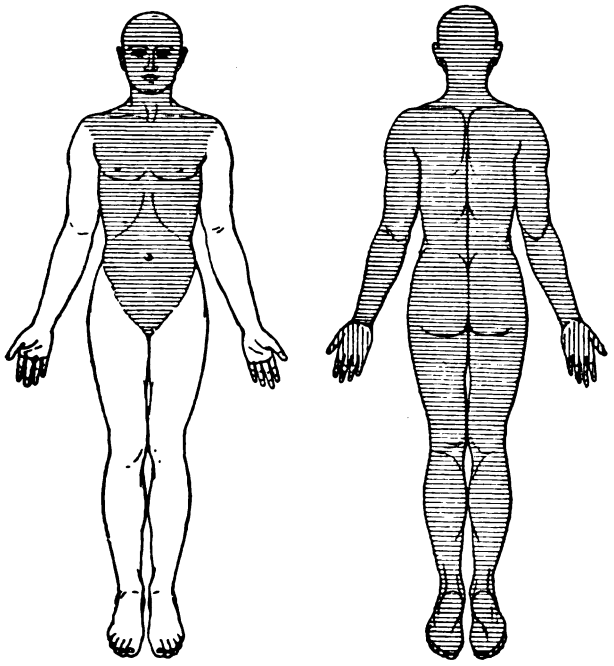
Present condition.—Nervous system.—He complains of severe stabbing and lightning pains in the feet, legs, back and arms, and along the course of the intercostal nerves, all of which symptoms are intensified after sunset. Tingling and numbness of the feet and legs are often complained of, and cramps, especially upon putting his feet on a cold surface, give rise to much suffering. During the cramps he states that the muscles of the calves are like hard balls, and are very tender to the touch. Continuous sensibility is deranged, as shown in the diagram, in obtaining the details for which great care had to be taken, as there is universal deep tenderness of the muscles, tendons and nerves. The shrinking away of the patient even when one grasps his arm lightly is most distinct, and on palpation of the nerves of the limbs, where capable of such inspection, they are found to be thickened, especially the ulnar nerves. The plantar, cremasteric,



and abdominal reflexes are present, but the knee and ankle jerks are absent. In testing for the latter there was some danger at first sight of coming to a wrong conclusion, as the tendons were so tender that the slight tapping, necessary to educe the jerk, was sufficient to give rise to such pain as to make the patient retract his leg even against his will. Voluntary power is considerably diminished in the legs, though less so in the hands, and the patient rapidly feels fatigued on attempting to walk or to write for any length of time. The muscles are all extremely tender and flabby, and although there is no marked atrophy of any set of muscles, the patient says that he is generally very much thinner than before he

began to feel ill. No tremors are present. *Heart*—normal; no breathlessness or palpitation on walking, as in Beri-beri. *Lungs*—normal. *Liver*—normal. The spleen is much enlarged. *Eyes*—He complains of a veil coming over the eyes towards sunset, which greatly interferes with vision. In the daytime, I found vision to be normal, but at night, about 7.30 p.m., it amounted to J.10 in a bright lamplight. On ophthalmoscopic examination, the disc and general fundus appeared to be paler than during the day, the vessels seemed to be rather empty and pulsation was distinct. There were no hæmorrhages, no signs of neuritis, nor other pathological conditions. A bright light enables the patient to see somewhat better, but sight does not return to the normal until after a good night's rest in the dark. There is no fever at present, although he has had it within the last few days. Cerebral system normal. In this and all the other nine cases any suspicion of alcoholic neuritis may be dismissed at once.

II.—J. R., Eurasian, male, has also been a clerk for over a year in the same saw-mill as the former patient, and like all the other employés, has suffered from very frequent attacks of malarial fever. Some three months ago he began to complain of pains in the legs, at first only at night-time, after returning from work. The pain usually passed off towards morning, but latterly it has been of an almost persistent boring character. Day by day the legs have gradually become weaker, until within the last two weeks he can only hobble about with the aid of a couple of walking-



sticks. He states that after sunset all his symptoms are so much more marked that unless he ascends to his bedroom before sundown, he cannot even hobble upstairs without assistance. He has become greatly reduced in weight during the last few months. The cutaneous sensibility is shown by the diagram to be less altered than in Case I., and the special senses are all normal. The muscles are flabby, very tender on deep pressure, and generally show signs of atrophy. This is especially the case with the peroneal muscles of the legs, and there is considerable paresis of both legs and arms. The superficial and deep reflexes are all exaggerated, the nerve trunks are extremely tender, but no swelling can be made out as in the last case. The lungs are normal. The pulse is slightly irregular and rapid, averaging 100, but the circulatory system is otherwise normal. The spleen is enlarged. There is distinct fluid effusion into both knee-joints without marked pain.

These are two typical examples of the disease, but cases may show marked gradations in severity from slight neuralgia and tenderness of the nerves to paralysis of muscles and great pain. It might be well to discuss in detail the various symptoms which were noted and the physical signs detected in these ten cases.

The nervous system may be taken first. Sensory symptoms are usually to be found if looked for in well-marked cases. In slight cases, they may be absent or there may be simply a slight dulling of sensation at the finger tips. Patches of anæsthesia may alternate with areas of hyperæsthesia, the latter being especially marked around the joints. It is not uncommon to find anæsthesia above and below a joint and a ring of hyperæsthesia around the joint. In the evolution of a case, hyperæsthesia often gives place to anæsthesia before sensibility returns to the normal. The hyperæsthesia may in some cases be so severe as to make even lying in bed a torture. This was so in Case I. in the earlier stages, until anæsthesia took the place of increased cutaneous sensibility.

Tenderness of the nerves can be elicited early in a case, and frequently, as in Case I., some swelling and thickening of the trunk can be made out, especially if such readily accessible nerves as the median or ulnar be implicated. The reflexes are usually exaggerated at first, but the deep ones, as for example the knee jerk, may be absent from an early stage. Of these ten cases, the deep reflexes were absent in six cases in which the duration of sickness had amounted to 1, 2, 3, 6, 6, and 10 months respectively. In four cases whose duration had already been 3, 3, 4 and 4 months, the deep reflexes were present and were exaggerated in all but one. The reason for this irregularity with regard to these reflexes is uncertain, but that the exaggeration in these cases of some months' duration was not due to an extension of the nerve trouble to the cord, I am certain.

Paralytic symptoms were not highly marked in the majority of these cases. In most, it amounted to a feeling of heaviness of the limbs and weakness at the knees. In two cases, however, locomotion was much interfered with, especially on account of weakness and atrophy of the extensor muscles. In both of these cases, it was noted that the amount of muscular power varied from time to time. In Case II. we find a daily remittent condition, for at sundown the power of independent locomotion, even with the aid of a pair of walking-sticks, was gone. The patient stated that not only did he then lose the power in his legs, but that also the hands and arms were so weak as to be useless in aiding him. In another case, in which fever

came on nearly every day, the rise of temperature was always accompanied by increased weakness of the limbs. In none of these cases were there noticed the typical wrist and ankle-drop which are so often seen in Beri-beri, alcoholic or lead paralysis, and in none were bladder or rectal troubles ever present.

Muscular System.—Cramps have been already noted. During these, the muscles gather themselves into knots and the pain is excruciating. Deep pressure even in slight and doubtful cases generally gives rise to pain. Early in the course of the disease, if not checked by treatment, the muscular tissue becomes flabby and atrophy ensues, although in none of these cases have I noted early, rapid and localised atrophy such as one finds in many other forms of neuritis.

Joints.—In one case, Case II., effusion into the knee joints was noted for a few days, accompanied by a slight rise of temperature to 99°F. Pain in the joints without effusion is an early and frequent complaint, and as I have already noted, a ring of hyperæsthesia around the joint is frequent, even although there be anæsthesia above and below.

Circulatory System.—In many cases the heart remains unaffected, and shows no excitability on movement such as one sees in Beri-beri. In a few cases the pulse is rapid and slight arhythmia is present. In one case slight angina with tachycardia came on regularly about two in the morning for some weeks. As anæmia is present in all cases and often is extreme —malarial cachexia—dilatation with hæmic murmurs is not infrequent as a result of the cachexia, whereas the irritable and perhaps irregular pulse found early in some of these cases is due to nerve affection. No distinct vaso-motor symptoms were noted.

Slight œdema of the feet and shins was present in two cases, but in both there were marked anæmia, dilatation of the left ventricle, and hæmic murmurs to account for it.

The Liver is sometimes found to be enlarged, but this is not such a constant symptom as enlargement of the Spleen. This was very distinct in most of these cases where the patients had all suffered from malaria off and on for months.

Diarrhœa of a typically remittent character and cured by quinine was present in one case in which astringents or sedatives without quinine always gave rise to fresh outbreaks of fever. The discharge from the bowels was, in fact, acting as a safety-valve.

Fever is often present from day to day and is purely malarial in character, being readily influenced by quinine or change of residence to a non-malarious district.

Ætiology.—Malarial neuritis is a toxic affection of the nerves—the toxin being the product of the malarial germ. The affection corresponds to other toxic neurites, and, like most of them, is readily cured, even although the condition has been of long standing and has given rise to considerable physical changes. In this it differs from the form of neuritis of which leprosy is a good example, where the germ itself invades the tissues. I think that it may be definitely stated that one attack of malarial fever rarely gives rise to well-marked neuritis. We have seen cases of very severe fever to be followed by loss of knee-jerks and even by a feeling of numbness and weight in the limbs. Should the patient make a good recovery from fever, these symptoms lessen with returning health and often entirely disappear. Should, however, he remain in a malarious district, suffer from repeated attacks of malarial fever and continually absorb the malarial poison, he is in a fair way to contract malarial neuritis. This is especially the case if his occupation entails the usual hardships of jungle life. It takes, however, frequent attacks of fever or residence for a considerable time in a malarious district to give rise to an attack of neuritis; in fact, the cumulative action of the poison on the nerves in repeated doses is required to bring about the pathological changes which give rise to the symptoms, for it would seem that one infection even of an acute and very severe nature is insufficient. Gowers mentions the fact of hemiplegia and aphasia having been met with in the course of severe malarial fever, and states that they "have been ascribed to obstruction of cerebral vessels by pigmentary matter." These conditions arise suddenly, however, and are altogether different from the conditions which we are now describing.

Incubation period.—Is it possible to determine such a period? This is a question which has often occurred to me, and it may be expedient to give the results of my experience. Eight of these cases arose in one locality—in a saw-mill on the outskirts of Singapore, built on reclaimed swamps bordering a low-lying tidal creek. The subsoil was composed of heavy alluvial clay, and was covered over with a layer of tan-bark, sawdust and general rubbish. At high tide the subsoil water rose to a few feet from the surface of the mill-yard. Reclamation was commenced in November, 1894, and was completed in March, 1895. Up till August, 1896—the date when I commenced to investigate these cases—no member of the staff had escaped from almost weekly attacks of malarial fever. Many were scarcely ever free from fever, and from the history of several patients who had left the mill before August, it is plain that they suffered from neuritis. In July, operations were commenced for the construction of a canal, by starting about two hundred yards inland and cutting a deep trench towards the river. Besides such an inevitable disturbance of old alluvial soil, there was left until the completion of the excavations this deep trench, forty feet or so wide, into which drained the surface water of the neighbourhood, and which was also flooded to a considerable depth by each tidal rise of subsoil water. The natural sequence was an intolerable stench and a rapid increase in the number and severity of cases of fever, and symptoms of neuritis soon developed in most of the employés who were strong enough to continue at work in the mill. The following table gives the nationality and the length of time which each of these cases (of which I have notes) had been employed in the mill before the onset of symptoms of neuritis:—

(1) Hindostanee	3 months.	(5) Eurasian ...	9 months.
(2) Eurasian...	3 ,,	(6) ,, ..	9 ,,
(3) Malay ...	6 ,,	(7) European ...	10 ,,
(4) Chinese ...	7 ,,	(8) Chinese ...	20 ,,

Adding to these another case, that of a European who suffered from severe malarial fever for three

months before the symptoms of nerve trouble began to show themselves, we have an average of rather less than eight months of an *incubation period*. This proves, therefore, what I have already stated, that prolonged and repeated infection is necessary for the production of malarial neuritis even in the case of Natives and Eurasians, whose conditions of living are far inferior to those of Europeans.

Prognosis.—Provided the patient can be removed from the infected district, and can have efficient hygienic and medicinal treatment, even the least promising cases may have hopes of ultimately making a good recovery.

Diagnosis.—To those who are familiar with Beri-beri, it will be at once apparent that in many respects the two diseases are wonderfully alike, and in some cases it is necessary to suspend one's judgment and to await further developments before coming to a definite opinion. Of course, with a marked case of moist Beri-beri there can be no mistake, but of this I feel certain, that many cases of multiple malarial neuritis have been described as Beri-beri. The distinctive features of the two diseases may be tabulated thus:—

Malarial Peripheral Neuritis.	Beri-beri.
Anæmia usually marked; always present.	As often absent as present.
Heart symptoms, if present, are not very marked, and are usually due to the anæmia.	Heart symptoms an important and almost constant feature.
Spleen usually enlarged.	Uncertain.
Local œdema rare.	Present at one time at least in history of case, and often extreme.
Serous effusions very rare.	Common cause of death.
Fever precedes neuritis for long time, and often accompanies it.	Fever need not precede neuritis for long, but often accompanies rapid onset of symptoms.
Fever distinguished by Malays and Malay-speaking races as "demam"—malarial fever and ague.	Called simply "panas"—warm, hot—as distinguished from ague and fever.
Symptoms practically never acute in onset.	Often acute.
Incubation period usually of months' duration.	Often apparently of only a few days.
Symptoms often show striking exacerbations of a daily remittent type.	Paralysis does not show nightly increase, as in M. P. neuritis.
Gait of a shuffling type in paresis or paralysis.	Gait equine and typical; loose knees and ankle drop.
During attacks of fever plasmodium may be found.	Absent, unless malaria present as a complication.
Cases readily cured.	Sudden death common.
Quinine and arsenic curative.	Not so in beri-beri.

Such are the distinctive features of the two diseases as they have appeared to me, but it must be remembered that, considering the fact of the two diseases having a similar *habitat*, a mixed infection is probably a very frequent occurrence. Treatment resolves itself into hygienic and medicinal lines. The patient should be at once removed from a malarious district, and when possible should take a sea voyage. The improvement which often follows from this alone is in many cases striking. Quinine, of course, should be given in large doses at first, and may be followed by arsenic and strychnia. When all symptoms of nerve irritability have passed off, hypodermic injections of strychnia, massage and electricity should be employed, and to those who can afford it, a visit to one of the European Spas will usually complete the cure.

The question of returning to the Tropics will often be put to the physician at home, and may be a difficult one to answer. No patient who has had malarial neuritis should be allowed to return to the district in which he has contracted the disease, nor should he be allowed to engage in any occupation which will necessitate his remaining near newly-turned up earth. If it be obligatory for him to return to the Tropics, then he should elect to reside in one of the larger towns or cities which are often peculiarly free from malaria, but even then he must remember that he is always risking a return of his old complaint.

THE SEQUELS OF "SUNSTROKE."

By L. Westenra Sambon, M.D. (Naples).
London.

The question of the sequels of so-called "sunstroke" is of great importance, especially in its medico-legal aspect. In the United States of America, hundreds of men are actually drawing pensions on the score of infirmities attributed to insolation.

The list of ailments given by some authors as enduring results of "sunstroke" is, like that appended by others to their writings on influenza, little less than the nomenclature of diseases. The following are said to be the most frequent sequels of sunstroke: inability to withstand heat, intolerance of light, vertigo, insomnia, difficulty of speech, irritability, persistent or frequently recurring headache, loss of memory, mental weakness, insanity, tenderness over the spine, twitching of the muscles, wrist and foot drop, cutaneous anæsthesia or hyper-æsthesia, muscular atrophy, hemiplegia, general paralysis, epilepsy, dyspepsia, obstipation, anæmia, functional disturbances of respirations, a variety of heart lesions both organic and functional, deafness and blindness—such very different conditions are mostly attributed to sunstroke in cases of pension claim and, it is important to note, are often found to have developed years after the alleged insolation.

Before attempting to discuss whether these ailments should be considered as sequels of sunstroke, it is well to determine what constitutes a true sequel in the case of any disease.

The true sequel of any disease is an abnormal condition directly resulting from the lesion produced by the specific cause of the disease and persisting a certain time or even permanently after recovery from the disease is seemingly established.[1] Paralysis is likewise a true sequel of diphtheria. It can be experimentally produced in animals by the inoculation of the toxic material produced by the Klebs-Loeffler bacillus.

Septic processes cannot be considered as true sequels, although in certain diseases they be so

[1] Stricture of the œsophagus is a true sequel of corrosive poisoning.

common that for a long time they were thought to be essential features of the disease. We all know the frequency and gravity of septicæmia in diphtheria, the streptococcus pyogenes being an almost constant attendant of the Klebs-Lœffler bacillus. But seeing that complications, which were formerly so common and serious, have now, through proper care and cleanliness, become altogether exceptional, we can no longer regard septicæmia or pyæmic complications as sequels any more than we should now, as in Parré's time, recognise erysipelas to be a sequel of gout.

Tuberculosis is frequently considered to be a sequel when following an attack of typhoid fever or of typhus, and likewise rheumatism has been classed amongst the sequels of scarlet fever. We might very reasonably invert the order and call the typhoid infection a sequel of tuberculosis, because the tubercular infection was probably contracted long before, but had remained in an almost quiescent condition until organic resistance was enfeebled by the new and exhausting disease. When we consider how complications vary in different epidemics we are bound to admit that they cannot be regarded as sequels, In some epidemics cerebro-spinal fever is complicated by arthritis, in others by pneumonia.

With the advance of modern science the old ideas concerning the entity and reciprocal connections of diseases fall before their true etiology, and every day we grasp better the frequency and importance of mixed infections. But old opinion dies hard, and it will linger long round the diseases which have not as yet come within the grip of modern investigation. In the light of what I have said regarding sequels, it is evident that the multitudinous ailments regarded as results of sunstroke are found to be mostly independent of the latter. But what is sunstroke?

In a paper published in the *British Medical Journal*, March 19, 1898, I endeavoured to show that two very different conditions were confounded under the common appellation of sunstroke. One, called ardent fever by the old Anglo-Indian surgeons, and thermic fever by American authors, is undoubtedly a specific infectious disease; the other, often termed sun-syncope or heat-prostration, is nothing more nor less than ordinary syncope, and is therefore erroneously considered to be a mild or incipient form of thermic fever with which it has no connection whatsoever.

Thermic fever, which I have called siriasis, to avoid a more inappropriate terminology, is a specific fever with striking and characteristic symptoms analogous to those of other infectious diseases. It is usually preceded by thoroughly established premonitory symptoms which indicate clearly a period of incubation characteristic of all infectious fevers. It is also frequently followed by relapse, which proves the occurrence of reinfection from within and is therefore another strong evidence of its parasitic nature. Siriasis often prevails in epidemic form, and its outbursts, which occur at irregular and distant intervals, are in no way connected with heat maxima, but are limited to restricted and well defined areas. The immunity, so similar to that of yellow fever, which this disease confers to those who have long resided in its habitat is another proof of its infectious nature; but the most evident, the most conclusive proof to all such as have any knowledge of the laws which govern the distribution of organic life on the earth is that of its geographical distribution. Siriasis is not bound by isothermal lines as its thermic etiology would necessitate, but is limited, like yellow fever, like dengue, to a few coast districts and to the valleys of some large rivers.

Heat prostration is not a disease, but only a condition symptomatic of disease. It may supervene in any place and at any time under the most varied conditions, but always in persons subject to organic disease involving circulatory mischief.

In his excellent paper on "Tropical Heart,"[1] Surgeon-Colonel Macleod shows that, notwithstanding age, selection and elimination, diseases of the circulatory system constitute a considerable and serious factor of sickness and mortality in the British Army, more especially in the tropics. The same can be said more or less of all European armies, and also of the army of the United States of America. This is the reason why on long forced marches in the winter, and on parade grounds during summer, so many men fall out of the ranks in a fainting condition, but none ever show the symptoms peculiar to siriasis except in the localities and season in which the latter prevails.

Having drawn this distinction, it is now our business to enquire whether the ailments referred to at the beginning of this paper as sequels of sunstroke are connected with true siriasis or with ordinary syncope.

Running carefully through the vast literature of so-called "sunstroke," we find that authors who report on true cases of siriasis very seldom mention any morbid condition which might be considered as a sequel of the disease, with the exception of extreme weakness and anæmia following immediately on the attack, and from which the patient makes usually a good and speedy recovery. Whenever severe disturbances are mentioned the alleged insolation turns out to be ordinary syncope.

Dr. T. H. Sherwood (International Med. Congress, Washington, 1887), says: "My experience in the army during our late civil war leads me to the conclusion that genuine cases of *coup de soleil*, or heat-stroke, in which the cerebro-spinal nerve centres are involved, are extremely rare, and that the majority of these cases which have gone upon our army record as sunstroke are simply cases of heat-exhaustion in which the heart is mainly involved. . . . . I have since had the opportunity, in the Pension Bureau, with which I am connected, of studying the after history of these cases, and in the great majority of them have found that the heart is involved, and that we have hypertrophy, dilatation, and sometimes valvular disease. The main object of my remarks, however, is to direct attention to the after history of these cases, since our pension records are full of applications for pensions based on disabilities attributed to "sunstroke," and we have such grave diseases as insanity, epilepsy, locomotor ataxia, and paralysis agitans, attributed to this cause. It will be seen, therefore, that it is a matter of great importance,

[1] The title of this paper is misleading; there is no such thing as a tropical heart.

with those who have the making up of the army records, that the diagnosis be accurate, and that the after history of those cases which recover from the primary lesion should be closely watched and accurately recorded."

Dr. H. Ernest Goodman says: "I had a long experience in the army, during the late war, of cases called sunstroke, and do not recall a pure case of sunstroke with high temperature, but I attributed the trouble to heart failure, and they usually recovered after a few hours and returned to duty. These cases of "sunstroke" since the war have applied for pension, and I have examined upward of 60,000 applicants for pension, among whom were many cases of alleged sunstroke. The symptoms are obscure pain in the head, inability to stand exertion or the sun, and loss of pluck. Examinations of these cases almost invariably show a hurt at base or apex of heart, and their history has almost invariably confirmed me in the opinion that heart disease was the cause of most of the alleged sunstroke during the war."

Dr. Spalding, another very able examiner says: "Verily the results of insolation upon the sight are manifold when we listen to pension claimants' complaints. . . . It is my opinion that insolation rarely affects the sight permanently, and if so, that the results in the form of optic atrophy ensue within a few months, never, as the innumerable pension claimants assert, at any interval extending from four to nineteen years."

To show how easily one can fall into error, I will mention Dr. Thomas B. Christie's case, published in *Lancet*, August 2, 1879. It was a case of general paresis of the insane, attributed to sunstroke. The patient, an officer in the Indian army, was reported to have had *coup de soleil* at Pulneys, Madras, in 1868, after which followed headache on the left side, difficulty of articulation, and other signs of cerebral disturbance. "This was followed by singing noise in the left ear, and mal-administration of vision of the left eye from dilatation of the pupil followed by great contraction. The memory became defective of recent events, and an eccentricity of conduct and manner became evident. Exaltation of ideas, great excitement and a loss of control, were followed by excess of depression."

The brother of this patient had never been in India and was never the subject of *coup de soleil*, and yet the very same disease supervened in him and ran the same course though more rapidly. The *post-mortem* appearances in both cases were identical.

I might adduce a great deal more evidence, but I believe what I have mentioned will be ample to prove that the numerous infirmities attributed to siriasis are in no way sequels of this disease, but most probably further and more grave manifestations of some chronic organic disease of which one or more prostrations had been an early indication. I do not wish to infer that siriasis has no sequels, because, like other infections, it certainly has its peculiar consequences, one of which is the extreme weakness which may persist for some time after recovery.

THE INFLUENCE OF RAINFALL ON DEATH-RATE IN THE TROPICS.

By P. Carmody, F.I.C., F.C.S.
Trinidad.

A concise statement of the influence rainfall bears to the death-rate in the city of the Port of Spain, Trinidad, may prove of scientific interest at the present moment, more especially as the system of sewerage in the town is about to be changed.

In a few years we shall be able to compare the statistics before and after the introduction of improved sewerage. Meantime I venture to record the pre-sewerage period.

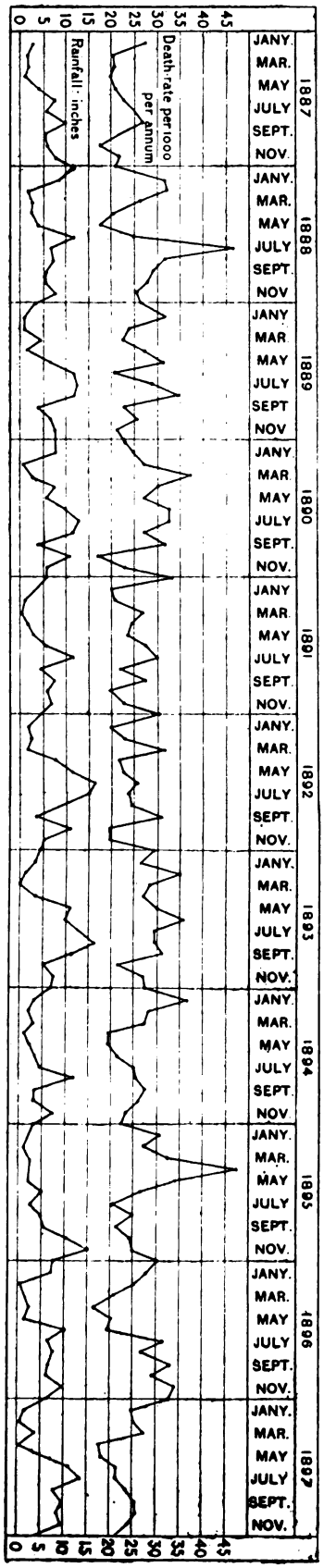
The accompanying diagram refers to the town of Port of Spain, Trinidad, B.W.I.; with the sanitary condition of which, as a member of the local Board of Heath, the writer is intimately acquainted. Including the important suburb of Belmont, the town *now* contains 50,000 inhabitants; but for convenience of calculation it has been assumed that the population has been the same during the eleven years under review. This assumption does not materially affect the *variations* in the death-rate curve. The town is sewered very inefficiently in parts; but the greater part of it has old-fashioned cess-pits the walls of which, in only a few exceptional instances, are water-tight. Of these cess-pits there are about 2,000, and each one is a focus for the pollution of the surrounding soil. They may fairly be compared to spreading colonies of microbes on a growing plate culture. The heavy rains carry the soluble constituents of the pits far into the subsoil; and the whole of the subsoil in the cess-pit area is one large hot-bed of pollution. The level of the ground water is at times subject to great fluctuations; at others, to months of comparative rest. In some parts of the town, the ground water rises after very heavy rains to such an extent as to cause the cess-pits to overflow; and all the year round it acts as a sort of natural subterranean punkah whose oscillations are dependent on the variability in the amount of rainfall. During a variable wet season the ground water rises and falls regularly, and the underground air is in consequence frequently changed; during a dry season, or a prolonged period of heavy rains with but little variation in the total amount of rainfall, the ground air remains longer in contact with the polluting soil, and its upward displacement by subsequent heavy rains would be likely to prove more injurious to health. The curves confirm this view.

The average death-rate of the town is about 27 per 1,000; when the death-rate rises above 30 it may be considered "high," and "low" when it does not exceed 25.

During the dry season (December to May), the rainfall per month is usually below 5 inches; and above 5 inches from June to November, with the exception of September, which is usually a dry month.

The most striking fact shown by the diagram is the remarkable rhythmic rise and fall in both curves, those of the rain curve usually preceding those of the other by a month or two.

Another striking feature of the diagram is that a rainfall of about ten inches causes a death rate of



PORT OF SPAIN.
(Population taken at 50,000.)
Comparative Curves of Rainfall and Death-rate, 1887 to 1897.

about or above thirty.[1] After heavy rains, a fall in the level of the ground water is usually succeeded by a marked diminution in the death-rate. The probable explanation of the latter is, that the heavy rain thoroughly washes the surface and subsoil, carrying with it all polluted matters, and that whilst the level of the ground water is subsequently falling, air is being drawn *down* into the soil. When the level of the ground water remains fairly constant for a few months, a subsequent slight rise in the rainfall may cause a heavy death-rate. This may be due to the expulsion of air more polluted in proportion to the length of time it has been in contact with the subsoil.

Abnormal seasons are notoriously unhealthy. If the dry season is unusually short, or unusually prolonged, or unusually late, there is a heavy death-rate, often an epidemic.

The whole of the town is about to be placed under a proper sewerage system; and for future comparison these curves are likely to prove serviceable.

The only other great influence likely to affect the death-rate curve is the water supply. This is analysed monthly, and is of great purity except when the rivers are in flood. It is allowed to deposit some of its suspended impurities by subsidence in small reservoirs, but is not filtered. The quality of the water has been the same during the period of eleven years. No epidemics can be directly traced to the water supply during the period. The flood waters are not collected in the reservoirs. But it is unwise wholly to exclude from consideration the undoubted influence which an unfiltered water supply might exercise on the general health, and consequently the death-rate. So far the writer's opinion inclines to the view that the contaminated air of Port of Spain is the principal factor influencing the death-rate; and the curve of 1897 strongly supports this view.

It would have been of great advantage to compare the corresponding curves of Georgetown (B. Guiana), Bridgetown (Barbados), and Kingston (Jamaica), in each of which towns the water supply is analysed regularly; but the figures cannot be obtained from the official returns for these colonies. The importance of the subject from a sanitary point of view may induce some local sanitarians to prepare and publish the curves for their respective towns.

AT Bangkok, Siam, a hospital has been opened, at which the nursing is to be solely conducted by a religious community of European Sisters. Whilst congratulating the Siamese upon this distinct proof of enlightenment, we only hope that the plan of entrusting the nursing of their Hospitals to Sisters of a "religious community" may be more successful than other attempts in the same direction, we know of in the Far East, proved to be.

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[1] The fact that the death-rate has not reached thirty throughout 1897 is significant, notwithstanding that two wet months (June and July) were succeeded by three months in which the rainfall was heavy but not varied in amount. During 1897, greater attention has been paid to sanitary matters, and especially to the quarterly cleansing of the frequently used cesspits in barrack yards, where the poor live in large numbers.

QUININE AMAUROSIS.

By M. T. YARR, F.R.C.S.I.

Major, R.A.M.C.; Chief Clinical Assistant, Royal Ophthalmic Hospital, Moorfields; Member Ophthalmological Society of Great Britain; &c.

THE series of cases published by Dr. Fernandez of Havana (commented on in a recent number), together with the recent remarkable researches of Holden in experimental quinine amaurosis, have focussed our attention once more on the toxic effects of quinine on the retina and optic nerve and incidentally emphasised the grave responsibility incurred by those who persist in prescribing large doses of the drug. A short sketch of the affection, its symptoms, pathology, and diagnosis seems therefore opportune at the present moment.

What is quinine amaurosis? Briefly, it is a temporary blindness, which leaves on its subsidence certain permanent defects of vision, the chief being contraction of the visual fields, diminished light perception, and diminished colour sense; caused by a toxic dose of quinine* producing in succession spasm of the retinal vessels, then breaking down of the less resistant layers of the retina from the diminished blood-supply, and finally, an ascending degeneration of the optic nerve fibres. Granting the word "temporary" a somewhat wide signification, the above may be accepted as an accurate and fairly complete definition.

For the last seventy years visual disturbances due to quinine have been recorded by writers, in fact, almost since the first introduction of the drug. Unfortunately, however, until comparatively recently, malarial eye affections and quinine amaurosis have been confounded together, visual defects being attributed to malaria, which were too often the result of quinine. At the present day such confusion ought not to exist; the points of distinction between quinine amaurosis and the group of affections commonly included in the term "malarial amaurosis" are many and clearly defined.

At one time it was thought possible that the effects on vision might be due, not only to quinine, but also to the diseases for which it was prescribed; but the uniformity of the signs and symptoms of quinine amaurosis in all kinds of diseases, its repeated occurrence in healthy people who have taken quinine by mistake[1] and the experiments of Barbaschew[2] on human beings, and Brunner,[3] de Schweinitz[4] and Holden[5] on dogs, have finally disposed of this theory, if it were ever seriously entertained.

Any of the alkaloids of cinchona appear capable of producing toxic effects, though sulphate of quinine is probably the most active. One case of amaurosis following the administration of tincture of cinchona is recorded.

Toxic Dose.

It is impossible to fix definitely the amount of quinine which will produce amaurosis as individual idiosyncrasy comes so much into play. It has been produced by doses amounting to only fifteen grains in the twenty-four hours; on the other hand, as much as an ounce in the same time has had apparently no ill effect—probably through non-absorption. De Schweinitz mentions that he himself has suffered from transient amaurosis after fifteen grains in twenty-four hours, and I have had a precisely similar experience in China. A single large dose seems to be less dangerous than repeated doses reaching a large total in the twenty-four hours, and it is always advisable to remember the liability to relapse after quite small doses.[6]

It may safely be asserted, however, that single doses over 20 grs., or repeated doses amounting in the aggregate to more than a drachm in the twenty-four hours, are always dangerous. Personally I have never given more than 15 grs. as a single dose, or more than 40 grs. in twenty-four hours; I have never seen the slightest benefit from larger doses, but on the contrary have often seen harm. A high authority on the subject—Dr. Patrick Manson[7]—writes thus:—

"There is great difference of opinion and practice about the dose of quinine. Some give thirty grains at a dose, some give three. The former, in my opinion, is too large a quantity for ordinary cases, the latter too small. . . . I believe that nothing is gained by excessive doses: in ordinary circumstances thirty grains spread over two or three days is usually amply sufficient to check an intermittent. . . . *If a supposed ague resists the doses of quinine mentioned, it is advisable to revise the diagnosis.*" (The italics are mine.)

It is difficult to characterise in language of judicial moderation the action of qualified medical men who prescribe quinine in eight or twelve-gramme single doses, and even reach the astounding total of *forty-two grammes in the twenty-four hours*;[8] one may, however, be permitted to hint at the likelihood in the near future of such cases possessing a medico-legal, as well as an ophthalmological, interest.

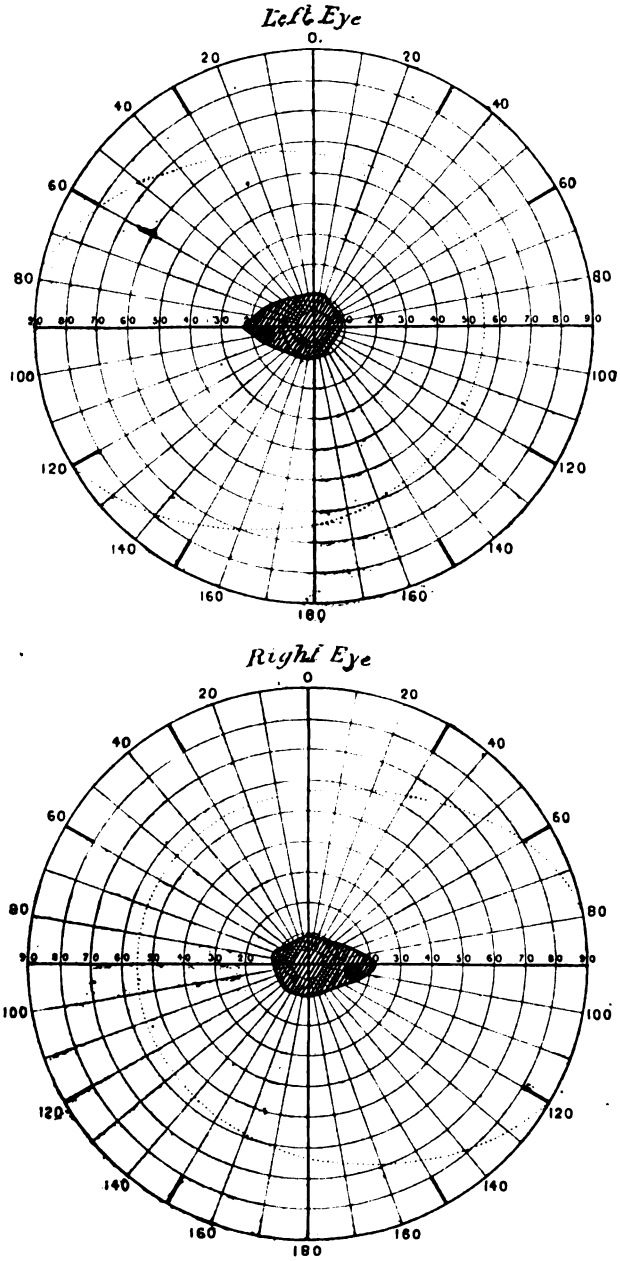
Symptoms.

The general evidences of cinchonism—throbbing headache, ringing in the ears, and deafness—are well known. Shortly after the onset of these symptoms, patients often complain of dimness of sight, without, perhaps, giving the symptom much prominence; and should the eyes be examined in such cases, probably nothing will be found save slight diminution of light perception, some loss of accommodation, and sluggish reaction of pupils to light. No serious results need be anticipated from these symptoms, but they should serve as a warning to discontinue the quinine, or at least to greatly reduce the doses.

A well-marked case of quinine amaurosis is, however, very different. Blindness, more or less complete, comes on with startling suddenness. In the case described by me in the September number of the Journal the patient said "his sight went all at once," and in Browne's case the sudden loss of sight was likened to "turning out the gas."[9] In severe cases there may be absolutely no perception of light, in cases of medium severity the patient can just distinguish day from night.

* The word "toxic," here and throughout, refers to "visual toxicity," if I may be allowed to coin a clumsy but expressive phrase.

On examination during this stage the pupils are found widely dilated and irresponsive to light, anæsthesia of the cornea and conjunctiva is occasionally present; in one recorded case there was increased tension.[10] Nystagmus is noted as an occasional concomitant by several writers; I have seen this



Fields in case described by the writer in Journal of Sept. 15.

The eccentric continuous line indicates the average normal field of indirect vision.

curious symptom (rotatory) in one case. The media are clear; Geschwind [11] described vitreous opacities in one case of quinine amaurosis, but it is doubtful if they were attributable to quinine. The fundus changes are well marked and characteristic: the disc is of a dead whiteness; the retinal vessels reduced to threads; the fundus generally pale. Occasionally the macula stands out vividly as a red spot, and the appearances simulate those of embolism of the central artery.

The stage of complete amaurosis varies in duration from a few hours to several weeks; only one case of permanent blindness has been recorded.[12]

The return of vision is slow, and in too many cases very incomplete, contracted fields and impaired light and colour sense remaining permanently. Where amaurosis has been complete some impairment of vision always remains; and even in cases of moderate severity some contraction of the fields and diminution of light-sense are apt to persist, though central vision has been completely restored. Where there is permanent visual disability, pallor of the discs and contraction of the vessels remain to a greater or less extent.

The contraction of the field tends to assume the shape of an ellipse with the long diameter horizontal (Knapp). In very severe cases—as in the case narrated by me in the Journal of September 15—the fields are so reduced that only "telescopic" vision remains.

Diagnosis and Prognosis.

The ophthalmoscopic appearances sometimes closely resemble those of advanced optic atrophy, occasionally those of embolism. Taken in connection with the history and symptoms they are so characteristic that an error in diagnosis should not occur. The points of resemblance between quinine amaurosis and any of the malarial eye affections are few, but of course it may occur in eyes already injured by malaria.

Prognosis is on the whole good as regards the restoration of central vision; but some—it may be slight—permanent contraction of field and diminution of light perception may nearly always be expected. The liability to relapse on administering quinine, even in small doses, should never be forgotten.

Pathology.

Thanks to the brilliant experimental researches of de Schweinitz and Holden, the pathological anatomy of quinine amaurosis has been worked out with remarkable precision and completeness. Owing to considerations of space, only a brief summary of the results arrived at can be given.

De Schweinitz produced quinine blindness in dogs, noted the exact reproduction of the clinical picture in human beings, and finally submitted, at different stages of the blindness, the optic nerves, chiasms, tracts, and visual centres to microscopic examination. The result of this examination is best given in his own words:—

"In *résumé* I may say in regard to the microscopical appearances that there are thickening and changes in the walls of the optic-nerve vessels (endovasculitis); organisation of a clot, the result of thrombosis, an organisation which has been carried on even to the extent of its being channelled by new vessels; widening of the infundibulum (?) of the vessels as the result of the constriction of the surrounding nerve-fibres, causing appearances not unlike a glaucomatous

excavation; and finally, practically complete atrophy of the visual path, including the optic nerves, optic chiasm, and optic tracts, as far as they could be traced.

"It seems, then, very likely that the original effect of quinine is upon the vaso-motor centres, producing constriction of the vessels; that finally changes in the vessels themselves are set up, owing to an endo-vasculitis; that thrombosis may occur, and that the result of all of these is an extensive atrophy of the visual tract." [13]

Quite recently Holden,[14] working on the same lines with new and improved methods, has been able to demonstrate that the changes in the optic nerves and tracts observed by De Schweinitz were secondary to changes in the retinæ. Retinæ examined on the 3rd day after toxic doses of quinine revealed changes in the ganglion-cells and in the nerve fibres; on the 9th day these changes were well marked. Not until the 17th day were changes in the optic nerves noticed —breaking down of the medullary sheaths of many fibres. By the 42nd and 47th days the ganglion-cell and nerve-fibre layers of the retinæ were almost gone, and the degeneration of the optic nerves could be traced up to the terminations of their fibres in the brain.

The following, therefore, would seem to be the order in which the pathological processes set up by a toxic dose of quinine manifest themselves:—

(1) Constriction of the retinal vessels.

(2) Breaking down of the less resistant layers of the retinæ as a result of the restricted blood-supply.

(3) An ascending atrophy of the optic nerves and tracts.

Treatment.

Quinine must be stopped, or, at all events, given very cautiously in greatly lessened doses, the moment symptoms of amaurosis appear. Given in the doses recommended by Dr. Manson, the drug is potent for good, and impotent for evil, except in rare cases of idiosyncrasy.

The temporary good effect of inhalations of nitrate of amyl in established cases is remarkable. I have had repeated opportunities of verifying it. A moment or two after the inhalation the discs become rosy, the fundus of normal colour, and the patient can perhaps read two lines of type lower than before. Unfortunately these effects are not permanent. Nitroglycerine, given every other day, combined with general tonic treatment, affords the best prospect of ultimate success in restoring, partially at any rate, the visual functions. The patient should if possible be removed from the malarious country in order to avoid the necessity of administering quinine.

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[1] See case related by the writer in the Journal of September 15.

[2] *Arch. f. Augenh.*, 1877, xxviii.

[3] *Inaug. Diss.*, Zurich, 1882.

[4] *Trans. Coll. of Phys.*, Philadelphia, Nov., 1890; *Trans. Am. Oph. Soc.*, 1891; and monograph on "Toxic Amplyopias."

[5] *American Oph. Soc.*, July, 1898.

[6] *Vide* Nettleship's case, *Trans. Oph. Soc. of United Kingdom*, vol. vii., p. 218.

[7] "Manual of Tropical Diseases," p. 112.

[8] *Vide* September number of Journal, page 53.

[9] *Trans. Oph. Soc. of United Kingdom*, vol. vii., p. 193.

[10] Tiffany, *Rec. d'Ophtal.*, 1890, 3 ser., xii.

[11] *Arch. de Méd. et pharm. militaires*, 1892, xix.

[12] Claiborne, *N. Y. Med. Journ.*, 1894, lix., part 1.

[13] "Monograph on Toxic Amplyopias," Philadelphia, 1896.

[14] *Vide supra.*

Papers read at the Annual Meeting of the British Medical Association.

INSANITARY ENVIRONMENT AS THE CAUSE OF THE SPREAD OF YELLOW FEVER AND BUBONIC PLAGUE.

By Alexander Turnbull, M.D., R.N.,
Inspector General of Hospitals and Fleets.

As there are still not a few who look on yellow fever as a contagious or infectious disease, only to be arrested by a comparatively low temperature, or with the greatest difficulty in the tropics, I propose to submit some facts, from naval records and from my personal observation and inquiry, which appear to support, if indeed they do not establish, the view now generally accepted, that good sanitary environment is inimical to and preventive of the spread of this fatal disease in all climates.

The belief that a low temperature was requisite to arrest its spread on board ship was, if not now, the view of naval authorities, and involved a procedure tending to derange naval arrangements. The contagiousness or non-contagiousness of yellow fever was a belief of different medical men from the first recognition of the disease, and it is recorded that the advocates of each opinion have been known, in former days, to defend them by recourse to duelling, so fierce did the controversy rage.

Thus the non-contagious view is no novelty in our naval medical records. In 1818 Staff-Surgeon Veitch, R.N., for five years in charge of the Naval Hospital, Antigua, as also Acting Physician of the West Indian Fleet for other five years, by the desire of the Transport Office of the day, addressed a "Letter to the Commissioners for Transports and Sick and Wounded Seamen on the Non-Contagiousness of the Yellow Fever." He cites the case of the Master of H.M.S. "Regulus" in 1794 dying on board that ship at Port Royal, Jamaica, from yellow fever and black vomit, "but none of his messmates or attendants, from whom he received the most assiduous attention, were affected. A Lieutenant was, at a period considerably subsequent, attacked with the same disease, and was sent as a convalescent to the hospital at the Mole, but no extension of the fever to others in the gun room followed, a circumstance inconsistent with a contagious fever in so small a space. The same ship served in the West Indies from 1796 to 1799, during which period the yellow fever repeatedly appeared on board among the ship's company and officers, but it never exhibited the character of a contagious disease."

At the Royal Naval Hospital, Antigua, he states "he had ample opportunities of witnessing the character of this fever. . . . and the result of my

experiences and observations in that capacity leads me decidedly to state that the fever which destroys so rapidly the young, vigorous European, and on which the name of Bulam fever has been improperly imposed, is not contagious at Antigua, nor in any part of the West Indies."

At this time Chisholm, Inspector-General of the Ordnance Department in the West Indies, narrates that 13,437 soldiers, our own countrymen, in a period little exceeding thirty months, perished by malignant, pestilential, and yellow remittent fever, while Arthy, in *The Seaman's Medical Advocate*, attempts to show about this time that 5,000 seamen are *annually* lost to the British nation in the ships of war and merchant ships in the West Indies through yellow fever.

In reference to the introduction of yellow fever into England by H.M.S. "Eclair" in 1845, Sir William Pym, K.C.B., the Superintendent-General of Quarantine, asserted that it is a disease of warm climates, hitherto unknown in England, and imported by means of an artificial warm climate having been kept up during the voyage from Sierra Leone by the fires of that steamer, urging that under similar circumstances fires should be put out and the ship sail to the northward, and that the Admiralty should prevent such affected vessels arriving in England save between November 1 and March 1, a view strongly opposed, I need hardly say, by the Naval Medical Director-General at that time.

H.M. steam-sloop "Eclair," with a complement of 146 officers, seamen and marines, commissioned at Woolwich on August 26, 1844, and arrived at Sherboro, West Coast of Africa, on January 18, 1845. In February the general standard of health in the "Eclair" is reported to have been high. Between February 15 and March 16 her boats were employed exploring the creeks of the Sherboro and Sebar branches of that river, the crews sleeping sometimes on their shores, but generally in the boats. From 30 to 40, including two officers, were so employed for 7 days at a time.

The fever about this time was as follows:—

April 3, one seaman, 24 days, recovered; April 18, 3 seamen, all died on the 5th, 6th, and 7th day respectively of the fever, a fourth recovered; May 3, one seaman, 13 days, recovered; May 6, one seaman, recovered on the 3rd day; May 22, one private, R.M., died on the 7th day—he was also servant to the Clerk of the ship; on the same day a seaman was seized, and died on the 13th day; May 23, a stoker, died on the 4th day; May 26, a petty officer, died on the 9th day; on this date 3 seamen, deserters from another ship, who had been some time on shore, were taken ill; the fever, however, was milder in their case, strange to say, and they were discharged to hospital on the 37th day; May 29, one seaman, recovered on the 7th day—this was a relapse of the first case; June 4, a seaman died on the 3rd day, a second seaman recovered on the 42nd day; June 5, a private, R.M., died on the 6th day; June 8, the Clerk, who had become mentally affected by the loss of his servant, and from the first had a presentiment of death, death resulted on the 7th day.

Of the above, 12 had been boat-cruising in the malarious creeks of the river; 2, the stoker, who died on May 27, and the Clerk, had never left the ship.

The fever in these cases was undoubtedly remittent fever common to the West African rivers and coast, though in some cases of a severe type. Sir William Pym, Inspector-General of Army Hospitals and Superintendent-General of Quarantine, clearly points this out in 1845.

July 2, the "Eclair" proceeded to Sierra Leone; July 5, arrived at Sierra Leone at what was considered the unhealthy season of the year. The crew were now employed in clearing the holds and surveying the stores of H.M. iron steamship "Albert," which had proved a fairly healthy ship at Sierra Leone, where she had lain some time without any disturbance of her holds and stores, though the holds were now found to contain a considerable amount of offensive matter.

It was unfortunately decided to give the "Eclair's" crew leave to go ashore here, on the understanding they were not to remain on shore at night; in many cases, however, they did, some from one to three days straggling out into the country, and committing great irregularities from drunkenness, &c. July 23, the "Eclair" removed to an anchorage some distance off the coast, towing the "Albert" with her. Her crew were then engaged in clearing out, painting, and re-fitting the "Albert." On this date one case of fever was discharged to hospital and two remained on board.

While at this new anchorage 13 cases of fever occurred, with a mortality as follows: July 27, an ordinary seaman and a stoker; 28th, a gunner, Royal Marine Artillery; August 3, a ship's steward's mate; 6th, an able seaman; 7th, an ord. and a gr. R.M.A., a civilian passenger from Sierra Leone is also reported to have died on board from fever at this time. In the case of all the crew who died here they had slept on shore at Sierra Leone for one or more nights. We now learn for the first time of black vomit and a yellowness of the skin, and undoubtedly they were cases of yellow fever, distinct from the remittent fever occurring from service at and in the Sherboro. August 9, the "Eclair" proceeded with the "Albert" in tow to the "Gambia," and arrived there on the following day. August 17, the "Eclair" left the "Gambia," a ropemaker having died on the 15th, and an A.B. on the day of her departure. August 21, the "Eclair" anchored at Boà Vista, having touched at Goree on the passage, where the French authorities refused her pratique. A caulker had died on the 18th.

At Boà Vista, after inquiry by the Portuguese authorities and a resident English medical man, pratique was granted, all, including the medical officers of the "Eclair," the resident English and Portuguese medical men, agreeing that the fever was the malarious remittent fever of that coast, though of a severe type. August 23, an A.B. died. August 24, the hold of the ship was cleared out by her crew, and the water tanks removed. August 28, a quarter-master and a stk. died. August 30, a qr.-mr. and stk. died. On this day the local authorities, not having changed their view of the malarial character of the fever, granted permission to the Captain of the "Eclair" to land "all hands" from the ship. The crew were therefore landed at a fort on a small islet near the town, where the sick had separate accommodation from the rest

of the crew. The officers, gun-room, midshipmen, warrant and engineer, took a house for themselves and their servants in the town. Commander Estcourt resided at the house of the Queen's Commissary Judge in Boà Vista. The "Eclair" was now thoroughly cleared out, ventilated, coaled and watered by native labour, and is stated to have been found quite clean.

The mortality now increased. September 2, a Gr., R.M.A.; 3rd, the Captain's cook and a boy 2nd class; 4th, a naval cadet, a stk., and a boy 2nd class; 5th, a master's assistant and a leading stoker; 6th, the Paymaster, a stk., a boy 2nd class, and a private Royal Marine Light Infantry; 7th, an A.B. and Ord., a steward and a gr. R.M.A.; 8th, the Assistant-Surgeon and an A.B.; 9th, the chief cook; 10th, a stk. and a gr. R.M.A.; 11th, a boy 1st class; 12th, a boy 2nd class. September 11, Commander Escourt applied to his Senior Officer for a report from the Surgeons of the "Eclair" and "Growler" of their opinions on the steps they consider would be most conducive to the recovery of the health of the men and officers. On that day Commander Escourt was stricken with the fever.

September 13.—The Surgeons of the "Growler" and "Eclair" reported that "the most desirable measure to be adopted for the benefit of the ship's company is for the 'Eclair' to proceed immediately to England, or certainly as far as Madeira." On this date the officers and crew were re-embarked, including the sick.

Since landing on August 30, 37 cases of fever had occurred with a mortality of 25; the quarters provided for the crew are said to have been ill ventilated, and the sick accommodation latterly very crowded. No treatment appeared to benefit the sick. September 14, a stk. died, and on the 16th Commander Escourt. Here I desire to quote the Queen's Commissary Judge's report to the Earl of Aberdeen on this officer:

"Captain Escourt only lived two days after the 'Eclair' left Boà Vista on her voyage to Madeira and England. Calm, firm, and self-possessed under the most trying circumstances, he wholly devoted himself, during the last weeks of his life, to alleviate, as far as it was possible to do so, the sufferings of such men as were seized with fever. From an early hour of the morning to a late hour in the evening, barely allowing himself, in the interval, to take his meals, he passed day after day with the numerous sick in the hospital, cheering, consoling and supporting them. His language up to the day when he was taken ill was that of determination to perform his duty to the uttermost, leaving the event with perfect submission and resignation to the will of God. Such an example of entire self-devotion as was exhibited by Captain Escourt, so worthily followed as it was by Lieutenant Isaacson and Mr. Surgeon Maconchy on board the same ship, forms a real subject of consolation under the affliction which drew it forth." Both these officers in their turn forfeited their lives in their devotion to their stricken shipmates.

September 20.—The "Eclair" arrived at Madeira. The fever appeared to acquire additional malignancy as soon as the "Eclair" reached the open sea, and the cases became more numerous than before. Despite the now asserted contagiousness of the fever, it is recorded that several men volunteered from the "Growler" to join the "Eclair" ere she left Boà Vista.

The mortality was further increased. September 17, one A.B.; 18th, a Surgeon volunteer, a pvt. R.M.L.I.; 19th, a boy 1st cl.; 20th, an A.B.; September 19, "Eclair" arrived at Madeira. September 21, "Eclair" left Madeira.

The mortality: 21st, the Surgeon, a stk., and a gr. R.M.A.; 25th, a stk.; 26th, an ord.; 27th, one pvt. R.M.L.I.; September 28, the "Eclair" arrived at the Motherbank, inside the Isle of Wight; a gr. R.M.A. died on this day; 29th, a chief steward; 30th, an ord.

The Surgeon who joined the ship at Madeira describes the fever as he saw it: "Intense frontal headache, severe lumbar pain, in six hours vomiting of greenish fluid with epigastric pain, late on second day brownish flocculi might be detected in vomit, which increased on the third day, with coldness of the extremities, delirium and death."

The Kroomen on board "Eclair" employed as attendants on the sick did not suffer; their immunity is characteristic of the resistance of black races to yellow fever, which is however almost invariably contracted, in the first instance, by the white man after association in some way with the black man, his habitations, &c.

The experience of the natives of Boà Vista, however, was very different. The "Eclair" left Boà Vista on September 13, as stated, after re-embarking her officers and men, the sick included.

The British Consul subsequently reports, on December 22, that on September 20, seven days after the "Eclair" sailed, one of the white Portuguese soldiers, associated on the small islet with the crew of the "Eclair," had died of fever in the fort; a second also died, and the remaining soldier in the fort, a coloured man, was reported sick; another coloured man was sent to assist his comrade, who being also taken sick, the authorities at once abandoned the fort and islet, and caused the two sick men to be brought to the town, and lodged in a house near the sea beach.

He further records that at this time extraordinary heat prevailed and rain fell; the fever spread, it was said, from the house where the two coloured men were treated and recovered.

At the date of his report 250 had died, no Portuguese resident of any note had suffered, but the British residents lost a third of their number; the fever prevailed mostly among the lower orders of the natives, the leading symptoms were black vomit and suppression of urine (strangely unnoticed in the reports of the medical officers of the "Eclair"); the nurses of the sick were affected; this was unexceptional.

On March 9, 1846, he reports that the sickness still continues in the villages of the interior of the island and that the deaths amounted to 400.

Dr. Lind, in 1779, reported Boà Vista Island as especially injurious to strangers; and Dr. Stewart, R.N., subsequently Inspector-General of the Royal Hospital, Plymouth, in his report on the "Eclair" fever, states that yellow fever existed in the adjoining Island of Porto Praya whilst the "Eclair" was at Boà Vista.

To return to the record of the fever on board the "Eclair" on arrival at the Motherbank.

September 28.—The Surgeon, owing to the great depression on board, recommended all should be landed to prevent further spread of the disease; and the Inspector-General of Haslar Hospital desired that the sick should be transferred to that establishment and isolated in the airy wards there. The quarantine authorities, however, considering only the commercial interests of the country, refused both propositions, and ordered the removal of the ship, with sick and healthy on board, to the Foul Bill Quarantine Station at Sandgate Creek, in the Medway, opposite the present Port Victoria.

October 1.—The "Eclair" proceeded to Sandgate and arrived there on the 2nd in charge of a pilot, embarked on the previous day.

October 3.—The pilot and such officers and men as had not had fever were transferred to the "Revenge," the convalescents to the "Benbow," both line-of-battle-ship hulks at Sandgate; the sick, medical officers and Kroomen remained on board the "Eclair" until the 8th, when all who could be moved were transferred to the "Worcester" frigate hulk.

Mortality.—October 3: a master-at-arms, a blacksmith and a pvt. R.M.L.I.; 6th, a corporal, R.M.A.; 9th, Surgeon Barnard, who had volunteered for the ship at Madeira; 11th, the pilot who had embarked at the Motherbank; 12th, Lieutenant Isaacson, whose devotion to the sick at Boà Vista, and subsequently has been already mentioned, he, strange to say, was the last victim.

Assistant-Surgeon Rogers having volunteered for service in the "Eclair" and joined her on October 5, was attacked on the 10th, but fortunately recovered.

In recording the mortality and furnishing the rank and rating of the fatal cases, after the appearance of the disease on board at Sierra Leone, it will be observed, the remarkable exemption of the warrant officers and the engineers, none of whom perished. There is no evidence as to whether any of them were attacked at any time; the stokers, however, lost nine of their number out of fourteen.

Six medical officers contracted the fever; of these but two recovered. After the removal of the sick from the "Eclair" on October 8, she was thoroughly cleared by Kroomen supervised by three white men, two of whom and five Kroomen are reported to have had slight febrile attacks while so employed.

The total deaths from yellow fever from September 5, after arrival at Sierra Leone, were 63, exclusive of the passenger, and with the 12 fatal cases of remittent fever at Sherboro, gives a grand total of 75 deaths, or a little more than half of the ship's complement; 79 survived on October 31, when they were liberated from quarantine. Though the microbe of yellow fever may not yet have been authoritatively determined, its existence may be assumed, and that the soil must be congenial for its development. From Dr. Stewart's report on the "Eclair," though the main or berth deck may be considered as having been fairly ventilated and spacious, in proportion to the number of the crew, he states the lower deck was badly ventilated, as also the store rooms, a serious matter in such a climate as that of the West Coast of Africa, and an insanitary environment may be fairly assumed as the cause of the spread of yellow fever in this historic outbreak.

At the time of the "Eclair's" yellow fever experiences, the squadron employed on the West African station had not been visited with any unusual sickness.

In H.M. steam-ship "Growler," however, which joined the "Eclair" at Boà Vista on September 7, communicated with her there, and accompanied her to England, arriving at Plymouth on the 30th of the same month, there was evidence of a tendency to the same fever to develope and spread, arrested doubtless by the return to England, though two fatal cases developed in her, as in the "Eclair," in English waters.

September 7. At Boà Vista, a Lieutenant, the Paymaster and the Clerk of the "Growler," surveyed the stores of the Paymaster of the "Eclair," who had died of yellow fever on the preceding day.

The Superintendent-General of quarantine quotes the following report from the surgeon of the "Growler" of the fever on board his ship: September 7, the Lieutenant; 11th, the Paymaster; 12th, the Clerk; 14th, two cases; 17th, two cases; 25th, one case; with no death; October 9, two cases; 11th, one case.

Two of the last three cases — seamen — were landed at the Naval Hospital, Woolwich, where the "Growler" was paying off, but not in quarantine; both died of yellow fever. The Medical Director-General of the Navy reports that on visiting the "Growler," he found these men had slept over the scuttle of the fore hold, which when the hold was disturbed emitted a most unpleasant smell; these men had doubtless been working in the hold by day and inhaling the effluvia therefrom at night. This striking instance of insanitary environment causing the spread of yellow fever, even in England in October, and its failure to spread in the sanitary wards of the hospital, strongly supports the view this paper is written to advocate.

Sanitary Environment a bar to the spread of Yellow Fever.

The Governor of the island of Barbadoes reported to the Minister for the Colonies:

"H.M.S. 'Dauntless' arrived at Barbadoes on Nov. 16, 1852, from St. Thomas, with yellow fever on board. The sick were landed in the General Hospital of St. Anne's, while the remainder of the crew were, as far as possible, landed on the island and encamped; as all could not be landed, fresh cases occurred on board the 'Dauntless' almost daily.

Nov. 17 to January 5.	Admitted.	Died.	Remained.
Officers ...	22 ...	13 ...	2
Men ...	135 ...	52 ...	34

"Sixty-two of the 'Dauntless' yellow fever cases were treated in the wards of the 34th Regiment's hospital, and indiscriminately mingled with the soldiers of that corps affected with various complaints. In no instance has any individual been attacked under such circumstances, nor has any hospital attendant suffered."

"Authorised by J. H. MUNRO, Insp.-Genl." "W. DUMY. Surgeon, 34th Regt."

The Governor of Barbadoes further reports:

"Not a single case of fever, either in the garrison or city, can be traced to the ship or to her crew."

He further dwells on sanitation as a preventitive of the spread, and unsanitary conditions as the occasion of the spread of yellow fever.

The Captain of the "Dauntless" also attributed the landing of the crew as their safety.

I have been recently informed that at the present time such views are not held at Grenada, W. I., but that the yellow fever patients are still quarantined in the ships in which they reach this British colony.

H.M.S. "Highflyer," having contracted yellow fever at St. Thomas, arrived at Port Royal, Jamaica, on December 23, 1852. Sick sent to the Naval Hospital, 13 officers and 38 men; there they mingled with the other patients, at the same time pratique was granted to the ship, and the officers and crew had free intercourse with the colonists, who, however, were advised not to visit the ship. "No single instance of any kind of fever followed."

J. WATSON, *Surgeon-in-charge.*

This naval medical officer on reporting on this procedure, remarks: "His lengthened experience of yellow fever causes him to state, without dogmatically asserting that yellow fever is never under any circumstances contagious, any contagious powers the disease may possess in crowded ships is speedily rendered inoperative by moderately good ventilation, and that the best mode of checking the progress of such diseases is to remove the sick to roomy quarters on shore as speedily as possible."

H.M.S. "Esk" arrived at Nassau November 7, 1853, from St. Thomas, with eight cases of yellow fever, seven of whom had black vomit; the sick were landed in hospital and residences, while unrestricted communication by the colonists with the sick and the ship was permitted. The surgeon of the ship and the medical men ashore reported: "There is no instance of any one person having derived the slightest ailment whatever from the introduction of the eight cases into the port of Nassau, or from the six landed and treated in the town."

As a contrast to the spread of yellow fever in the "Eclair" and "Growler," the experience of H.M.S. "Bristol," a frigate, on the West Coast of Africa in 1865, is now recorded.

December 28 and 29, 1865, H.M.S. "Bristol" at Sierra Leone sent a working party of 112, including two officers, to H.M. store-ship "Isis" to unmoor her, and shift her anchorage, the "Isis" being extremely unhealthy, as evidenced by the mortality on board, more especially among fresh arrivals from England embarking on board her.

The working party did not sleep on board the "Isis," but returned to the "Bristol" at 9 p.m. and 10 p.m. respectively, on the days employed as stated.

An officer of the "Bristol" visited Sierra Leone on December 26, and slept on board the "Isis:" he contracted fever, but no officer or man of the "Bristol" who did not visit the "Isis," and not one of the crew of that ship who attended on the yellow-fever patients treated on board the "Bristol" was affected.

In the "Statistical Report of the Health of the Navy" for the period 1866, this outbreak of yellow fever is thus commented on: "The extraordinary fact that no one was attacked in this epidemic who had not at one time or other, been on board the 'Isis,' and that in no instance did the disease spread to the medical officers, or to the attendants on the sick, might reasonably create a doubt as to the specific character of the fever. That the disease, however, was specific yellow fever appears to be clearly indicated by the peculiar and Protean character of the symptoms by which the cases were distinguished, and were just such as occur in extensive epidemics of that fatal disease, and that such marvellous exemption from infection should be granted to those who were in constant attendance on the sick, and to the ship's company who had not visited the 'Isis,' as well indeed to many who had, is only one of those puzzling phenomena which are so liable to occur in all epedemics. Those who did contract the disease did so, however, from a notoriously infected ship."

The medical officer of the "Bristol," in his report, had stated, in reference to the immunity of the attendants upon the sick, and the crew who had not visited the Isis, "the distinction between true specific yellow fever and malarious yellow fever is hardly well established." The idea of a specific contagious yellow fever and a malarious non-contagious yellow fever was prevalent in the Navy at that time. When, however, the so-called specific contagious yellow-fever of the "Eclair" and the malarious non-contagious fever of the "Bristol" are recognised as both to have originated at Sierra Leone and the symptoms to have been similar, it may be asserted the disease was identical in both cases—yellow fever—spreading in the "Egeria" owing to insanitary environment, but not in the "Bristol" with good sanitation.

My observations at Rio de Janeiro in 1869-70, and at Buenos Ayres in 1871, I maintain, support the view advocated in this paper, one so generally accepted at the present time. H.M.S. "Egmont," line-of-battle ship and stationed at Rio de Janeiro as a store ship, was docked at Cobras Island on December 4, 1869. The dock was cut off from the sea breeze, the ventilation of the ship was virtually impossible, an adjoining dock was full of stagnant water, while at a short distance the then existing city sewers emptied into the inner or merchant-ship harbour, likewise cut off from the sea breezes, but open to the night breezes from the low-lying marsh grounds in the vicinity. Yellow fever was at this time prevalent in the city, near this harbour and dock, and among the merchant shipping anchored there these crews suffering greatly from this fatal disease.

Prior to the "Egmont" being docked, with her crew on board, working parties from H.M.S. "Pylades" had discharged her stores, and were berthed on board the store ship during the operation, but returned to their own ship before the "Egmont" was docked; no sickness occurred among these men.

December 10.—"Egmont" in dock, two cases of fever; on the 11th, one; all recovered. 15th, one case of yellow fever, fatal on fourth day; another case of fever recovered on the sixteenth day; 17th, a case of fever, twenty-nine days sick.

December 18.—"Egmont" removed from dock to

Euchadas Island, a superior anchorage from a sanitary point of view; here her crew were employed in embarking the stores previously landed.

December 20.—One case of yellow fever, fatal on the fourth day.

January 9.—A case of so-called "bilious remittent fever," discharged to duty on the forty-first day; 12th, a case of yellow fever, fatal on the fourth day; 13th, a case of fever recovered on the fourth day.

January 15.—I was a member of a medical survey on the case of yellow fever entered on the 12th; the surveying officers reported that the fever was "malarial yellow fever" (a legitimate view it was considered at that time) "due to local causes, and imperatively demanded the "Egmont's" removal to the outer harbour in the full force of the tide and open to the sea breeze."

January 16.—"Egmont" moved to her customary moorings in the outer harbour.

January 30.—A case of yellow fever occurred; but as I had left Rio de Janeiro, I am unable to state whether the disease was contracted in the ship or in the city when on leave, nor can I report the result in this case.

In 1874 I revisited Rio de Janeiro, and found that, in consequence of an outbreak of yellow fever on board H.M.S. Egmont, the crew had been landed and sent to the hills. She was never again manned, but sold. The ship had doubtless become insanitary as a wooden ship long moored in this tropical harbour.

It is noteworthy how the yellow fever abated as the ship moved from the insanitary dock where it originated to more sanitary anchorages.

About this time I heard of a man-of-war sloop and some mail steamers contracting a case of yellow fever at Rio de Janeiro, which, though treated on board, caused no spread of the disease among the crews or passengers, though all these vessels proceded north in the tropics. My inquiries at this time among medical men afloat and in the city failed to satisfy me that yellow fever was contagious, and few, if any, of my informers considered it was so.

Buenos Ayres Yellow Fever Epidemic, 1871.

I was stationed at Buenos Ayres in a gun vessel when this epidemic began, but as the vessel was ordered away, my information regarding this apalling visitation, entailing a mortality variously estimated from 13,000 to 26,000, and officially returned as 13,614, was obtained subsequently, though in the same year, and from a medical man and others who had been in the city during the epidemic.

An asserted reliable local newspaper stated that in April of that year, when, owing to the general exodus of the inhabitants, but 30,000 remained in the city, the mortality was 9,200. As regards insanitary environment as the cause of the spread of the disease in the city of Buenos Ayres, it is only necessary to mention that at that date the city was devoid of drains and sewers, and as an old city honeycombed with cess pits, the usage having been when cess pits filled to cover them over and dig another one, the rain-water reservoirs for household supply adjoined these cess pits, while cess pits and rain-water reservoirs were both situated in the *patio* or court around which the residence was built, and even underneath the houses.

Yellow fever was reported to have spread from one imported case of the disease, the patient having evaded quarantine and entered on residence in the city.

Though the fever was apparently "contagious" in the city among whites and blacks, all the unaffected who left the city of an evening and slept in the open country in the vicinity escaped the disease, though climate, altitude and temperature were the same as in the city. The environment was sanitary, however.

The British Consul informed me that, though the captains with their boats' crews from the vast shipping anchored off the city, in the full force of the tide, visited the city by day during the epidemic, not one case of the disease occurred on board the British shipping, a striking contrast to the great mortality from yellow fever among similar shipping in the closed-in, sewage-saturated merchant-ship anchorage at Rio de Janeiro.

In the same year yellow fever spread to the riverine towns on the banks of the Parana, Paraguay and Uruguay Rivers, but it was observed that though yellow-fever-stricken patients from these towns might move to the open country and die there, the disease did not spread as in the cess-pitted towns.

I understand the immunity long enjoyed at Newcastle, Jamaica and Petropolis, and St. Paulo, Brazil (all hill stations) has not proved permanent, insanitary conditions having been established in the mean time.

Prior to the introduction of yellow fever into Rio de Janeiro in 1847, that harbour and its surroundings were considered quite healthy, and naval officers indulged in shooting and fishing expeditions with impunity.

Bubonic Plague.—The experience of the Hong Kong outbreaks evidently proves that good sanitary environment is protective, and that the only Europeans affected there were more or less intimately connected with the Chinese victims, their insanitary dwellings and surroundings.

It would therefore appear that yellow fever and bubonic plague differ as to their spread from water-borne cholera and enteric fever and from the infectiousness of variola and scarlatina, though all such diseases are aggravated and intensified by insanitary environment.

At the Congress of the British Association, M. D. Morris, Director of the Botanical Gardens, Kew, has directed attention to the peculiar qualities of the wild Tamarind—the Jumbai, a tree of the East Indies. This plant, though excellent food for animals, has the remarkable property of causing the coats of animals feeding upon it to fall off, and in particular the manes and tails of horses. If the animals are given a change of food their coats grow again, but are neither of the same colour nor texture as formerly. Sometimes the disease attacks the hoofs, but the general health is unimpaired. Ruminants are not affected by the action of the Jumbai, the active principle of which is supposed to reside in the leaves and fruit.

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NOVEMBER, 1898.

THE PLAGUE COMMISSION FOR INDIA.

THE Government is to be congratulated on the excellent nominations which have been made to form the scientific part of the Commission to investigate the plague in India. The medical men selected are: Dr. T. R. Frazer, Professor of Materia Medica and Clinical Medicine, University of Edinburgh; Dr. M. A. Ruffer, President of the Sanitary, Maritime and Quarantine Council, Alexandria; and Dr. A. E. Wright, Professor of Pathology at the Army Medical School, Netley. On arrival in India, towards the end of November, they will be joined by Messrs. J. R. Hewett and A. Cumine, two eminent Indian civil servants familiar with the language and administration of the country. There is no mention of one or more medical men in India joining the Commission. We presume that this serious omission will be rectified later. The choice of the English medical members of the Commission will give great satisfaction to those who are anxious that the plague in India shall be thoroughly studied. Professor Frazer, by his valuable researches, is peculiarly fitted to bring a sound judgment to bear on the problems connected with the serum treatment of plague, and the anti-plague prophylactic; he is ably seconded by Professor Wright in this respect; and Dr. Ruffer's experience in bacteriology as former Director of the British Institute of Preventive Medicine will not only add strength to his colleagues' investigation into Haffkine's system, but will also ensure attention being paid to the plague bacillus in nature apart from man.

We have mentioned the omission of any medical men in India being placed on the Commission. We cannot but think that the omission is only of a temporary nature, the reason being that the Commission is not yet complete in its numbers. There can be no doubt that it would be a decided advantage if one, at least, of the Sanitary Commissioners of India were added to the Commission. Acquainted with the private habits and social customs of the people more intimately and thoroughly than any civilian, a Sanitary Commissioner of long experience and eminence, such as Dr. King of Madras, would bring a ripe experience to bear on the several questions arising, which would be of the utmost value to a Commission in India in an investigation of this kind. No one in India comes so closely in contact with the people in their everyday life and domestic affairs as the Sanitary Commissioner. In the early part of his career, first as a medical practitioner among the natives of India, he learns more of their private life and customs, idiosyncrasies, and constitution, than any one else; and later, as Sanitary Commissioner, he adds to this useful experience a practical knowledge of the medical history of the country as well as of the effect of climate and soil on the inhabitants and on their diseases. In an enquiry of such importance as that which is about to be entered upon, the problems to be solved require to be approached from many sides, and the more carefully it is arranged that this shall be the case the more likely is the investigation to be successful. The Commission would indeed have been stronger if some Medical Officer of the Local Government Board or Medical Officer of Health, trained in the investigation of outbreaks of disease, had been also appointed, for then,

in addition to the aspects now fully represented, the epidemiology of plague and all the other circumstances familiar to such an officer appertaining to an infectious disease would be sure to be fully investigated, and not overlooked.

It is with satisfaction we see that on this Commission, notwithstanding the omissions mentioned, the medical men have been assigned their true position. Professor Frazer will be President, and the Commission will not be one on which laymen preponderate, thus reversing the extraordinary composition of the Plague Commissions and Plague Boards with which India has hitherto been too familiar, and the results of which have for a long time past emphasised the importance of the appointment of a strong scientific commission which shall enjoy the confidence of all, and which shall be likely to assist the authorities in solving what has now become the most serious problem in India.

OVERCROWDING IN INDIA.

THE population in India increases at so rapid a rate that, according to the Secretary of State for India, the question of overcrowding, of even large tracts of country, has come to be a problem of vital importance. During the ten years from 1881 to 1891 the census returns show an addition to the population of India of 27½ millions, or well nigh an increase equal to the population of England. The security of life and property under British rule no doubt has had an important bearing on this increase, and the very success of the system of government seems likely to cause a difficulty to the administrators of the future.

In Lower Bengal the immensity of the population in proportion to the soil has, ever since we have known India been astounding. But when we consider that in this district alone, in ten years, 4½ millions, or a population equal to the whole of Scotland, were added to the numbers, it is evident that a limit to the productiveness of the soil to sustain the increase will soon be, if it has not already been, reached.

In several districts of Bengal the density of the population exceeds 900 to the square mile, and the land, according to Hindoo custom, has been so minutely parcelled out that it is beyond the capacity of the soil to even maintain the population at the "verge of starvation" at which they chronically exist.

No doubt security of property contributes towards this state of things, but, in addition, British rule has diminished the death rate by reducing the mortality from famine and disease. Cholera, the former scourge of India, has lost many of its terrors of late years, and by controlling its spread, and limiting its ravages, the mortality has been greatly diminished. As with cholera, so with many other epidemic diseases—plague, small-pox, typhus, famine fever, &c. This hygienic fight is still steadily going on, and the result is a continuous and continued increase of population, more especially in those districts which already teem with people.

The alleviation for this congested state is to be met with by emigration. But whither? The Malay Peninsula and the Islands of the Pacific Archipelago are already well stocked, and it is difficult to see where fresh ground is to be opened up. Assam, however, is but sparsely populated. Burmah can supply food for a population far in excess of that which it at present supports. The climate of both these countries should suit the Hindoo, and if it were not for the great difficulty of persuading the natives of India to emigrate there might be hopes of relieving the strain by encouraging emigration thither. The persistence with which the natives of India, especially the poorer and more ignorant, cling to their rice patch is proverbial, and it will require a mission imbued with an attractive programme indeed to bring about an end so desirable. Still, it can be encouraged; the readiness with which male emigrants proceed to the African coast as coolies, under British supervision, might be taken as a sign of possible success, and if the women of India could be brought to see that their conditions of life would be bettered, say, in Assam or Burmah, at least a temporary outlet would be found whereby the congested areas of India might be relieved.

A POST-GRADUATE UNIVERSITY FOR INDIA.

Mr. J. N. TATA'S princely offer to endow an Institute in India for higher scientific and technical teaching and research has naturally attracted much attention, and has occasioned a good deal of discussion as to the best means by which such a project may be carried out. Among the numerous schemes propounded, Dr. Choksy, whose distinguished services in connection with the plague epidemic are well known, advocates in the *Times* of India a post-graduate University for India. In a graphic manner he points out the effects of famine and plague, the former having heavily burdened the peasantry with debt, and the latter by its devastations having dislocated commerce, trade and industry. History, archæology and other advanced studies in higher art, he contends, will not help to regenerate the country, nor add to India's industries, nor revive those that are in their decadence. Salvation, he believes, however, is likely to be obtained by the establishment of an institute which shall have for its aim, firstly, the study of scientific and practical sanitation; secondly, the investigation of Indian diseases; thirdly, the investigation of Indian drugs and their practical application to medicine; and fourthly, the study of applied chemistry in arts, industries and science, and agricultural chemistry. His reasons are that practical sanitation, sanitary knowledge, and sanitary wants are but imperfectly known and injudiciously, unscientifically and improperly applied; that Indian diseases, which annually carry away vast numbers of the population, and others which impair its stamina, remain unstudied; that the large storehouse of Indian flora capable of yielding indigenous medicines of undoubted value is an almost untrodden field; that the mineral resources of the country, its rare products, and their practical application require development, and that vast agricultural problems which chemical science can alone solve are awaiting research. The Indian Medical Congress said much the same thing in 1894, in the form of a number of resolutions forwarded on behalf of the Congress to the Government of India, so that Dr. Choksy has in support of his views an influential body of Native and European opinion.

Article for Discussion.

THE POSSIBILITY OF EUROPEANS AND THEIR FAMILIES BECOMING NATURALISED IN THE TROPICS.

I.

IN the December issue of this Journal will be published Professor Stokes' remarks on the theory that it is possible for Europeans to become naturalised so as to be capable of continuing their race in the tropics. This is a subject well worthy of discussion, for it strikes at the very root of colonisation. Generally, it may be stated that the present day belief is against the idea. It is held, perhaps erroneously, that the children of Europeans born and brought up in the tropics have not the same physical stamina as those reared in the temperate climate of Europe, and that it is well-nigh impossible for a third generation to attain maturity. A few facts, however, are worth a host of theories, and it is my purpose to induce medical practitioners in the tropics to help forward this argument by citing instances of survival, or the opposite. In entering upon this discussion it is necessary to be precise. The question appertains to continuous residence in the tropics, not to those who have been sent, when, say seven years of age to Europe, and kept there at school, or learning a trade or business until they are 18 or 20. Nor to those who every four or five years take a year's holiday to Europe, but to those who make the tropics their permanent dwelling place and whose children and children's children are reared there. As a preliminary let us consider the history of the migration of a few of the peoples we are acquainted with. The general tendency of an incoming race is to expand westward or eastward more than towards the north or south. That is to say, the spread is towards a region whose

climate is more or less similar to the parent country. The Mahommedan invasion spread almost directly eastwards, even to the shores of the Atlantic. The Hungarians, with all their wanderings, now east, now west, remained at much the same distance from the Equator. The Teutonic invasion crossed a strip of northern Europe including Germany, Holland, the British Isles and across the Atlantic to North America. Two offshoots of the race, however, went south, the Goths invaded Italy, but became by intercourse and marriage absorbed in the Latin races; and the Franks, although they gave their name to France, lost their racial characteristics by blending with the indigenous Celts. These are but movements in countries north of the tropics, but the migration of a northern people, to even the warmer climates and sunny plains of Italy and southern France proved disastrous to the race as a distinct race, for to the softer charms of more southerly influences the northern barbarians fell a prey and permanently settled in the land they over-ran.

In our own day we have several examples of European races seeking a home or dwelling place in the tropics. The Portuguese and Spaniards have given their languages and their religion to the people of a country reaching from Texas and California to Cape Horn. The whole continent of South America is an offshoot from the parent stocks in the Peninsula, and elsewhere, in many parts, the Portuguese more especially, have in Asia and Africa several tracts of settlements. But the inhabitants of these countries, although with Spanish (or Portuguese) blood in their veins, and with the language of these countries in their mouths, are not Spaniards. The red woman and the negress have contributed to the production of the Creole. This mixed race, no doubt, thrives, if continuance of the species is reckoned as a primary test of thriving. Elsewhere, also, the Portuguese and Spaniards, by intermarriage, produced races in America, in Africa, in India (at Goa), in China (at Macao), which became blended with the native, whether Red Indian, Negro, Hindoo or Chinese. The possibility of such a future for European emigrants at the present day is not the question. The problem is rather, is it possible to continue any of the European races "uncontaminated" in the tropics as a permanent dwelling place? The perpetuation of a mixed race has been proved.

At the present moment it is chiefly sections of the Anglo-Saxon family that afford us opportunities of studying the spread of European people towards the tropics. The United States of America, with its huge Anglo-Saxon population, reaches southwards until within 100 miles of the tropic of Cancer. Australia is crossed by the tropic of Capricorn, so that well nigh its northern half is within the tropical zone. A great part of the United States, therefore, is sub-tropical, whilst almost the whole of Australia is either tropical or sub-tropical. In these two countries Anglo-Saxons have taken up their abode and made them their abiding place. How are they faring?

J. C.

(To be continued.)

Recent Literature in Tropical Medicine.

OPHTHALMOLOGY IN ITS TROPICAL BEARINGS.

A Case of Male Filaria Loa.—Dr. Paul Bernard, chief of Landolt's clinic, describes a case of successful removal of a male filaria loa from the eye in the *Archives d'Ophtalmologie.*

A man, aged 31, presented himself complaining of a "worm" in his left eye. He stated that he had seen it himself that morning under the conjunctiva of the lower lid. Inquiry elicited the following history. Patient had lived in the Congo (at Talagonga on the Goë, 120 miles from the sea) from 1894 to 1896. A year after his arrival there he found he had filaria loa, the symptoms indicating the presence of the parasite being well known to almost everyone in that part of Africa. The first symptom he noticed was slight itching at the root of the nose, extending to the left upper eye-lid; later he often saw the worm, which moved quickly, appearing at one time under the skin of the lids, at another under the conjunctiva, but always in the left eye. It caused slight itching, but no swelling or pain, and on the whole gave little trouble. From time to time indurated swellings, the size of filberts, itching violently, appeared on his hands and arms, disappearing after a few days. Unsuccessful attempts to extract the worm from the eye had been made in the Congo and since his return to France.

On examining the eye Dr. Bernard found the conjunctiva of the lower lid and adjacent part of globe slightly injected; at the inner side of the globe, about 2 mm. from the cornea, a body like "a little vein" was noticed; a moment later it disappeared. After a few minutes the patient himself pointed out the worm at the outer angle of the eye under the skin of the upper lid. An attempt to seize it with forceps failed, the parasite again wriggling out of sight. After a short interval, however, it was again perceived in the middle of the upper eye-lid near the free border, where Dr. Bernard managed to catch it with a pair of forceps; he then made an incision over it, and by exercising great

care and making very gentle traction, was able to remove it whole.

The worm was examined by Professor Blanchard, who pronounced it to be a male filaria loa. It was 22 mm. long, pointed one end, obtuse the other, with cloacal papillæ—conforming in every respect to Manson's descriptions (*Trans. Ophthal. Soc. of Great Britain*, vol. xv., 1895, and "Manual of Tropical Diseases," 1898). It seems unfortunate that no examination of the patient's blood was made.

This is the second case on record of the removal of an intact male filaria loa from the eye, the other case being that described by Dr. Argyll Robertson (*Trans. Ophthal. Soc. of Great Britain*, xv., 1895).

Although filaria loa is common in West Africa, and well-known to the natives, the literature of the subject is scanty, and the life-history of the parasite has still to be worked out. Manson's conjecture that filaria diurna is the embryonic form of filaria loa—a conjecture based on the co-existence of the parasites in one case, and on the close resemblance between filaria nocturna and the sheathed embryos of the female filaria loa—remains only a conjecture, though undoubtedly a plausible one. Assuming the conjecture to be based on fact, we would still be ignorant of the mode by which the embryo escapes from the human body and of its life-history afterwards.

In a paper on "The Entozoa of the Eye," which will shortly appear, I hope to discuss fully this subject, and the subject of ocular parasites in general.

ITALY.

F. Rho. *Text-Book of Tropical Diseases. (Malattie predominanti nei Paesi caldi e temperati.)* Torino: Rosenberg & Sellier, 1897.

Rho's treatise on the diseases of warm climates is the first and only one that has appeared in the Italian medical literature of this century, and truly it is a masterly piece of work. Dr. Filippo Rho, now Principal Medical Officer in the Italian Navy, is a young and illustrious scientist who began his studies in tropical pathology in 1882, during a cruise round the world. His book is an octavo volume of 800 pages, printed in very small type and illustrated by several zincographs within the text and two plates. A work that covers so wide a field as the pathology of tropical and sub-tropical climates is necessarily a compilation and as such Dr. Rho's treatise is one of the best ever published. It is written in a clear and elegant style, it is throughout exhaustive, sound and up to date. It shows on the part of the author not only erudition, but a most admirable eclecticism. Rho's book, however, is more than a mere compilation, it is enriched with many original observations. The chapters on cholera, dysentery, and malaria are exceptionally good. There is only one chapter on which we differ greatly from the author. It is the ninth, which treats of non-malarious fevers. Dr. Rho very justly remarks that the study of the unclassified fevers of the tropics is an arduous one, but his way of disposing of it is to our mind very far from plausible. He states that the *climatic fevers* of British authors, the *frèvres inflammatoires* of French writers, *ardent fever*, *rock fever*, and other such fevers are some of them mild or abortive cases of typhoid fever, and others merely cases of autotoxis, which he believes to be far more frequent in tropical than in temperate climates. The ideas expressed in this chapter are strangely in contradiction to those which permeate the greater part of his work. Contrary to Dr. Rho, who would reduce the number of types, we believe that many very different diseases of tropical countries have been wrongly included in the few orthodox types. In this way we believe that the descriptions of already well differentiated diseases have been unfortunately obscured. The pathology of the tropics is surely not restricted to the few diseases which now occupy our text-books. Hundreds of diseases are doubtless as yet unknown. Since Lewis first described the *Filaria sanguinis hominis* already five filariæ have been differentiated in human blood. Since Laveran's discovery of the hematozoors of malaria already four varieties of malarial protozoa have been specified, and recent studies tend to show that the parasites of blackwater fever and Kala-Azar constitute possibly additional varieties.

Rho's treatise contains a chapter on the influence of tropical conditions on some of our commonest diseases, and another one on the surgical diseases of warm climates. The book closes with an excellent chapter on acclimatisation, in which the author sustains on very good grounds that the acclimatisation of Europeans in tropical climates is quite possible.

Rho's book was published last year, and therefore it does not contain the most recent studies on yellow fever, sunstroke, plague, blackwater fever and malaria. Tropical medicine for many years lay almost dormant. The noble works of Annesley, Morehead and Martin seemed to have exhausted all that could be said on the diseases of warm climates. But with the expansion and development of tropical colonies, with the wondrous advance of modern science, with the ever-increasing number of investigators, it soon became evident that we had but broached a mighty work. No branch of medicine has ever shown so swift, so amazing a progress as tropical medicine in the last few years. In the work under notice we have a striking illustration of this phenomenal progress. Rho's book was only published last year, and already it requires many additions and emendations. We hope we shall soon have to review a new edition of this admirable treatise, but even if it were to remain always in its present form, we should still strongly recommend it for the wealth and accuracy of its historical data, for the excellent distribution of its material, for the great clearness of its diction which make of it a model text-book.

AUSTRALIA, QUEENSLAND.

Pemphigus Contagiosus Tropicus.

In the *Australian Medical Gazette*, Dr. Hunter Finlay, of Townsville, draws attention to an outbreak of pemphigus contagiosus tropicus, This affection of the skin is similar to that met with in the far East, but it is the first time the disease has been recorded as occurring in Australia.

Dr. Hunter's description in no way differs from that of the typical disease, and it must be admitted that he has proved the existence of pemphigus contagiosus in Queensland. Dr. Hunter recommends a lotion of one part of liq. plumbi subacetat., to seven parts of distilled water, when the skin is irritated or inflamed; and a disinfectant ointment of acetanilid. and hyd. subchloridi aa. grs. v. to vaselin 1 oz.

Hirschfeldt. *On the Dengue Fever of Southern Queensland. (Inter-Colonial Medical Journal of Australia*, March, 1898.)

Hare. *The* 1897 *Epidemic of Dengue in North Queensland. (The Australasian Medical Gazette*, March, 1898.)

During the past four years Queensland has been swept from north to south by a series of epidemics of dengue fever. In the two papers under review the nature of the disease, together with many interesting points relating to symptomology and treatment are discussed with much minuteness.

The suddenness of the attack, the peculiar duration of the febrile period, the pains, the initial and terminal rashes, left no doubt as to the nature of the disorder. In the southern towns about 40 per cent. of the inhabitants were attacked, but in the north the incidence was heavier, scarcely 25 per cent. escaping. There were well marked differences in the type of disease as observed in the different localities and in many points the clinical phenomena differ from those recorded in the classic accounts of du Brun and Manson.

Hirschfeldt states that the incubation period cannot be placed at less than a fortnight, whilst Hare brings evidence to show that it does not extend beyond one week; similarly,

whilst the former is inclined to regard the immunity conferred as permanent, the latter fixes its duration at about two years. The remittent type, however, was by far the most common; in the account of the epidemic in the south the occurrence of intermittent cases is only briefly alluded to; in the northern territory they did not constitute 10 per cent. of the whole.

Termination by crisis was usual in Hare's cases, whilst Hirschfeldt found the lytical decline to be the more common. Other marked points of difference were the comparative freedom from desquamation in the south, and the constant presence of cardiac and pulse changes. Hirschfeldt devotes considerable space to the study of the circulatory changes, the most common being an absolute and relative bradycardia, and accentuation of the second sound over the mitral and tricuspid orifices. Far from being a mild and non-fatal disorder as described by Manson, the epidemic in 1897 in North Queensland was responsible for many deaths. Hare has collected accounts of sixty fatal cases, one half of which were adults. The direct cause of death was either rapid hyperpyrexia and coma, or more frequently sudden heart failure and collapse occurring at the period of crisis. The case mortality he thinks would be about 1 per cent. In the south the figure has been placed at 31 per cent.

Amongst other symptoms and complications not usually described in the text book accounts of the disease are nephritis, abortion, mania, and melancholia. It is noteworthy that both observers agree that swelling of the joint was exceptional, not occurring according to Hirschfeldt in more than 10 per cent. of the cases. Quinine and antipyrin were the drugs found most useful in treatment. The antipyretic action of lactophenin is spoken of as being longer sustained than that of other drugs, whilst it does not at the same time depress the action of the heart. Of the etiology of dengue fever practically nothing is known. Manson classes the disease with the exanthematous fevers, and suggests that the pathogenic organism is probably allied to those of scarlatina and measles. On the other hand the disease suggests in many ways an invasion by hæmatozoa. The author of the account of the epidemic in North Queensland records the fact that Dr. Hunt has made fresh blood preparations in thirty or forty cases and noted the presence of actively motile granules, similar bodies being also found in broth through which the breath of a dengue patient was made to pass. J. Odery Symes.

GERMANY.

Analgen in Malaria.

In the *Keilkunde Monatsh. f. Pract. Med.*, Dr. Scognamiglio strongly recommends analgen in doses of 0·5 to 1·5 gm., to be given three times, two or three hours before the attacks of the quotidian type of malarial fever, and eight to ten hours before malarial fever of other varieties. Microscopic examinations of the blood showed destruction of the plasmodium malaria in twelve to sixteen hours after the administration of analgen. Dr. Scognamiglio considers the drug as the best substitute for quinine known.

History of Plague, from a Pamphlet on "Plague." By Dr. Scheube.

By the "plague" at the present time is meant a certain acute infectious illness, distinguished essentially by a severe affection of the lymphatic apparatus, and by acute inflammations of the external and internal lymphatic glands, leading to blood poisoning.

In olden times and during the middle ages the term "plague" was applied collectively to the most diverse epidemics, which spread rapidly and depopulated countries.

In consequence of this confusion of *plague* with other infectious diseases, the geography and history of the former are shrouded in obscurity. The oldest witness for the existence of this affection which we possess is in a medical excerpt of Oribasius, and is written by Rufus of Ephesus, a contemporary of the Emperor Trajan (98-117) and reports on the "pestilentes bubones maxime letales at acuti," which had already been observed 200 B.C. in Libya, Egypt, and Syria. It is questionable if the plague of the third century described by Cyprian was "Bubonic plague." Probably that, as also the epidemic in Athens at the period of the Peloponnesian War, described by Thucydides, was a mixture of various illnesses. It may be that plague played a part in these, but there is no proof. On the other hand, plague is undoubtedly described with its symptoms in the reports on the great epidemic which in the sixth century spread over almost the whole of Europe, and which, on account of its having taken place during the reign of Justinian (527-565), was called the Justinian Plague. The duration of the same was fifty or sixty years, and small-pox has been proved to have existed simultaneously. During the following years Europe was frequently visited by epidemics of plague, but none attained such dimensions as the terrible "Black Death" of the fourteenth century. Hecker computed the total of deaths in Europe from this epidemic as 25,000,000, that is the fourth part of the civilised world at that time.

From the end of the seventeenth century the plague became more rare in Europe, and since the middle of that century the west of Europe has not again been visited by it. The south-east parts of Europe, Turkey, Caucasus, South Russia, Italy, Dalmatia, Greece, and the Mediterranean Islands have occasionally been the scenes of epidemics during this century, the last of these being confined to Turkey. Since 1841 plague has died out even there entirely, except a small epidemic in Astrachan, the origin of which is unknown, which took place in 1876 to 1879.

Turkey forms, and has always undoubtedly formed, the bridge for the transmission of plague from the east into Europe.

Egypt is another country formerly visited by plague at intervals, and formed the source of the epidemics on the north coast of Africa, more especially Tripoli, Tunis, and Algeria. The plague has, however, not been seen in Egypt since 1844.

Nevertheless, plague has by no means disappeared from off the face of the earth. It is at home in Asia, which represents its *native place* (Hirsch says *India*), and until the most recent period appears endemically as well as epidemically; at the present time it is on its travels and it cannot be said where it may end. The history of plague in Asia cannot be traced back further than to the sixteenth century. Arabia, Mesopotamia, Persia, India, and S. China are to be looked on as the five endemic seats of the disease.

As regards Arabia, the west coast of the mountainous country, Assir, was visited by epidemics 1853, 1874, 1879, 1889, 1890, 1892, 1893 and 1895.

In Mesopotamia, in which between 1856 to 1865 a light form of the illness (bubonic fever) had reigned in limited areas, larger epidemics occurred in 1866-1867, 1873-1874, 1876-1878, 1881-1883, 1885-1886, 1890, 1891 and 1892.

In India, plague appeared for the first time during this century in 1815. According to the *Indian Lancet* (Nov. 1, 1897), the Mohammedan chronologists noted four great outbreaks in previous centuries, 1529, 1577, 1615 and 1658-1707. Since then (excepting the present epidemic) plague only occurred in the Himalayan provinces of Cumaon and Gharwal in the year 1846, 1876 and 1884.

In regard to South China, it was only in 1878 that we were made aware of a probably long standing seat of plague in the valley of the province of Yu-nan. Probably the present epidemic had its source here. After the illness had shown itself repeatedly in Lieu-schau and Pakhoi on the Gulf of Tongking, it was taken from the latter place to Canton in March, 1894, where within a few weeks it caused the mortality of 60,000 persons, and in May it also attacked Hongkong, and later on Amoy. In Hongkong, in three or four months, 2,500 persons fell victims to the plague. In 1895, Swatow, Foochow, Macao and many other places of South China were befallen, and in the following year it

reappeared in Canton, Hongkong, Macao, Swatow, Amoy, &c., and spread also to the Isle of Formosa.

In September, 1896, the plague broke out in Bombay, having been probably brought from China, and spread north and south over a large part of India. The principal centres of the epidemic were formed by Bombay, Karachi, and Poona. The plague reached its height in Bombay in February, 1897; in April there was a pause, and in July it seemed to be dying out. In August, however, it flickered up again, and inland had a great hold on the folks weakened by famine, &c.

The outbreak of the plague in India had a bad influence on trade, and on account of the European interests involved, the various States sent out scientific commissions to study the plague; amongst others Germany sent one out under Robert Koch. Japan had already sent one to Hongkong in 1894. Thus our knowledge of the plague (its ætiology, pathology and therapy) has been considerably augmented.

INDIA.

Treatment of Filaria Medinensis in the Human System by the Hypodermic Injection into the Parasite itself when possible, or into the part where it is situated, of a Solution of Hydrargyrum Perchloride.

By E. Mackenzie, M.D.
Manora, Karachi.

Cases arising from the presence of filaria medinensis are not common in Sinde, but having been an eye witness to the suffering and inconvenience caused by it in the Dharwar and Poona districts, I have always kept in view the settling upon some better line of treatment than the stereotyped poultices and fomentations and to wait patiently for the worm to be extruded. Poultices and fomentations are very good in their way, but do not cut the case short. I have tried several country remedies but with no better results. When the worm has been found superficial, good results have followed cutting upon it in its course, and holding it up over a probe, when by gentle and prolonged manipulation, with the aid of a lubricant, the worm slowly wriggles out or can be drawn out. Lately, however, in addition to this, when the worm has been broken and its situation is not well defined from swelling of the contiguous parts, I have injected into the swollen parts—over the site of the worm and into it when possible—a solution of hydrarg. perchlorid. grain ½ and repeated the operation every third day three times, and have met with complete success. The swelling subsides, the pain lessens and the worm is absorbed; the opening ceases to discharge, heals up and the patient can go about his work in less than a couple of weeks. In one case where the worm had just begun to emerge from under the skin in the thigh, two hypodermic injections into the part cured the patient in less than a week and he had not to lie up at all.

The solution used is the following:—

Hydrarg. Perchlorid	8 grs.
Sodium Chloride	8 grs.
Glycerine	3i
Spirits Rectified	3s
Distilled water added to make	3i of solution.

—*Indian Medical Record*, October 16, 1898.

SOUTH AFRICA.

Observations on Leprosy: Clinical and Pathological.

By R. Sinclair Black, M.B., C.M., D.P.H.

In the *South African Medical Journal*, Dr. Black, Medical Officer to the Robben Island Leper Station, states:—

"During the past year sixty *post-mortem* examinations on lepers have been made.

"The cause of death in the anæsthetic cases was in the great proportion of cases due to tuberculosis. The leprosy itself does not produce fatal lesions; but in the nodular cases phthisis was much less common; in fact, a severe case of nodular leprosy seems, as a rule, much less predisposed to tuberculosis than the anæsthetic form. Of course, cases are found where the unfortunate patient is attacked severely, both by the bacillus tuberculosis and the bacillus lepræ. In such an event the bacilli appear each to pursue their destructive tendencies independently, but it is the bacillus tuberculosis that first deals the mortal injury. In the purely nodular cases the lethal lesion seems to be chiefly in the kidneys, Bright's disease accounting for the majority of the deaths, with the associated condition of the liver and spleen producing slow dissolution from exhaustion. In this respect, therefore, the two types of leprosy show a distinct contrast.

"In nodular leprosy the internal organs most affected are the liver and spleen. From the literature on the subject I certainly was not prepared for the extensive lesions which are found in these organs. We are very apt to think of leprosy as a disease of integument, mucous membranes, and peripheral nerves; but such an idea is entirely erroneous. In every well marked case of nodular leprosy the liver and spleen are extensively involved. In such cases the liver is greatly enlarged, and its cut surface is seen to be covered with yellowish arborescent spots or mottlings; this is due to the leprotic infiltration. But even more striking than the condition of the liver is that of the spleen, the enlargement of which is often enormous. From the enormous quantity of bacilli in the liver when it is affected there is no doubt that bacilli and spores must find exit through the bile-duct, and thence by the bowel to the exterior. In the diarrhœa to which patients thus affected are very prone enormous quantities of infectious material must thus frequently be discharged. The bowel discharge of a leprous patient should thus be disinfected as carefully as that of a typhoid patient, just as his sputum should be treated like that of a phthisical patient."

Dr. Black tried the anti-toxin treatment of leprosy originated by Dr. Currasquilla of Colombia, South America, but without obtaining either ameliorative or curative effects.

DUTCH WEST INDIES.

In the *Geneeskundig Tijdschrift voor Nederlandsch Indie*, xxxvii., Dr. V. Dierin says: "It is not long since I saw Beri-beri patients on board a Norwegian ship (the men were mostly fed on rice, the treatment amongst the Norwegians is exceedingly bad)." This observation should be of great interest, as hitherto no case of Beri-beri had occurred on board a steamer in Europe.

The author does not say if the vessel was returning from a hot country, nor does he give any other particulars. We must, however, express surprise that Dr. V. Dierin did not seize the opportunity of showing these patients to some of his colleagues, who were in the habit of observing Beri-beri, in order to confirm this interesting fact. Nevertheless, Professor Stokvis affirms that this affection was not Beri-beri but acute ænemic hydropsy, which had been observed several times lately, and which might perhaps be attributed to insufficient food.

TURKEY.

Leprosy, Contagious and Hereditary. Von Düring, Constantinople.

In the *Deutsche Med. Wolheusch*, Nos. xx. and xxi., 1898, Von Düring states that after a study of the contagion of leprosy, he has come to the conclusion that leprosy is a contagious and infectious disease, but it is only communicable after prolonged contact.

In support of this statement he described two cases at the Berlin Leprosy Congress which came prominently under his notice. (1) One was that of a Greek girl who lived with a leprous Jewess and was intimately associated with her. In a few years the girl became leprous. (2) The other was that of a Greek whose wife after ten years of married life became leprous and from whom the man himself contracted leprosy, the disease declaring itself some four years later.

Von Düring affirms the following premises will be

universally admitted:—(1) That leprosy attacks man only; (2) that leprosy is spread from one human being to another; that it cannot arise spontaneously. He is a strong advocate for segregation, and totally disagrees with the step the Japanese are recommending, viz., to do away with segregation, isolation, and all prophylactic measures against leprosy.

Medical News.

THE COLONIAL MEDICAL SERVICE.

The Training of Candidates.

The following circular is sent from the Colonial Office to the Medical Schools:—

Downing Street, *November* 9, 1898.

Sir,—(1) In the fifth paragraph of the letter from this Department of March 11 last it was stated that Mr. Secretary Chamberlain, with a view to supplementing the instruction afforded by the Medical Schools, was endeavouring to make arrangements for giving to Colonial Medical Officers special clinical instruction in tropical medicine such as is given at Netley and Haslar in the case of medical officers of the Army, Navy, and Indian Medical Services, and which, from lack of the necessary material, cannot invariably be given at the Medical Schools.

(2) These arrangements have now been made. The Directors of the Seamen's Hospital, at the Albert Docks, which offers exceptional opportunities for studying cases of tropical disease, are providing the necessary buildings and teachers for the accommodation and instruction of the medical officers who may hereafter be selected by the Foreign Office and the Colonial Office for appointments in the tropics. A substantial contribution towards the initial cost of the buildings is being made by the Government, and it is hoped that, by October 1, 1899, it will be possible to receive Medical Officers at the hospital for purposes of instruction.

(3) It is proposed that, as is at present the case, candidates for medical appointments in the British Colonial Possessions shall be fully qualified before they can be put upon the Secretary of State's list, that from this list a certain number shall be selected annually to fill the vacancies which may occur in the Colonial Medical Service, that the selected candidates shall be trained for a period of at least two months at the Seamen's Hospital, and that they shall then be sent to the Colonies or Protectorates to which they have been allotted, where, when practicable, they will be attached, in the first instance, to the Headquarters' Hospital for the purpose of gaining additional experience. In estimating the respective merits of candidates on the Secretary of State's list, regard will be had to the fact whether or not they have already received instruction in tropical medicine.

(4) Judging from the replies which have been received from the General Medical Council and the Medical Schools, Mr. Chamberlain believes that the above arrangements will prove acceptable.

(5) Although the school at the Seamen's Hospital is designed for the training of medical officers for the Government Service, doubtless there will be many other medical men, such as the medical officers of missionary societies, and trading Corporations, and private practitioners who propose to settle in tropical countries, who will be glad to avail themselves of the advantages which such a school can offer.

(6) The Colonies are being asked to make pecuniary contributions, to collect pathological material for use in the school, and to support the scheme in every possible way. So far, then, as this department is concerned, no effort is being spared to make the school a success, and Mr. Chamberlain feels confident that the medical schools of this country will also do what is in their power to assist the development of an institution which is likely to be of general service, and to benefit medical science not only by giving a stimulus to the investigation of tropical disease, but also by qualifying a body of men to become investigators.

(7) Mr. Chamberlain is so impressed with the importance of this subject as affecting the administration and well-being of the tropical Colonies that, in addition to this scheme for providing a thoroughly efficient Colonial medical staff, he wishes to encourage by every means in his power scientific inquiry into the causes of tropical diseases. Accordingly, he has already, after correspondence with the Royal Society, instituted a Commission to study the subject of tropical malaria on the following lines:—

(8) The Royal Society has nominated two competent observers who have already proceeded to Italy for a short preliminary study, and will afterwards go to some place in Africa, probably, in the first instance, to Blantyre, in the British Central Africa Protectorate, where it has been ascertained that there exist good opportunities for carrying out the purpose in view.

(9) In addition the Secretary of State has nominated an experienced medical officer of the Colonial Service to aid in the investigation. This officer will, in the first place, proceed to India in order to study under Surgeon Major Ronald Ross for about two months, so as to make himself acquainted with the result of that gentleman's researches. He will then join the other two observers in Africa, where they will together pursue their studies, which will probably occupy about two years, and report from time to time to a Committee in England, nominated jointly by the Royal Society and the Secretary of State.

(10) Mr. Chamberlain has been glad to learn, from the replies which have been sent to the letter referred to above, that arrangements already exist, or are about to be made, for giving special instruction in tropical medicine in upwards of twelve British medical schools, and he trusts that these schools, some of which (such as that of University College, Liverpool; University College, Bristol; and the University of Durham College of Medicine at Newcastle-on-Tyne), being situated in large seaports, possess exceptional facilities for the study of tropical disease, will keep in correspondence with the School of Tropical Medicine at the Seamen's Hospital, with a view to mutual assistance and advice.

(11) In conclusion, Mr. Chamberlain desires to express his thanks to the General Medical Council and the British Medical Schools for the warm interest which they have taken in this matter, and for the ready and cordial support which they have afforded him. He will welcome any suggestions which may be made in furtherance of the object in view.

I am, Sir, your obedient servant,

Edward Wingfield.

Reviews.

Notes on Malaria in Connection with Meteorological Conditions at Sierra Leone. By Surgeon-Major E. M. Wilson, C.M.G. 2nd edition. London: H. K. Lewis. 1898. 16 pp., price 1s.

In this concisely written pamphlet Surgeon-Major Wilson gives the results of a six years' careful record of the relation prevailing between climate and West Coast malarial fever. He moreover compares the relative prevalence of malaria amongst white and coloured troops in the Sierra Leone command. It is to be gathered from his statements that white troops suffer more from malaria than the black, but the black are more liable to be affected by variations in the meteorological conditions than are the white. The writer comes to the conclusion by close observation, that the relative humidity of the atmosphere influences the prevalence of fever more than the amount of rainfall, and shows that the

smallest number of cases occur in the period of least rainfall, least relative humidity and greatest heat. Surgeon-Major Wilson declaims against the excess of meat diet served out to the soldiers, and recommends that vegetables be increased in amount. Amongst black, as amongst white troops, the greatest amount of sickness prevails during the first year of residence, and with each consecutive year of service the number of cases of fever diminishes. He even advocates that it would benefit the service were these seasoned soldiers allowed to re-enlist for a further period of service, rather than that freshly recruited men should be imported, amongst whom the same weeding out process by illness has to be again undergone.

News and Notes.

THE fact that the War Office authorities have asked several medical schools to nominate medical men to the Royal Medical Staff Corps without entering for the competitive examination, is causing and is likely to cause considerable comment upon, and discussion of, the procedure.

COMPANY PHARMACY IN BRAZIL.—In Brazil (Bahia) the commercial association of a doctor or surgeon with a pharmacist for the exploitation of the industry of pharmacy is prohibited, but silent partnerships are not included in the prohibition.

THE PENNY POST TO INDIA. — It is officially announced at Simla that one anna postage to England will commence on Christmas Day.

INVESTIGATION OF NATIVE MEDICINE.—The Natal Medical Council have set aside a sum of £50 to be devoted to the expenses incurred for chemical analyses of native drugs. This is a step which medical societies in the tropics generally might advantageously imitate. In the event of no local analyst being available for the work we are sure that the authorities at the Imperial Institute will be willing and ready to take up the work.

PLAGUE.—The recent returns from all parts of the Bombay Presidency as regards mortality from plague, show a decline. In the city of Bombay, the mortality for the week ending November 7 was 59, as compared with 200, 116 and 96, for the three weeks immediately preceding. The reported outbreak of plague near Samarkand in Central Asia, seems to be causing the Russian and Turkish Governments some uneasiness; for whilst the Turks have ordered their Eastern frontier to be strictly watched, the Russians have despatched forty doctors to the reputed seat of the outbreak. Russian movements are at all times so eminently political, that we would perhaps attach no great importance to the mission were it not from the fact that ten of the medical staff are lady doctors.

THE report in the Daily Papers that a death from Siberian Plague had occurred in a hospital in Warsaw, is sufficiently explained in the *British Medical Journal* of November 12, 1898. A Russian correspondent writes to the Journal to say that "Siberian Plague" signifies "Anthrax."

THE important announcement in connection with the School of Tropical Medicine, printed in this issue, will be read by all tropical residents with interest and satisfaction.

Correspondence.

HOW TO DESTROY MOSQUITO LARVÆ.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—In view of the strong evidence brought forward by Surgeon-Major R. Ross in support of Dr. Manson's mosquito theory of the spread of malaria, it is clear that if finally proved we must adopt our means of prevention to this theory. I have recently read in the *Journal of Public Health* (America) that the addition of a small quantity of permanganate of potash to the water of swamps, tanks, &c., will kill the larvæ of mosquitos by depriving them of the organic matter on which they feed. Therefore I have thought that use might be made of this to prevent the growth of malaria-spreading mosquitos. Our experience in India of the use of permanganate in disinfecting cholera-polluted water (introduced by Mr. Hankin, of Agra), is on the whole very favourable and depends upon the same principle. The method is not difficult nor expensive. I have already begun to make a trial in this jail, and I offer the suggestion, which is new as far as I know, for what it is worth to the readers of the JOURNAL OF TROPICAL MEDICINE.

Yours, &c.,
W. J. BUCHANAN, B.A., M.B.
Surgeon-Capt. I.M.S.

Central Jail, Dacca, Bengal,
Sept. 20, 1898.

IS THE INFECTION OF MAURITIUS IN 1866 WITH MALARIA A "MEDICAL MYTH?"

To the Editors of "The Journal of Tropical Medicine."

SIRS,—The story of the supposed infection of the island of Mauritius with malaria is well known. The arrival of the ship *Spunky*, carrying emigrants from India, is often credited with this deed. The question arose lately in connection with the spread of the "Burdwan," or Lower Bengal epidemic of the same period, into Assam (*vide* Rogers' *Kala Azar* Report), but the Sanitary Commissioner to the Government of India gives reasons for believing it to be, as he says, *a medical myth*. From the A.M.D. Report for 1866, it appears that *malarial fevers did exist in Mauritius previous* to this period, but did not affect the British troops there much, and were chiefly confined to natives of India. It is also said that what was called "bilious remittent" was endemic.

The outbreak of severe fever in Mauritius occurred at the same time as the Lower Bengal fever epidemic, and was very similar and very severe. It was apparently malarial, and controlled by quinine. The chief sufferers were Indian immigrants, who lived amid very insanitary surroundings, and their misery was aggravated by scarcity and high prices. At this period there was in the Island an extension of the cane cultivation, which led to deforestation of the land. The rains of 1865 had been very heavy, and the following season hot and dry, therefore all the causes for an intense malarial fever epidemic were present, assuming the pre-existence of the parasite, and the introduction of Indian infection is not needed to explain the occurrence. At the same time there appears to have been another disease prevalent, viz., typhus, called "Bombay fever" because it was chiefly found among immigrants from Bombay. Dr. Barrant showed that this disease was very like typhus, if not really that disease. It was continuous, lasting fourteen to twenty days, unaffected by season or by quinine. It may have been brought from Bombay.

If the above facts are correct, there must have been two epidemics co-existing in Mauritius: (1) malarial, probably produced in the Island under above conditions, and (2) typhus, probably imported from Bombay. The co-existence of these two diseases in the community probably gave rise to the idea of the infectivity of the supposed malarial cases. It is curious that cases of typhus also were supposed to be mixed up with the severe malarial cases of the great Lower Bengal epidemic (1865 to 1872). Those interested (who have a medical library handy) will find the following references give full accounts of these epidemics:—*Lancet*, August 3, 1867; December 7, 1867; February 29, 1868; A.M.D. Report, 1866; Sanitary Commissioner's (India) Report for 1896, p. 161. For the Lower Bengal epidemics, see Sanitary Commissioner's (Bengal) Reports for 1872, 1873, and 1874; L. Rogers' *Kala Azar* Report, 1897, and *Indian Medical Gazette*, November, 1897; L'averan, "Traité du Paludisme" (1898), p. 17, and authorities therein quoted.

Yours, &c.,
W. J. BUCHANAN, M.B.
Surgeon-Captain I.M.S.

Jail House, Dacca,
September, 1898.

Communications, Letters, &c., have been received from:—

A.—Dr. R. E. Adamson (Labnan).
C.—Mr. J. T. Clarke (Perah); Dr. Cunninghame (New Zealand); Capt. T. F. Clark, R.A.M.C. (Hong Kong).
D.—Dr. C. J. Davey (Transvaal).
F.—Major Fallow, R.A.M.C. (Mauritius); Mr. M. Foster (London).
H.—Sir W. H. Hutchinson, K.C.B. (Natal); Dr. H. Campbell Highet (Bangkok).
J.—Mr. A. E. Jerman (Uganda Protectorate); Major J. M. Jones, R.A.M.C. (Bombay).
M.—Capt. J. Mulvany (Bombay); Dr. Jenny C. Müller (Delhi).
P.—Dr. R. L. Price, R.N. (China Station).
R.—Mr. William Russell (Assam); Mr. C. Rose (Pubna); Dr. H. R. Robertson (Tientsin).
S.—Staff-Surg. Wm. Spry (Devonport).
T.—Dr. Ethel Tribe (Amoy).
W.—Major G. E. Weston, R.A.M.C. (Rajputana).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Liverpool Medico-Chirurgical Journal.
Pacific Medical Journal.
Public Health.
South African Medical Journal.
The Hospital.
The Therapist.
Treatment.

Births, Marriages and Deaths.

The charge for inserting announcements of Births, Marriages and Deaths, is 3s. The notice should be accompanied by a remittance.

BIRTHS.

EDWARDES.—On the 2nd inst., at Malabar Hill, Bombay, the wife of Stephen Meredyth Edwardes, I.C.S., of a daughter.

MARRIAGES.

PHELPS—HAMILTON.—On the 2nd inst., at Fyzabad, Oudh, India, by the Rev. Harry Menzies, Edwin Ashby Phelps, I.C.S., eldest son of the Rev. Philip Ashby Phelps, rector of St. John the Baptist's, Bristol, to Constance Isabella, daughter of the late R. St. George Harding Hamilton, Esq., 65th Foot, and step-daughter of Major F. G. Pollock, 7th Bengal Cavalry.

VON HAAST—YOUNG.—On November 8, at St. Michael's Church, Bournemouth, by the Rev. F. E. Teyne, vicar, George Augustus von Haast, second son of the late Sir Julius von Haast, K.C.M.G., F.R.S., to Maud Elizabeth, only daughter of Henry Young, Esq., M.B., C.M., of Lancaster House, Bournemouth, and Averias, Monte Video, Uruguay.

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Notices to Correspondents.

1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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Original Communications.

HÆMOGLOBINURIC FEVER IN ASSAM.

By ARTHUR POWELL, M.B.R.U.I., M.Ch., M.A.O.

THE existence of this fever in India was denied some years ago, but whether justly so or not, it is now either undoubtedly on the increase or, perhaps, more accurately diagnosed, where formerly it was confounded with bilious remittents. In the *British Medical Journal* received this week, Crombie says it "is practically unknown in India."

I now record eleven cases which occurred in this district, which were identical with the fever I have seen in those returned from the Gold Coast.

In five the blood and its parasites were examined, and it is to be noted that in all five non-pigmented, small parasites, often ring-shaped, were found during the attack, and in all crescents, after prolonged search in two cases, either during or after the fever. In all no other parasite was found at the time of the fever, but in one case certainly, perhaps in two, small, pigmented, malignant parasites were found during ordinary remittents some months before the hæmoglobinuric attack.

In view of Koch's recent revival of Plehn's theory that this fever is caused by the abuse of quinine, my cases are of startling interest, in that, of my last seven cases, no less than six were *either medical men or members of medical households*, i.e., *patients most likely to have thorough dosing with quinine.*

As in Africa, so here, it is Europeans, and those adopting European habits and medicines who suffer most from this disease, though the parasite found in the blood differs in no way that I can detect from the malignant parasite common in this district.

I can recall four cases in the practice of other men in the district; two were Europeans who had never been in any other malarious country. One of them had an attack in London on his return home, diagnosed either by Thin or Manson as hæmoglobinuric; the other two were well-to-do Babus, living with the Dr. Babu on tea estates—all unlikely to neglect the use of quinine.

The notes of cases I., II., and III. are lost. The first two died, the third recovered. All were treated with large doses of quinine.

Case XI.—Head Clerk, Brahmin, aged 28, of a very nervous, hypochondriac disposition, continually using patent and other medicines; frequent attacks of mild, malarial remittent; moderate spleen.

October 5, 1898.—Felt chilly; got fever reaching 102° F. Quinine 10 grs. thrice daily, given by hospital assistant, with whom he lived.

October 6.—Slight rigors; temperature 103·2°. At night first passed smoky, reddish urine, with greyish sediment.

October 8.—Urine like stout, one-third albumen; frequent chills, temperature varying from 102° to 105°; greenish-yellow coloration of skin and conjunctiva; black urine continued from this on till October 10, when I was called in just as he died.

Being a Brahmin, I could not get leave for a *post mortem*, even to the extent of removing some blood.

In this case quinine 10 grs. thrice daily, by no means a large quantity, was given by the Dr. Babu, but as the patient was such a glutton for drugs, and kept his own quinine, it is probable he did not limit himself to 30 grains.

Case X.—European surgeon, aged 29, contracted fever on Gold Coast four years ago. In England had relapses for two years, then one year free from fever. Came to Assam a year ago, had frequent attacks of fever, latterly lasting three days, with a regular interval of ten days' freedom.

I examined his blood in February last, finding both pigmented and non-pigmented, small, malignant parasites in the corpuscles, also crescents and crescent-derived spheres.

On August 23, 1898, he rode eight miles on a trolly in very heavy rain, wearing only white cotton banian, coat and trousers. Got thoroughly chilled, then rode a lame horse slowly for five miles to my bungalow. He felt fever coming on, so took quinine 5 grs. and a hot bath.

August 24.—Low fever all day, feeling very weak, 10 grs. quinine morning and afternoon. Blood contained many pigmented leucocytes, "cadavers," non-pigmented, intra-corpuscular bodies, and a very few 7-spored bodies ranged in the corpuscles in more regular rosettes than I have ever observed in this form of parasite.

August 25.—Rigor 7 a.m., lasting an hour; frequent vomiting; temperature 105·8°. Quinine 5 gr. tabloid. When damp behind ear, 10 gr. tabloids. Two hours later, sweating; temperature 104·4°. Quinine 10 grs. dissolved in acid. Temperature soon fell to 102·8°, but no relief to aching in limbs and loins. Cinchonism for first time. 2 p.m.—Rigor; a little later tempera-

ture 106·4°; urine red, bloody. The previous time urine was passed with stool and not observed. Feeling of cold over loins throughout hot stage; vomiting continuous. Quinine stopped. Liver and spleen tender and enlarged. 8 p.m.—Temperature 105·2°. Calomel 12 grs., chloral 20 grs. twice repeated. Urine black: ½ grey sediment.

August 26.—Rigor 7 a.m.; 8 a.m. temperature 105·8°. Calomel 5 grs. Rigor 10 a.m.; vomiting; rigor 12.30 p.m.; temperature 106·2°; skin green. Sodii sulph. and mag. cit. effervesc. 4 p.m.—Chloral 20 grs., pilocarpin ⅛ gr. Inside an hour profuse salivation, lasting only twenty minutes, and profuse sweating; vomiting; temperature fell steadily to 102° at 8 p.m.; pains greatly relieved.

August 27.—Sweating; temperature 100·2°; at 9.30 temperature 98·6°; urine still opaque black, like stout: $\frac{1}{12}$ sediment; slight pain in liver; tenderness of ribs in liver region, perhaps from vomiting, as patient lay on that side on hard edge of wire mattress while vomiting. From this stage onwards the patient slowly recovered his strength.

On October 10 urine red, translucent in the morning, normal afternoon.

Case IX.—Dr. Babu, aged 38, attributes attack to a chill caught in a storm while paying a night visit. Low fever next day; rigor and temperature 105° second day, when black urine first seen. Took acetanilide 10 grs., and three unmeasured doses of quinine—he estimated 5 grs. each. Urine nearly clear in evening.

3rd day.—Very prolonged rigor; temperature 105.2°; black urine; pain in loins, head, and limbs; bilious vomiting. Acetanilide 6 grs. twice, quinine as before.

4th day.—Conjunctiva and skin deeply coloured; vomiting; black urine continues; temperature 102·4°. Quinine stopped; diuretics; calomel 10 grs., followed by sodii sulph. Urine reddish in evening. Blood: unpigmented bodies in corpuscles; no crescents seen. A week later crescents present.

Temperature and urine normal next day. Convalescence rapid.

Case VIII.—Brahmin cook (and relative to clerks and hospital assistants on tea estate), aged 25. Malarial cachectic; large spleen. Rigor; temperature 105·6°; urine first passed brown, smoky colour; rigors; vomiting; hæmoglobinuria; fever continued to 6th day, when he died. Hiccough began 5th day. Treated throughout with calomel and saline purges, quinine in repeated doses of 10, 15, and 20 grs. by mouth and rectum.

Case VII.—Coolie man, aged 27, a malarial cachectic, had low fever for two days after a chill caused by a flood, in which water covered the floor of his house for some inches.

On 3rd day had a rigor; received three doses of quinine. Dose not recorded; probably 10 or 15 grs. each. Evening, temperature 105·4°; urine hæmoglobinuric; pain in loins severe.

4th day.—Morning, temperature 105·4°; acetanilide 5 grs. Slight sweating, quinine 15 grs. Three rigors during day. Evening, temperature 105·6°; quinine 15 grs. twice.

5th day.—Temperature 103·8°; one prolonged, three slight rigors during day; urine black, muddy, scanty; ¾ albumen in morning. Quinine 55 grs. given in course of day. Crescents and unpigmented rings very numerous; corpuscles very pale, very few, lying separate in even thick preparations. Evening, unconscious; temperature 105·8°.

6th day.—No urine; coma; temperature 104°. Quinine (neut. sulph.) 17 grs. hypodermically. Death two hours later.

Post Mortem.

Liver not much enlarged, stained yellow; spleen 19 ozs., black, pigmented; kidneys enlarged, not weighed, congested; pelves and ureters contained almost grumous fluid, same as deposit in urine; brain pale, soddened; membranes dark, not congested, their small vessels and those of brain blackened with pigment.

Case VI.—Dr. Babu's son, aged 11, malarial cachectic. Very large liver and spleen; crescents; many pigmented leucocytes; unpigmented, small intracorpuscular bodies.

After some days' indefinite low fever, a rigor occurred; temperature rose to 106°. In evening urine noticed black; skin and conjunctiva coloured; great pain in loins and liver. Fever continued, with remissions and sweats, but no further rigors till patient died on 6th day, delirious and in convulsions. Temperature shortly before death reached 107°. Treatment: purgatives; quinine, freely by mouth and rectum; occasional cold baths and antipyrin.

Case V.—Hospital dresser, aged 18. Suffered frequently from remittent fever; spleen reached within two fingers' breadth of umbilicus. Blood contained always crescents, and occasionally unpigmented intracorpuscular bodies.

Particulars of this case are mislaid, but the hæmoglobinuria was noticed first on the 2nd day. He died on the 5th, suddenly, of hyperpyrexia. The urine remained black, but not markedly diminished in quantity to the end.

Case IV.—Coolie woman, aged 23. This case combined the features of hæmoglobinuria and pernicious comatose remittent.

1st day.—Rigor; temperature 103·8°. Hæmoglobinuria noticed from onset. Quinine 10 grs. thrice daily. Black water, sweats, rigors, temperature from 102·2° to 105·4° continued till afternoon of 4th day, when coma set in; temperature 105·4°. Urine black, fair quantity: deposit $\frac{1}{10}$ fresh, ⅝ boiled. Quinine 1 gramme (neut. sulph.) hypodermically. Sweating slightly two hours later—10 p.m. Calomel 10 grs., croton oil 1 m.

5th day.—6 a.m., coma. Quinine chlorhydrosulph. 16 grs. hypodermically. Temperature 104·8°. Profuse sweating soon followed.

6th day.—Mind clear; urine reddish. Recovered rapidly.

There are some points in these cases to which I would draw attention.

The Urine.

Reaction was in all cases acid. Specific gravity varied from 1·024 to 1·034 (in Case VII., on 4th day). When thick and scanty doubtless it was higher, but was too scanty to immerse the hydrometer, and dilution gave uncertain results with

clinical instruments. Those figures refer to the black, reddish, or smoky stages only.

Sediment of a greyish-brown colour varied from $\frac{1}{12}$ to $\frac{2}{20}$ in height where the quantity was enough to be collected in graduated glasses.

Albumen was always present, and in all cases reached at least $\frac{1}{3}$ at some stage of the disease. In Case VII. it reached $\frac{1}{2}$.

Casts.—Here I found considerable variation from the descriptions of the text-books. Brown, granular casts of hæmoglobin were present, but in all cases there were also epithelial casts—in some cases, IV., VII., IX., and X., exceedingly numerous—and the most striking feature under the microscope. The numbers were so great the marvel is any epithelium was left in the kidneys; yet as three of these four cases recovered, it cannot be regarded as a bad prognostic sign.

Blood casts were never seen, and corpuscles in very few numbers in only two cases—VII. and X. Connolly (*British Medical Journal*, vol. ii., 1898, p. 882), says "large numbers of red corpuscles are present."

After boiling, a red flake like rust was often observed above the grey albuminous precipitate.

Straining to pass urine was not marked in most cases.

The *stools* were usually black instead of pale, as is usual in bilious remittents.

The *colour* of the skin seemed to me somewhat different to that in jaundice. In fair skins it seemed a more greenish yellow, like that of a bruise in certain stages. In some cases where the coloration of the skin was marked, that of the conjunctiva, though definite, was less than I would have expected in a case of jaundice.

Bilious *vomiting* was present in all cases. All cases were old malaria patients; ten of the eleven cachectics. Of the eleven, seven died. The four who recovered all suffered from anæmia, severe in three cases.

Chill was apparently the exciting cause in seven of the eleven cases.

Pain over the liver was generally present, over kidney always. In some cases a sensation of coldness, or of a cold wind playing over the loins, was complained of, even during the hot stage.

Rigors were present in all cases, initial only in Case VII., a child. Possibly the later convulsions in this case were those commonly replacing rigors in children. In Case IX. rigors occurred about the same period on two successive days, suggesting intermittence. This was the mildest case. In most of the other cases the rigors showed no regular interval, and were frequently repeated two or three times in a forenoon, suggesting a subintrant or a septic form of fever.

With regard to the question of quinine, I would prefer to simply lay the facts unvarnished before the reader. Of nine cases treated throughout with fairly large doses of quinine, seven died, two recovered. Two, in whom the quinine was stopped when I first observed the hæmoglobin, recovered.

Of the eight cases with records, one began hæmoglobinuria at the onset of the fever, before a grain of quinine was administered. She was treated with large doses of quinine, and recovered. In all the other seven quinine was administered freely before the colour of the urine became bloody.

I have given to malarial cachectics in pernicious attacks large doses of quinine—40 grs. sulphate—dissolved in acid, by mouth, one drachm by rectum. The chlorhydrosulphate, which represents a larger quantity of quinine than the sulphate, 31 to 32 grs. hypodermically to adults, 17 grs. to a small girl of 13. No hæmoglobinuria has ever resulted.

With the authority of Kelsch and Kiener and Copeman I never hesitated to use quinine freely. Till we hear further from Koch his opinion must "give us pause," and be the plasmodia absent from or teeming in the blood, I shall stand by calomel, diluents, salines, and pilocarpin.

In reading detailed cases, one finds "Bovril," Brand's essence, Liebig's extract, favourite modes of treatment. To me this seems adding a stack of straw to the lame camel's back. These preparations are mainly albumen and the nitrogenous salts of muscle, not far removed from urea and hæmoglobin in composition. The poor denuded and half-blocked kidney tubules and the liver have enough refuse in the blood to excrete without these added salts.

A NOTE ON THE DOSAGE OF QUININE IN MALARIAL FEVER.

By W. Jones Greer, F.R.C.S. and D.P.H.Irel.

Some years ago, when in the first flush of a licence to practise, I found myself hurriedly preparing—having had short notice—to proceed to West Africa, I felt considerably excited and elated at the prospect; during occasional calm moments I endeavoured to stir up my memory and to ask myself, What do you know about tropical diseases? I am very much afraid this question was soon answered, not to the entire satisfaction of my conscience. Of course I first thought of malaria—quinine was the remedy for that; then there was dysentery, which yielded promptly to large doses of ipecacuanha. I was rather surprised there were no more tropical diseases; however, these were the chief. Yet was I well and carefully taught, and this lamentable ignorance was due to no fault of my teachers—the fact was it did not pay from an examination point of view to know much about tropical diseases. When I look back on this time I am bound to confess that my knowledge of tropical diseases then, whatever it may be now, was about as detailed as the average medical student's knowledge of theology. In those days—and they are not so long ago—there was practically no instruction in the diseases of warm climates, and only occasionally had a teacher any personal experience of them. I still have the impression with me which was made by a few minutes' conversation with an old West Coaster just before my first trip to the Coast; the most interesting facts that I could elicit with regard to the endemia were that Smith, whom I knew well, had died in Bonny; Jones, who was qualified the year before me, was lying in the cemetery at Old Calabar; the others, Brown and Robinson, occupied freeholds in

the subsoil at Sierra Leone and Axim respectively. This was my preliminary instruction in tropical diseases. But we have changed all that; not only have we books devoted to the tropics, but we have a school in process of being equipped for special instruction in their peculiar diseases. And now a distinct want is satisfied in the form of a JOURNAL OF TROPICAL MEDICINE—a notable achievement and an inestimable boon.

In selecting books, amongst others I was fortunate in choosing M. Laveran's work, and with its aid I was able to be of some benefit to my patients.

In endeavouring to work out from my books a line of treatment, I had great difficulty in coming to a conclusion as to what was the proper dose of quinine. Now it seems to me that this difficulty still exists for others, for I read in present day prints that 7 to 15 grains of the sulphate may be given daily; or 5 grains daily; or again, 20 grains every four hours. Surely it cannot be immaterial which dose is given? One medical man with a mathematical mind will probably strike an average, another will make a choice. Now I think I had better say at once that, in my humble opinion, the man who in treating West African fevers, at any rate, chooses to give 20 grains of quinine every four hours, will very quickly form a strong opinion of the absolute deadliness of the climate. The fact, quoted by M. Laveran and others, and which I have verified for myself, must not be lost sight of, that a large dose of quinine, say 20 grains, in a case of simple fever may produce an attack of hæmaturia. With this serious complication in view, surely such a wide range of dosage should not be allowed; there ought not to be any insuperable difficulty in coming to a conclusion as to what is the proper dose. Of course I am willing to grant that it is not possible to fix a dose that will suit every case, but I think that the range of dose ought not to be so extensive as it is at present; my own opinion is that 30 grains in divided doses in the twenty-four hours should be the maximum, and that 20 grains in the twenty-four hours is as a rule sufficient, this quantity to be gradually diminished as the fever subsides.

It appears to me that the great risk in not having a more definite dosage is that an inexperienced practitioner, if he exists, in treating a case where the temperature was high might trust too much to the antipyretic properties of quinine and be led into giving repeated large doses, and as a consequence produce an attack of hæmaturia. With regard to hæmaturia, whether existing as a special affection or as a complication, I feel convinced that quinine should be altogether withheld.

This difficulty of dosage seems to me to be a real one, but there ought to be a solution of it. There are many able and distinguished practitioners with extensive experience of the treatment of malaria, who could authoritatively settle this point and not leave it to guess work or the idiosyncrasies of the individual practitioner.

A CASE OF MALARIAL FEVER, WITH INTERCURRENT ATTACK OF TYPHOID FEVER, ILLUSTRATING THE VALUE OF MICROSCOPICAL EXAMINATION OF THE BLOOD AND WIDAL'S TEST IN DIAGNOSIS.

By F. KENNETH WILSON, M.B., B.S.Lond., M.R.C.S., L.R.C.P.
Senior House Surgeon, Seamen's Branch Hospital, Royal Albert Docks.

N. N., aged 24, native of Sweden, had his first attack of malaria in September, 1897, while in Mexico. The attack lasted about three weeks, the fever recurring nearly every day. His second attack was two months later, when he was in Philadelphia; it lasted about two weeks, the fever recurring irregularly. His third attack was on his voyage to England, in January, 1898, when the fever occurred each day, beginning early in the morning.

On February 24, 1898, he was admitted to the Seamen's Branch Hospital in a condition of cachexia. His blood was examined and found to contain the benign tertian parasite. On the day of his admission, and again on the third and fifth days after his admission, he had typical malarial rigors followed by heat and sweating. Treatment with quinine in the form of 5-grain pills every six hours was then commenced.

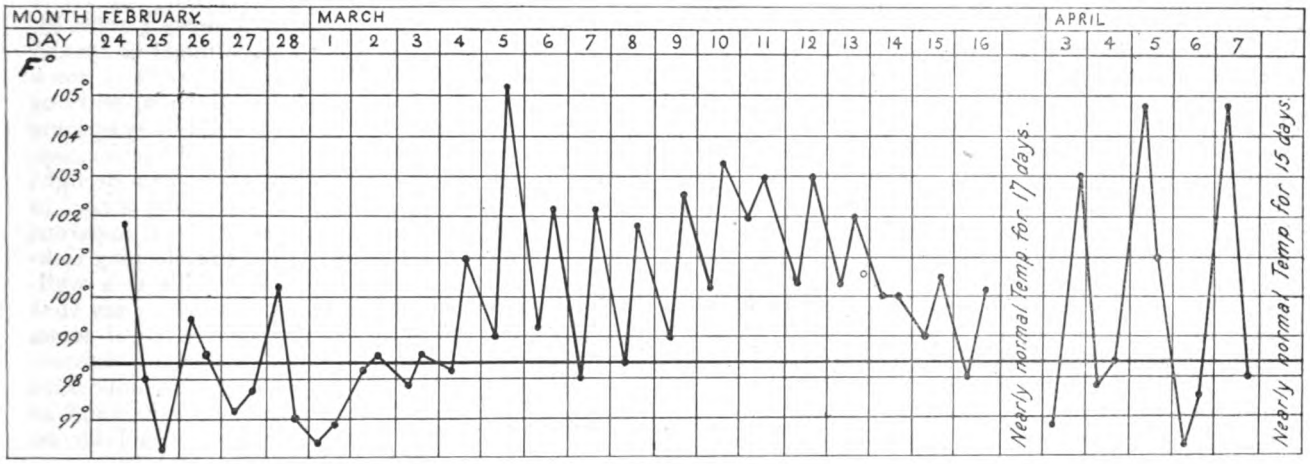
After being normal for three days, on the fourth day of this treatment the temperature rose to 101°. By the next day it had fallen to 99°, but rose again on the following day to 105°, and he had a sharp rigor. From this time the temperature assumed at first an intermittent, and later a remittent, type, as can be seen in the chart. At the time of the rigor mentioned no parasites could be found in the blood; the quinine was therefore discontinued two days later. From the time the administration of quinine was stopped the temperature, although making daily excursions of 2° to 4°, did not touch normal for seven days. As suspicion was aroused during this week that the quinine in the form of pills might not have been absorbed, films of blood were prepared every hour on one day, from 7 a.m. until 3 p.m., and after being stained with methylene blue were examined, but no parasites were found.

During the last three days of this continued temperature, an injection of five grains of quinine hydrochlorate was given night and morning hypodermically in the supra-scapular muscle.

In the middle of the week he was very pale, with a furred moist tongue, and had a moderately distended abdomen, but showed no spots. He had much headache. Widal's reaction was now tried, and gave a positive result. Next day some rose spots appeared upon the abdomen. The motions were formed, of a slightly lighter colour than normal, and there was slight bronchitis. A day or two later some fresh crops of spots appeared; the spleen could be distinctly felt, the motions became more yellow, the pupils were dilated and the headache had gone.

At the end of the week mentioned the blood was again examined, but no malaria parasites were found. The temperature remained about normal for a month. His condition about half way through this month was

one of emaciation, the spleen could not be felt, and Widal's reaction gave a positive result. At the end of the month the temperature suddenly went up to 103° and he had a typical malaria rigor; the temperature fell again to rise two days later, when another rigor occurred—the spleen could then be felt. Amœboid



pigmented parasites (benign tertian) nearly filling the corpuscles and enlarging them, and some flagellated bodies were found on examining the blood. After the occurrence of three rigors, quinine was commenced again in doses of five grains every six hours.

When the temperature was due to rise for the next paroxysm no parasites could be found in the blood, and the spleen had diminished in size. Quinine was continued until a drachm had been taken; then iron and arsenic pills were substituted for the quinine, except on Sundays.

The patient left the hospital on April 22, having had a nearly normal temperature for a fortnight.

Comments.—The combination of most of the ordinary clinical signs of enteric fever, such as headache, abdominal distension, crops of rose spots, enlarged spleen, yellow stools and slight bronchitis with a repeated positive result to Widal's test, even in spite of the absence of a quite typical chart (of which the atypical character is probably due in some measure to the administration of quinine), and of a quite typical tongue (which was moist and furred throughout the illness), will, I think, be taken as sufficient evidence of enteric fever.

Positive proof was given by the microscope of the presence of the malarial parasite, both before and after the occurrence of the enteric fever.

The case might be described as one of typho-malaria; the expression being used not as indicating a specific disease, but as Dr. Manson says, as indicating "an ordinary typhoid occurring in an individual who has been exposed to malarial influences."

Of the three clinical types of cases he describes this case will come partly under the first and partly under the last. Osler says: "Of nearly four hundred cases of typhoid fever, all with blood examinations and a majority of them coming from malarial regions, in not a single instance were the malarial parasites found in the blood."

The behaviour of the spleen in the case is of interest. I have no note of its condition when the patient was admitted; but it evidently enlarged from the enteric fever in spite of the administration of quinine, and later when the enteric fever was over enlarged again from the malaria and was controlled by quinine.

A CLINICAL LECTURE ON THE SLEEPING SICKNESS.

Delivered at Charing Cross Hospital, October, 1898.

By Patrick Manson, M.D., LL.D., F.R.C.P.

Medical Adviser to the Colonial Office; Physician to the Seamen's Hospital Society Branch Hospital, Albert Docks.

Gentlemen,—I propose to speak to you this afternoon about the two negro boys who have been in Alexandra Ward for the last few weeks. They come from a village called Mbanza Manteka, on the Lower Congo, and were brought to England principally through the instrumentality and enlightened benevolence of Dr. Grattan Guinness and the missionary body he represents. They were brought to this country partly that they might have the chance of treatment under conditions more favourable than any their own homes could supply, partly that they might afford an opportunity for careful study of the deadly disease from which they are suffering. Although it is not likely that any of you, unless you visit the West Coast of Africa, will ever see similar cases again, nevertheless, their study is capable of affording useful lessons of a practical character. Apart from such practical consideration there is much about these cases of theoretical interest. If the medical student aspires as he should aspire, to be a student of medicine, he must be catholic, and embrace in his studies all forms of disease, no matter though some of these diseases happen to be confined to a limited geographical area, no matter though they affect only what we, in our pride, consider an inferior race. The science of pathology embraces black men as well as white men; the principles of pathology are the same for all.

Nomenclature.

The disease from which these negro boys are suffering is called the "sleeping sickness;" sometimes, and because it is especially prevalent there, the

"sleeping sickness of the Congo;" sometimes "negro lethargy." A better name would be "African lethargy."

Geographical Limits.

It has a singularly limited geographical distribution, being, so far as we at present know, absolutely confined to that part of West Africa which lies between the Senegal to the north and Loanda to the south, some 1,500 miles of latitude. It extends into the back country; how far it is difficult to say. I am told by Dr. Sims, of Stanley Pool, who has had many years' experience of Congoland, that it occurs as far up the Congo as Stanley Falls, that is to say, to the very centre of equatorial Africa. So that, after all, the geographical limitations are not so restricted. The area involved, though large, like everything that is at a distance from us, bulks small in the mind's eye. It probably exceeds that of Europe; it certainly carries a population of several tens of millions.

Although widely diffused throughout this huge region, fortunately for the inhabitants, it is only here and there, in certain circumscribed spots, in certain villages, or groups of villages, that sleeping sickness occurs as an endemic affection. Nor is it always equally prevalent in these places. In the spots affected it seems to come and go, to wax and wane as it were. And it is fortunate that it is so; for, when sleeping sickness breaks out in a community it attacks a large proportion of the members of that community, and all who are attacked surely die.

Mortality in Epidemics.

Thus in the village from which these boys come, out of a Christian congregation of about 1,000 last year 28 died of sleeping sickness; and this year, Mr. Richards tells me (it was Mr. Richards who brought these boys home), that up to the month of August, when he left the Congo, out of a congregation of about 1,150, 28 had already died, a mortality at the rate of about 40 per annum. Corre, a French writer, who studied the disease in Senegambia, tells us that in some districts of the Lower Senegal it sweeps away whole villages at a time, half the people dying, the remainder fleeing from the pestilence.

Racial Proclivity.

Hitherto we have no well authenticated account of the disease in a white man; doubtless, however, were the white man exposed to the cause, whatever this may be, he would prove as susceptible as the negro. It has been met with in half-breeds and in Moors. Some have suggested that sleeping sickness is in some way inherent and peculiar to the negro, a sort of ethnic disease. This cannot be the case, for the negroes of the States, of the West Indies and of Brazil, and the natives of north, east, and south Africa never, so far as we know, nowadays get the disease. I say nowadays, because formerly, in the old evil times of the slave trade, sleeping sickness was well known on the plantations in the West Indies; but it was then known only as an imported disease. It never attacked negroes born on the plantations, only those who had been kidnapped in Africa. It did not spread on the plantations, or become domiciled, as it were, in America. I beg of you to note this circumstance; it is an important one to keep in mind in connection with the question of etiology.

Prolonged Latency.

Another significant fact about the disease is that it may remain latent for years—as long as seven years it is said. Thus, in the evil times I refer to, what appeared to be a healthy slave might be landed in the West Indies, and might do excellent work for his owner for several years, and then, without apparent reason, he would begin to exhibit symptoms of sleeping sickness, and by-and-by die of the disease.

Some years ago a little Congo boy was brought home to England, and placed in a training school in Wales. Here he worked for three years in apparent health, but at the end of this time the sleeping sickness laid hold of him, and he died. This is a well-authenticated case. The negroes themselves say that a man is never safe from sleeping sickness until seven years have elapsed after a visit to a sleeping sickness district. This prolonged latency is another important fact which I would also ask you to bear in mind as having a bearing on what I shall advance later on about the etiology of this singular disease.

Before proceeding further I will give a summary of what we have been able to learn and observe about our cases. The account is very incomplete, as there are linguistic difficulties which, as you will readily understand, make it difficult to get clear and reliable information about the subjective symptoms as well as about the medical histories of the cases.

Case I.—Eli Mboko, aged about 20, until his present illness began had the reputation of being an exceptionally bright and intelligent lad. He taught himself English, built his own house, and in many other ways displayed an amount of enterprise unusual in a negro. He was regularly employed as a teacher in the Mission School at Mbanza Manteka, a village in the hilly country on the south bank of the Lower Congo, and a notorious haunt of sleeping sickness.

He discharged his duties with ability and energy until about twelve months ago. He then began to be listless and vacant-minded. He would now lie abed in the morning, neglect his work, and cease to take an interest in his former occupations and amusements. Mrs. Richards, who superintended the school, told me that on several occasions she found him standing motionless in the middle of the class-room regardless of what was going on around, and simply gazing into vacancy. When she spoke to him he would rouse himself and resume his work, and presently he would relapse into the same listless, vacant condition. He complained of headache, particularly occipital headache, and at times fever. By May of this year he was quite unfit for work; his gait had become tottery and uncertain, and he drowsed or slept most of the time. He never had any fits or maniacal attacks. He volunteered to come to England, and left the Congo on August 27. He improved somewhat on the voyage, for when I fetched him from Bow on the day of his arrival in London—September 24—though very quiet and reserved, he appeared to be wide awake, taking an interest in the traffic in the streets as we drove along to the

Case 1.—ELI MBOKO.

(By permission of the "British Medical Journal.")

Bale & Danielsson, Ltd., London.

hospital. He was quite able to walk upstairs to Alexandra Ward.

On admission under Dr. Abercrombie he seemed to be in good general health. He was fairly well nourished, and even sleek. Lately, however, he has fallen off somewhat in condition, and his hair and skin have become dry and lustreless. At first his breath was offensive; this has now improved. His appetite and digestion are vigorous. The bowels on admission were constipated — as, indeed, they still are. Microscopic examination showed that the stools contained large numbers of the ova of ascaris lumbricoides, of ankylostoma duodenale, and a few of trichocephalus dispar. The thoracic and abdominal viscera appeared to be healthy. There was no sugar or albumen in the urine. Temperature has ranged from 97° to 99° F., being usually slightly subnormal. Pulse 80 to 90, respirations 20. The blood count gave 5,300,000 per cmm., hæmoglobin 60 per cent. The blood contained no malarial parasites, but filaria perstans was, and is, present in moderate abundance —about 1 filaria in every cmm. The senses were, or appeared to be, normally active. Mr. Treacher Collins, who was good enough to make an ophthalmoscopic examination, found fundus and media perfectly healthy. The pupils were equal and reacted briskly both to light and accommodation. A very few and ill-defined papules could be made out on the skin of the chest and abdomen, but there was no postulation and no marked pruritus. The knee-jerks and other deep and superficial reflexes were active; but his gait was shuffling and feeble, and his hand grasp was markedly impaired. When he walked he progressed slowly, his knees slightly bent. He was easily fatigued, and always seemed glad to sit or lie down. There was no muscular tremor, no local anæsthesia, no paresis. The lymphatic glands, particularly those of the neck, were slightly but distinctly enlarged. In size they varied from an almond to a hazel nut; they were not at all tender.

Since admission his condition has varied very little. For the most part he keeps his bed, lying perfectly still with eyes shut, as if asleep. His face has an expression of deep melancholy, At times he sits up by the fire, but even then he seems to be asleep. The only time he rouses himself thoroughly is when food is brought, or when for a short time he goes on the balcony and watches the traffic in the streets. As a rule he is glad to get back to bed. A touch or a loud sound causes him to open his eyes, so that if he is asleep, the sleep must be a very light one. When spoken to he opens his eyes and answers questions slowly, in few words, and to the point. Even while he is being spoken to, often his eyelids fall as if he were overpowered by an irresistible desire to go to sleep. He never seeks to enter on, or to prolong, a conversation. He rarely smiles; if asked, he says he is very unhappy on account of his drowsiness. Since he entered the hospital he has become distinctly more lethargic, and, I think, has lost flesh and muscular power.

Case II.—Tendo Mkaloo, supposed to be about 11 years of age, comes from the same village, Mbanza Manteka, as Mboko. Very little is known about his antecedents. He is believed to have shown symptoms of sleeping sickness for about two months, that is to say, since May or June, before he left the Congo. During the voyage home he became much worse. When brought to hospital he was in a state of great emaciation and weakness. He could not walk without support, and was barely able to stand alone; he had to be carried upstairs. His cheeks were sunken, his eyes unusually prominent, his lips were swollen and dry, the saliva dribbling constantly from the corner of his mouth; his hair was dry and scurfy, and his skin harsh and lustreless. Arms, abdomen, and chest were streaked with white lines, nail marks from the scratching provoked by an incessant pruritus associated with numerous scaly papules. The superficial lymphatic glands and even trunks were universally enlarged, particularly so those on the back and sides of the neck. Some of the glands appeared to be very tender, for he carried his head stiffly as if movement hurt him, and he shrank and cried when an attempt was made to examine the little swellings distinctly visible at the side of the neck.

His breath was foul, and his abdomen tumid and lax. The spleen was much enlarged, extending as far as the umbilicus; the liver also was enlarged, its lower border being readily felt about a hand's breadth below the costal margin. As in the case of his companion, his fæces contained myriads of ova of ascaris lumbricoides, ankylostoma duodenale, and trichocephalus dispar, and he was markedly constipated.

His temperature was considerably above normal for some days after admission. Eyes, lungs, heart, and kidneys appeared to be healthy.

The superficial and deep reflexes were active. He slept and drowsed almost continuously during the first few days. At first he had to be coaxed to eat, and had often to be fed by the nurse. Sometimes he would fall asleep at his meals. There was some suspicion of muscular tremor.

His blood count gave 4,500,000 per cmm., and a hæmoglobin value of 50 per cent. Filaria perstans was found in considerable abundance—8 per 5 cmm. No malarial parasites were discovered.

After due preparation he had a course of thymol (15 grains for four doses at intervals of an hour and a half); this effectually rid him of his intestinal parasites. Probably partly in consequence of this, partly in consequence of the warmth and good feeding, and partly in consequence of the large doses of arsenic he was put upon, his general health quickly underwent a marked improvement, and his spleen and liver diminished in size. Concurrently with this his drowsiness got less, and he is now much more lively; he even laughs occasionally. He sits up in bed or by the fireside and watches what is going on about him. He is also putting on flesh, and is able to walk about a little, though his gait is slow and shuffling. The pruritus, however, is as bad as, or even worse than, ever. His lymphatics, though diminished in size and no longer painful, are still distinctly enlarged. The prominence of the eyes is less marked; he no longer dribbles from his mouth, and he feeds himself and eats with some appetite. He has had one or two feverish turns, but, as a rule, his temperature is normal or subnormal.

So much has he improved during the few weeks he has been in hospital that we are sometimes tempted to think, were we not assured to the contrary by those familiar with sleeping sickness, that the diagnosis may be wrong. We are told, however, that there can be no doubt the little fellow is the subject of this disease, and that ere long the old symptoms will recur with increased severity, aud that others of graver character will by-and-by be superadded.

From these accounts you will be able to form some idea of the earlier phases of one type at least of sleeping sickness.

Symptoms.

Onset.—A negro, otherwise apparently in good health, is unaccountably smitten with a gradually increasing mental and bodily lethargy which goes on deepening in intensity until he is bedridden. Those who are familiar with the disease, including very often the patient himself, can recognise the earliest threatenings of the impending calamity. There is a characteristic expression of face and body; a significant droop of the upper eyelids; a listless carriage of the body; an indifference to old amusements and occupations; a stolid, rather mournful expression of countenance; perhaps slight puffiness of the features. It is noticed that the patient is easily tired at his work; that he lies long in bed in the morning; that he often falls asleep, even at his work; that he becomes morose and unsociable, and ceases to take share in conversation. Nevertheless he will answer intelligently and to the point when addressed. He may have to be asked once or twice before he replies and the answer when it comes may be a brief one; but, from the appropriate nature of the answers, it is evident that questions are properly, though perhaps slowly, comprehended.

The Sopor.—While you speak to him his eyelids may fall and sleep seemingly overpower him. This is very apparent in the elder of these two boys. If you put the question to this lad he may tell you that he has headache, and he can be got to indicate with his hand the aching part; but he never spontaneously complains of his suffering. People may be talking all around, but apparently he takes no notice of what they are saying, although an unusual noise or a light touch may make him open his eyes for a moment. If he is really asleep at these times the sleep is a light one. The condition seems to be one of mental vacancy rather than one of true sleep.

Muscular Debility.

Equally striking is the muscular debility from which both patients suffer. Their movements are all languid; and they are very easily tired out. Sitting up for an hour or two in a chair in the afternoon thoroughly exhausts them, and they are glad to get back to bed. When they walk they shuffle along, sometimes tottering like one half asleep, or like a drunken man; their knees seem to give way under them.

At first sleeping sickness patients can feed themselves, although they may take a long time over it. The little boy takes an unconscionably long time over dinner. He pauses between each mouthful, gazing into vacancy the while; he has to be reminded now and again that he is dining. Sometimes such patients will fall asleep with the morsel half way to their mouths, or even with the half chewed food between their teeth, and may have to be wakened up to swallow it. At a later stage of sleeping sickness muscular tremor usually sets in; sometimes this is so marked, even at an early stage, that the patient can no longer feed himself.

Early Nervous Symptoms.

The symptoms of the disease, at all events at this stage of its progress, point to a pathological condition of the higher nerve centres only. The knee jerks and other reflexes are intact; there are no bladder nor rectal symptoms; no trophic lesions. The discs and fundi are perfectly normal. Nutrition is not impaired, and digestion and assimilation are satisfactorily performed.

General Symptoms.

From the commencement the patient may be subject to short daily spells of feverishness; or he may have a high temperature for several days on end. Fever, however, is not, as a rule, of an urgent character, unless there is concurrent malaria. Occasionally there may be a little diarrhœa. At other times, and as a rule, the body temperature is subnormal—96° or 97°F. Such patients evidently feel chilly, for they like to coil themselves up in some sheltered spot and bask in the rays of the broiling tropical sun.

Skin Lesions.

A very striking and common symptom in sleeping sickness is the intense pruritis from which many of the patients suffer. It is especially marked on the trunk, but it occurs elsewhere. Little papules can be seen in many places, especially about the chest and abdomen. In healthy young negroes the skin is soft and glossy, like velvet; but in this disease—as is particularly apparent in the younger of these boys—it becomes dry and lustreless, and is generally scored all over with the white streaks produced by the incessant scratching. If you notice the elder of the two boys you will often see him carry his hand to his nose or forehead, and rub the part as if it were irritated.

Enlarged Glands.

Another and interesting feature of sleeping sickness is an enlargement of the lymphatic glands, especially of the posterior cervical. In some instances, as in the smaller boy, less so in the elder boy, most of the superficial glands are affected. The individual glands can readily be felt enlarged to the size of an almond or of a small nut. In the little boy at one time the posterior cervical glands on the right side appeared to be painful, for he would cry when his head was moved; he held his head rather stiffly, apparently in consequence of subacute cervical adenitis.

Progress of the Disease.

For a long time, perhaps for several months, this is about all that can be made out in the type of the sleeping sickness we are considering. Some days the patients seem a little brighter, some days they seem a

little duller. Occasionally they become quite active and intelligent; and it may even seem that they have recovered. Such improvement, I am assured, is invariably only temporary; sooner or later the lethargy returns, and the disease once more advances to the inevitable and fatal issue.

Convulsions and Mania.

In certain instances, and by no means rarely—and this is the other type of sleeping sickness I have hinted at—the disease is introduced by, or its progress is interrupted by, maniacal outbursts; not infrequently by epileptiform seizures, very like those of general paralysis of the insane. A whole series of convulsive fits may follow one after the other. The maniacal outbreaks may take very different forms—delusions of all sorts, hallucinations, homicidal or suicidal impulses. These epileptic and maniacal outbursts are supposed to be characteristic of the more acute cases; such cases are believed to advance more rapidly than the purely lethargic ones. I asked Mr. Richards why he did not bring a case of this description home. He told me that it would have been very difficult to have managed such a patient on board ship; that very probably he would have jumped overboard in a maniacal fit, or, at all events, he would have been an intolerable nuisance to his fellow passengers.

Terminal Symptoms.

Whatever may have been the exact characters and progress of the earlier phases of the disease, ultimately the patient becomes completely bedridden. Nutrition now begins to suffer, if it has not done so before. Choreic, convulsive, or tetanic spasms of groups of muscles or, it may be, of a more general character, in addition to the tremor already mentioned, are apt to occur from time to time, indicating grave implication of the motor centres. Muscular prostration is now extreme, torpor more profound and continuous. Bed-sores may form, or diarrhœa or other complication set in and carry off the patient, or he may die in one of his convulsive or tetanic seizures. Some years ago there was a case of this disease under Dr. Stephen Mackenzie in the London Hospital. I saw this man when he was dying. For hours his head was violently retracted by tetanic contraction of the extensor muscles of the neck, and every now and again he seemed to be on the point of asphyxia from spasm of the glottis. The disease may run its course in two or three months, or it may last for as many years. Nine months seems to be about an average time.

Pathology.

Although a considerable number of *post-mortem* examinations of cases of sleeping sickness have been recorded, little, if any, light has been thrown by them on the morbid anatomy or pathology of the disease. In some of the records, fulness and even varicosity of the vessels is reported; in other instances the vessels are described as being empty. In some instances the brain substance was said to have been abnormally hard; in other instances abnormally soft. In the earliest recorded *post-mortem* examination the pineal body was found to be enormously enlarged; in subsequent examinations this condition has not been remarked. In Dr. Stephen Mackenzie's case, beyond a cysticercus on one of the anterior lobes of the cerebrum, no morbid lesion of the brain was detected; certainly there was no meningitis, and no gross lesion of the cerebral substance.

Natural sleep, as you are aware, is associated with, and probably depends on, anæmia of the brain. Morbid sopor depends sometimes, apparently, on the direct action of a toxic substance on brain cells; sometimes on a cerebral anæmia produced by the presence of a tumour, or by an action of the toxic substance on the intracranial circulation. In which of these ways the sopor of sleeping sickness is produced it is as yet impossible to say. I have sometimes been tempted to think that perhaps in these cases the pituitary body is the original seat of disease, and that the brain becomes secondarily affected. Such a hypothesis receives some, though I confess very slight, support from experimental physiology as well as from recorded cases of disease of this organ. I can only hope that our cases may yet throw much needed light on the nature of what is at present a pathological puzzle.

[Note.—After the delivery of this lecture I received on October 27 a letter from a friend on the Niger giving some particulars of the *post-mortem* examination of a case of sleeping sickness. The pituitary body was found to be enlarged. There was an old blood clot on or in it, and, as I gather, some cystic formation as well.]

Etiology.

It has often been asked, What is the cause of sleeping sickness? I cannot give any decided answer to that question, but we may very properly seek in the symptoms, distribution, and, so to say, the natural history of the disease for some indication as to this important point.

Sleeping sickness has been attributed to all manner of things, amongst others to sunstroke; but the case I have alluded to as having developed in Wales effectually upsets such an idea; a Welsh sun is not likely to penetrate a negro's cranium. It has also been attributed to the inordinate consumption of palm wine—a common vice in the negro; to excessive venery; to the use of improperly prepared manioc—the staple food of many of the negro tribes, and when improperly prepared apt to be poisonous. Manifestly it can be due to none of these things, for children, who are just as subject to the disease as are adults, do not drink intoxicants, do not indulge in sexual excess; and even negroes, when they visit Wales, do not feed on manioc. In the slave days it was sometimes attributed to nostalgia, to grief at being torn from home and friends; but at the present day, at all events on the Lower Congo, there is nothing of this sort. Like all tropical pathological puzzles, sleeping sickness has been attributed to malaria—that blessed cloak for ignorance; but there are none of the clinical or pathological marks of malaria about the disease. It is true that one of our patients has an enlarged spleen, but the type of the fever he sometimes suffers from is not that of malaria. Moreover, I have examined his blood carefully on several occasions and found no plasmodia, no pigmented leuco-

cytes. The other boy's spleen is not enlarged. In neither is there marked anæmia, such as there is invariably present in pronounced malarial cachexia. Some years ago two Portuguese pathologists declared that they found a specific bacterium in a case of sleeping sickness, and that they communicated the disease to the lower animals by injections of cultures of this bacterium. Dr. Bullock has attempted to grow a bacterium from the blood and lymphatic glands of one of the patients but his flasks have remained sterile. No, sleeping sickness can be attributed to none of these things.

There are some circumstances which, to my way of thinking, seem to suggest a clue that is well worth following up. I have already told you that sleeping sickness is limited to a certain region of Africa. In the endemic districts it attacks old and young, but especially the latter, particularly those between the ages of 10 and 20. It may pick out one or two in a household, or it may attack an entire family. Mr. Richards tells me that he heard on the Congo of an instance in which it was introduced into a hitherto immune village by a case that came from a neighbouring infected village; a circumstance suggesting transmission more or less direct of some form of infection. Indeed, the natives say that it is infectious; they say, doubtless erroneously, that the saliva, which sometimes dribbles from the corner of the mouth, in advanced cases, conveys the disease. All these facts are compatible with and suggest a living contagion.

I have also told you that it may remain latent for many years, and develop ultimately thousands of miles away from the endemic centre—that is, from the spot where its cause was acquired. There are many disease germs which are capable of remaining latent for years, and then of springing into pathological activity—tubercle and leprosy, for example. But, then, these diseases differ from sleeping sickness inasmuch as they are not confined to limited geographical areas; and, moreover, when introduced into virgin populations, being directly communicable like all bacterial diseases, they spread. Sleeping sickness will not spread. Why does it not spread? Manifestly because it depends on certain local conditions, conditions found only in limited districts in West Africa. This implies that these conditions exist only outside the human body; and, moreover, that these conditions cannot be transported.

This further implies that sleeping sickness depends either directly on a food of some sort peculiar to West Africa; or, directly or indirectly, on some plant or animal equally limited in its geographical distribution.

In Nature it is only such things as these that are geographically limited in the way that sleeping sickness is limited. Did it depend on a food—that is to say, on some organic poison in food, it is strange that the manifestations of the poisoning are at times delayed for years. It must therefore depend on some plant or animal. Further, the phenomena of the disease, the prolonged latency especially, demand that the cause must be something which can remain alive, though it may be pathologically inert for a very long time.

Filaria Perstans.

When in difficulties about the explanation of some obscure pathological or other phenomenon, in the absence of a better guide it is perhaps permissible to turn for assistance to analogy. Let us try if analogy will help us in our present dilemma, for there is an *a priori* probability that like diseases are produced by like causes. Let us first formulate precisely what it is we want from analogy. Are there any diseases which, like sleeping sickness, although produced by a living cause, can remain latent for prolonged periods, and which, moreover, can be acquired only in certain limited spots, are not directly infectious, cannot be introduced into virgin districts, and which depend directly or indirectly on the fauna or flora of their endemic areas? Yes; there are malaria, elephantiasis, and many others which I could mention. Why are these diseases so limited? Recent investigations enable us to answer this question. It is because their specific germs—the plasmodium in the one case, and the filaria in the other—before they can enter the body of man have to pass through the bodies of certain of the lower animals, which animals are so constituted that they can live only in certain more or less limited geographical or rather zoological areas.

If this be the case in the diseases mentioned and exhibiting the peculiarities enumerated, the same explanation may apply to other diseases with similar peculiarities. I think, therefore, that the germ of sleeping sickness in this respect resembles that of malaria and that of elephantiasis—that is to say, that at one stage of its existence it must necessarily live in some living host other than man; some animal or plant found only in the equatorial regions of West Africa.

There is one fact about sleeping sickness that greatly encourages this hypothesis, as it fits in exactly with such a view of the etiology of the disease. A peculiar parasite—filaria perstans—has been found in the blood of both the patients which you have seen. Moreover, this same parasite was found in the blood of the other patient whose case was studied in London some time ago—Dr. Stephen Mackenzie's case. The particulars of this case are fully recorded in the Clinical Society's *Transactions*, vol. xliv. In fact, it was in the blood of the latter patient that this parasite was first discovered. Dr. Mackenzie told me that his house-physician (Mr. Fagan) while examining the blood, had found in it what he took to be the ordinary blood worm—filaria nocturna. Dr. Mackenzie remarked, however, that some of the worms appeared to be larger than others, and, knowing that I took an interest in this class of parasite, he asked me to look at them. I procured slides of the patient's blood, and carefully studied the worms. I remarked that the larger worm differed not only in size, but also in shape, in structure, and in habit, from the smaller. You are aware that the ordinary filaria of the blood, filaria nocturna, is an active, wriggling creature, about $\frac{1}{75}$ inch in length by about $\frac{1}{300}$ inch in breadth; that it has a sharp-pointed tail; that it is enclosed in a loose trailing sheath or sac; that although it wriggles about, it practically remains at one spot on the slide, and does not locomote; and that it comes into the peripheral circulation only during the evening and

night. Now this new worm was only about $\frac{1}{125}$ inch in length by about $\frac{1}{5000}$ inch in breadth; it had a blunt tail; it had no sheath; besides wriggling about, it travelled through the blood on the slide, often at a great rate; further, it was present in the peripheral circulation at all hours of the day, as well as of the night. Manifestly it was a new species.

Naturally enough the concurrence of a strange parasite in a strange disease suggested, though it by no means proved, a cause-and-effect relationship between the two. Accordingly I set to work to find out something more about this new worm, among other things its geographical range, its degree of prevalence, and if it were invariably, or often, present in sleeping sickness. I procured slides of blood from hundreds of natives from different parts of Africa and of the tropical world. I found that it was only in slides procured from Congoland and from one or two other parts of West Africa that filaria perstans occurred. It does not occur, as far as I have been able to ascertain, in Egypt, in East Africa, or in South Africa. I also obtained strong evidence that it does not occur in Dahomey, nor in the Illorin district in the Niger bend, districts where, although well within the geographical limits of sleeping sickness, that disease does not originate. I also found this filaria in blood sent me from cases of sleeping sickness on the Lower Congo. But when I came to investigate the degrees of prevalence of filaria perstans in its geographical area, I seemed to find too much. I found that quite 50 per cent. of the healthy inhabitants of Congoland and of some other West African places harboured this parasite. This fact of course told against the theory that the new filaria stood in causal relationship to sleeping sickness. After all, their relationship might only be one of concurrence—a very different thing from cause and effect. In other respects, however, the new filaria seemed to fulfil all the requirements. It could be acquired only in a very limited area; it could be carried in a patient's body and live in a foreign country for years. I found it in the blood of a negro who had not been near Africa for over six years. Like other parasites of its class it could very well exist without causing disease. From its nature we know that it cannot spread by direct infection, but that it must first pass through the body of another animal; and, for the same reason, that it cannot be introduced and become domiciled in a virgin country where this other intermediate animal does not occur. We come to this then, that though there are some facts pointing to an intimate relationship between the parasite and the disease, yet the extreme frequency of filaria perstans, and the relative rarity of sleeping sickness in the endemic area seem to be strong arguments against this parasite being the cause of the disease.

At the same time we must bear in mind that there are many parasites which, though sometimes pathogenic, are nevertheless generally innocuous. Filaria nocturna, for example, does not by any means always give rise to elephantiasis; the cysticercus does not always become lodged in the vitreous humour and destroy the eye; distoma Ringeri does not always stray to the brain and cause Jacksonian epilepsy; hydatids do not always produce disease of the lungs or liver; so it may be with filaria perstans. It might very well be that it is only in a certain possibly small proportion of instances that it gets into a position to damage the encephalon. Similar parasites are known to be great travellers and to hunt each other, as it were, through the tissues of the body. Filaria loa, for example, may be felt one day in the connective tissue of a finger, and a few days later may be crossing the eye under the conjunctiva; in fact, it can be seen thus travelling about. The guinea-worm is also a notorious traveller; so are the young trichina and hundreds of other parasites. It is therefore quite in conformity wtth the teachings of analogy that filaria perstans may occasionally wander into some tissue, either in the brain or connected with the brain, and so in certain individuals set up serious cerebral disease, whilst in other and in the majority of cases it does not do so. This is quite conceivable. The fact of the presence of filaria perstans in a large proportion of people who may never suffer from sleeping sickness is therefore not an insuperable obstacle to accepting it as a cause of this disease.

There are several other facts, however, which seem to militate against this hypothesis. There may be an explanation for them, and the hypothesis suggested by geographical distribution and the presence of filaria perstans in so many instances of sleeping sickness may be perfectly correct; nevertheless, I think it right to mention that filaria perstans occurs, apparently, all over the Congo valley, while sleeping sickness is confined to certain villages and districts; moreover, it tends to occur in outbreaks which from time to time assume epidemic characters. If filaria perstans be the cause of sleeping sickness, we should expect the distribution of the disease to be in closer conformity than it seems to be with the prevalence of the parasite.

Then in sleeping sickness the lymphatic glands, as I have mentioned, are frequently enlarged. It is difficult to see how this clinical fact is to be explained on the supposition that the parasite acts pathologically primarily on the encephalon.

The pruritus, which is so marked a symptom in the disease, may be the expression of a neurosis; still, for this clinical phenomenon also, such an explanation is not entirely satisfactory.

If filaria perstans be not the cause of sleeping sickness I cannot suggest any other cause. There are sufficient grounds, at any rate, for regarding it as a possible cause, and for following up the clue which its occurrence in this disease clearly affords.

Conclusion.

The working hypothesis suggested by the facts—analogical, clinical, and experimental—is to this effect: That the germ of sleeping sickness operates primarily on the encephalon; that this germ is possibly filaria perstans; that the parasite in its wanderings, either by entering the brain, or by interfering more or less directly with its nutrition, may gradually bring about a cessation of its function, ultimately leading to secondary neuro-muscular malnutrition and symptoms of sleeping sickness. If it can be shown that filaria perstans is the cause of sleeping sickness, the next

step will be to ascertain the life-history of this parasite outside the human body; this once known, it will become easy to indicate an efficient prophylaxis.

Treatment.

Sleeping sickness, so far, has proved incurable. The natives in some places excise the enlarged cervical glands. When such an operation has been followed by apparent recovery, doubtless there has been a mistake in diagnosis. I heard of what appeared to be a case of incipient sleeping sickness which was cured by large doses of arsenic; in this case, also, diagnosis may have been at fault. In the early stages of the disease purgation and tonics, the clearing out of intestinal parasites, and similar subsidiary measures, do good temporarily. The younger of our patients certainly derived benefit from a liberal dosing with thymol; it rid him of a large number of round worms and ankylostomata. At present he is rather better than when he entered the hospital, but we may not expect ultimate recovery. Some time ago hypodermic injections of testicular fluid seemed to give encouraging results in certain cases on the Congo; later experience, however, has not been favourable to this remedy. At present our elder patient is taking a preparation of pituitary gland. The younger is on arsenic, which we propose to push. I am bound to say that though I hope for permanent benefit from these measures, I am very far from expecting it.

Paper read at the Annual Meeting of the British Medical Association.

THE UNCLASSIFIED FEVERS OF HOT CLIMATES.

By A. Crombie, M.D.

Brigade Surgeon, Lieut.-Col. I.M.S.

In order that a discussion such as I have now the honour to introduce may be pursued with profit, it is necessary, I think, to define its object and limit its scope; and this seems to be the chief purpose which I ought to hold in view in this paper. It may at first sight seem a somewhat elusive one because of the fluid character of the phenomena which we are met to consider, namely, the unclassified fevers of India—a title which implies that they are undefined, if not undefinable—and it may seem paradoxical to attempt to classify fevers which are grouped under such a heading. But it appears to me to be impossible to move a step in this discussion without making at least a provisional classification of these fevers, and placing them with reference to other fevers of a more definite and settled character. Such an attempt is absolutely necessary in order that those who take part in the discussion may have a clear conception of what fevers are included among the unclassed groups, and may make use of the same terms with regard to the same phenomena. When this is done it will probably be seen that there is really no great fluidity, and that certain fairly well-defined febrile phenomena crystallise out, which it will be possible to weigh and discuss.

A study of what has been written by the older authorities shows that they also grappled with the fevers of warm climates, and were able to divide them into clinical groups, though it is not always easy to follow the language in which they express themselves. But since their time certain fevers have been completely separated from the rest, and our task, that of analysing the residue, has by so much become comparatively easy.

In the time of Annesley and Twining the great division between enteric fever and typhus had not yet been clearly distinguished, and they and their contemporaries were not able to separate with precision typhoid, which existed in their day much as it does now, from the continued and remittent forms of malarial fever; though it is evident that both of these writers, and especially Twining, saw that certain cases of fever occurring in the cold season differed from other continued or remittent fevers by their insidious course, and by the presence of what we now know were the characteristic lesions of typhoid. There is now comparatively little difficulty in distinguishing typical typhoid from the other fevers with which it was formerly confounded in Europe as well as in India.

The older writers, again, did not have at their disposal the diagnostic and curative effects of quinine to help them; and these, together with the recent discovery of the malarial parasite, place us in a position of the greatest advantage in the task of sorting out malarial from other fevers; and, thirdly, we have the further advantage of the work of Bruce, Gipps, and Hughes, who have opened our eyes to the existence of Malta fever as a distinct clinical entity, possibly of wide distribution.

It is with the residue left by these workers that I propose to deal to-day, and the object to be gained, at least to be aimed at—and, no doubt, ultimately gained—is to focus the attention of pathologists on these other groups of fever which seem to some of us to be equally well defined clinically, and to be calling for investigation.

In order to show the place in nature which these unclassified fevers seem to occupy, I have drawn up a provisional classification of the fevers met with in the tropics and placed them in it in the position into which they seem to fall most naturally—though, of course, further study will no doubt modify our conceptions with regard to them very considerably.

A Provisional Scheme Showing the Probable Position of the Classified Fevers of India:

I. Non-specific fevers of doubtful causation, probably climatic:—
- (*a*) Ephemeral fever.
- *(*b*) Common continued fever.
  - (1) Febricula,
    - var. Nakra, or nasha fever.
  - (2) Simple continued fever.
  - *(3) Ardent fever.
- *(*c*) Thermic fever, siriasis, heat apoplexy.
- *(*d*) "Low fever."

II. Specified fevers, of known or unknown origin:—
- (1) Aphthous fever.
- (2) "Milk sickness."

*(3) Urban continued fever ("bastard typhoid").
(4) Enteric fever.
*(5) Non-malarial remittent.
(6) "Malta" fever.
*(7) Double continued fever (Manson).
(8) Relapsing fever.
*(9) Acute febrile icterus.
(10) (Yellow fever.)
(11) Beri-Beri.
(12) Cerebro-spinal fever.
(13) Typhus fever.

III. Malarial fevers:—
(1) Intermittent { Quotidian. Tertian. Quartan.
(2) Remittent or continued (Laveran).

IV. Fevers of compound origin:—
*(1) Typho-malarial.
*(2) Kala-Agar.
*(3) Hæmoglobinuric fever (?).

Such is, I think, a fairly complete list of the ordinary febrile types met with in tropical and sub-tropical countries; but as some of them are of well ascertained causation and will not detain us, I need not mention them except when the question of diagnosis arises. In this way we get rid at once of the great group of the malarial fevers, and also of relapsing fever, yellow fever, typhus fever, cerebro-spinal fever, and beri-beri, and also of enteric fever, except that very important questions arise regarding its diagnosis and incidence among native races. Aphthous fever, and "milk sickness" will require only a passing remark, and to-day's discussion will become in this way limited to those fevers included in groups I. and III., and to those marked with an asterisk in group II., namely, to the non-specific fevers, probably of climatic origin—to what I call Urban continued fever, non-malarial remittent, "Malta" fever, the "double continued" fever of Manson, and acute febrile icterus. The fevers in group III. need hardly delay us.

Climatic Fevers.—With regard to the first group of fevers, the non-specific fevers of doubtful causation, probably climatic, and which for shortness I will speak of as climatic fevers, we are at once met by the fact that Laveran, with the exception of ephemeral fever, altogether denies their existence. This is what he says: "During my stay in Algeria I searched in vain for climatic fevers, and I have come to the conclusion that these fevers do not exist, at least as separate morbid entities." He does not say on what grounds he was led to reject their existence, but as in the chapter in which this sentence occurs he insists on the discovery of the Hæmatozoon by the microscope and the efficacy of quinine as the only trustworthy guides in the diagnosis of malarial from other fevers, I presume that he founded his opinion on these means of diagnosis. Norman Chevers ("Diseases of India," 1886, p. 216) is also inclined to deny their existence. He says, with the emphasis imparted by italics, "*No case of simple continued fever or of ardent fever ever fully developed itself under my observation.*" And on the following page he says: "Assuredly he who, in Bengal proper, treats all cases of ordinary fever (excluding jungle fever, enteric, and the exanthemata) with quinine as soon as perspiration shows itself, will find febricula a very ephemeral disease indeed, and that his experience of common continued fever and ardent fever is extremely small. I am confident that these three diseases, or, to speak more accurately with Dr. Morehead, these three degrees of the same malady, are in Lower Bengal the most usual types in which intermittent fever first attacks the robust, full-blooded, and perhaps free-living European, who arrives there during the hot weather. With the exception of these two authorities, all the other writers I have consulted agree in recognising these fevers as constituting a distinct group of fevers, possibly, and I believe probably mere variations in intensity of the same malady as Morehead asserts, but distinguished by their characteristic course, and by the effect of treatment from any form of malarial fever. I do not know what occurs in Algeria, but I am confident that in Lower Bengal and Burma, where my experience was chiefly formed they constitute for eight or nine months of the year the prevailing type of fever and that they are not malarial in their essential nature. I have arrived at this opinion not only from the clinical picture which they present, which is one of a continued fever of short duration, without intermissions or remissions, but from the fact that they are not amenable to treatment by quinine; and also from a microscopic study of the blood. I do not know of any researches conducted in Lower Bengal with this in view, except those of Dr. Maynard and myself. Fevers of this type are especially prevalent in the spring and hot weather before and during the early part of the rains. During February and March of 1895, I examined the blood of fifty patients admitted to the European General Hospital, Calcutta, all suffering from fever, and found the malarial parasite present in only four of them, *i.e.*, in less than 10 per cent. Maynard, who continued the observations in the Medical College Hospital during the succeeding hot months of the same year, and who was able to devote more time to the research than I was, found them in 28 per cent. of the cases. At another season of the year, that is in the months of September, October and November, I found them in some 75 per cent. of the cases admitted, but then the type of fever was distinctly different; they were malarial in character, and amenable to quinine. It is difficult to understand Chevers' contention. He says he was accustomed to give quinine in these cases "as soon as perspiration shows itself after the first paroxysm." But the cases we are now considering are not paroxysmal; they have no intermissions or remissions more than is usual in all febrile states, the ingress is usually gradual though often sudden, with a temperature steadily rising for a day or two, a stage of high fever lasting a few days, and a gradual defervescence. The attack consists of one febrile wave lasting from one to four or ten or more days, and the end of the first paroxysm is the end of the attack. You may give as much quinine as you like during the persistence of this febrile wave, I have sometimes given ninety grains a day, without in any obvious way influencing the progress of the attack except to add considerably to

the distress of the patient. It may be well to give quinine, as Chevers recommends, after the attack is over, as a safeguard against malaria, but it is not necessary, and I have treated many such cases to a successful issue in the average time without the exhibition of a single grain of quinine.

Jacquot (quoted by Laveran) distinguishes these fevers from malarial fevers:—(1) by their epoch of development, the apogee of climatic fevers corresponding with the highest atmospheric temperatures, and preceding that of malarial fevers; (2) by their symptoms, climatic fevers being continued, and not presenting any but the usual evening rises seen in all febrile diseases; (3) by the relapses proper to intermittent, but rare or absent in the others; (4) by the cachexia produced by the one and not by the others; (5) by the anatomical lesions, the tumefaction of the spleen occurring only in malarial cases; (6) by the difference with regard to the effect of treatment. To this I would add, the ease with which the amœba of Laveran can be found in the blood of malarial fevers, and its absence or rarity in these others.

There is, however, one form of these fevers which Laveran is disposed to admit the existence of. It is what the French call *embarras gastrique*, an acute febrile condition accompanied by general *malaise*, cephalalgia, anorexia, a white-coated tongue, often nausea, and nearly always constipation. If you add to this, bilious vomiting, you have the commonest type of what we call simple continued fever. These symptoms are present in a large proportion of the cases under consideration, and often in the ardent type to a most distressing degree. With regard to these cases of *embarras gastrique* Laveran says: "The conditions under which they arise, and the influence exerted by treatment, form the best means of diagnosis when the examination of the blood is not possible." If Laveran, as he apparently does here, admits the existence of this *embarras gastrique* as a distinct form of continued fever, and if, as I believe, it is indistinguishable from what we call climatic fever, then I think he yields the whole contention, and the question becomes merely one of name.

There are, however, some authorities who speak not of climatic but of "acclimatising" fevers; and if it was of these that Laveran denies the existence, I should be inclined to be at one with him. The climatic fevers of which I am speaking show no preference for the new arrival, except in so far as he is less careful of exposing himself to adverse climatic influences than his more wary seniors.

In my experience in Burma and Lower Bengal exposure to the direct rays of the sun, exertion in a heated atmosphere in the hot weather preceding the rains, are the most common alleged causes of these fevers, and sailors "chipping" or painting the sides of ships with small black cloth caps on their heads, and no other protection from the sun during the hot part of the day, are particularly obnoxious to them; and, chiefly as the result of my own exertions in this direction, bye-laws have been introduced into the port regulations of Calcutta, forbidding the employment of seamen in such work between 10 a.m. and 4 p.m. from April to October. Chills, such as are the result of a shower of rain when men are hot, or such as result from the sudden falls of temperature that follow the "nor' westers" of March, April, and May, seem also to be sufficient exciting causes; but one of the most frequent causes among the well-to-do is the habit many men have of discarding their punkah-wallahs and sleeping in the verandah exposed to the south wind during these same months. A sudden storm of wind and rain comes up in the night, and they are chilled through before they awake, and are down with a fever of this kind for the next three or four days.

The older writers speak much of ardent fever, but the term is gradually falling into disuse, whether because we differentiate better or because these fevers are less severe than they used to be, I do not know, but they are seldom nowadays the subject of much anxiety. They are generally mild in character and run a favourable course. I do not remember such a case terminating fatally in my own hands, but we do not bleed and purge as they used to do. The severe ardent fever running to a fatal termination, which the older authorities describe, was probably what I call non-malarial remittent. In accordance with the various causes to which this group of fevers seems to be due, we find that in addition to their common character of continuity with varying duration and intensity, they differ from one another in what may be called their local manifestations, which, however, bear a certain noticeable relationship to the cause in each case. When the fever is due to exposure to the direct rays of the sun, the cephalalgia is usually intense, and sometimes very persistent, and often out of all proportion to the febrile disturbance. Giddiness and syncope are occasionally the earliest symptoms, and I have known a sailor faint and fall from the scaffolding on a ship's side into the river and be drowned from these effects of the sun before reporting himself sick. In other instances nausea and vomiting are very urgent, and in others diarrhœa, especially where they can be traced to dietetic errors. I have found this in adults, but the last is especially noticeable in children, as is well known. In other cases a careful examination of the lungs will discover patches of congestion, or even a fugitive pneumonia, not only in those which are due to a chill, but more frequently, perhaps, in those due to exertion, say, in the heated atmosphere of a ship's hold, the highest expression of this condition being the pulmonary apoplexy of thermal fever or siriasis. Some slight tenderness over the liver is also common. Some practitioners look on these local manifestations of congestion of one or other of the internal organs as the cause of the fever, but I think it is more philosophical, and more in accordance with modern thought, to regard them as symptomatic of the general condition, the particular congestion manifested being determined by the two factors, the exciting cause and individual predisposition. I do not think it would be more reasonable to attribute the febrile condition in these cases to the varying congestions than it would be to attribute the curve of temperature in typhoid fever to the ulceration of Peyer's patches. Here, then, we have a distinct group of fevers varying in intensity from ephemeral fever of a day's duration, with hardly any disturbance of the general health, through febricula,

simple continued fever, and ardent fever, with varying incidence and severity of organic congestions up to sunstroke, with intense cerebral congestion and pulmonary apoplexy. They form an unbroken series with essentially identical phenomena, but of widely different intensity, and I would agree with Morehead and Chevers in regarding them as of essentially the same nature. I am aware that heat apoplexy or siriasis, to use the word revived by Manson for thermal fever, is regarded by some as an infective disease. But I do not think the reasons convincing, and prefer to leave this fever where it is, as one of the climatic group. But I would retain the classical names they have received as a convenient way of indicating their severity only, and I know of no better way of distinguishing them from malarial fevers on the one hand and specific non-malarial fevers on the other than by classifying them in accordance with their apparent causation as "climatic fevers."

"*Low Fever.*"—There is, however, a fever which I have included in this group which does not belong to the foregoing series. I have included it among fevers due to climatic conditions because it is apparently not malarial, and it is certainly not specific; and my reason for thinking it climatic as to causation is that the only treatment that seems to affect its progress is alteration of the climatic conditions. I refer to what is called "low fever." It is the name which patients themselves give to it, and it is descriptive without committing one to any theory. These cases differ widely from the preceding. The patient is hardly ever able to tell you when his illness began. He comes to you complaining of being out of sorts and good for nothing; he has lost his appetite, and is tired all day long, and his work is a burden to him. He may have a certain amount of headache, and there may be a sort of bilious diarrhœa. If you take his temperature you will probably find it a little over 100° F. After a day in office it may rise to 101·5°, and in the early morning it may fall to 99°; but it ranges between these two extremes, never rising above 101° if you keep him in bed, and never coming down to normal. You may load him up with quinine, and you may dose him with arsenic, with the only results of disordering his stomach, increasing his headache and his tendency to diarrhœa. At the end of some weeks of this futile treatment—and quinine is often given in these cases with astounding and blind persistence, notwithstanding its obvious inefficacy—you may, if he is still your patient, recommend a change of air, and send him to the hills or to sea according to the season of the year. If you send him to Darjiling he loses his fever at Kurseong *en route*, and he never has a touch of it again. If you send him to sea he loses it at the Sandheads at the mouth of the Hughli, and comes back from Madras in a week perfectly well.

I do not know whether you would find hæmatozoa in the blood of these cases or not. It is not impossible; just as they have been found in the early days of typhoid fever and other maladies where they may happen accidentally to be present, but I think that the resistance of this kind of fever to the most persistent and heroic use of quinine shows that they are essentially not malarial. The spleen is never enlarged no matter how long the fever lasts, and though the patients get weak and thin from the long-continued febrile state, with its loss of appetite and possibly diarrhœa, they never put on the features of the malarial cachexia. That it is not a specific fever is shown by its irregular and uncertain duration, and by its being instantly cured by change of climate, which is not the case with Malta fever, the only disease with which it could possibly be confounded. Besides this, those who practise on the Mediterranean littoral, where it is also found, are able to differentiate this form of fever from true "Malta" fever (*vide* Hughes in the *Lancet*, March 2, 1895). Manson ("Tropical Diseases," p. 216) has also recognised it from my description as occurring with the same essential characters in China. I am therefore inclined to regard it as a distinct clinical entity; anyhow, there is no difficulty in recognising a case of it when you have once seen it; and I believe it is due to some obscure effect of climatic conditions which persists as long as the patient submits himself to them, and ceases with the cessation of the cause.

Nakra or Nasha.—I have included this form of febrile attack in the group of climatic fevers in deference to the opinion of many excellent native practitioners, who regard it as a distinct clinical entity. The people of Bengal do not question its existence, and attacks of "Nakra" are of frequent occurrence. It is a febricula of moderate severity, with all the general symptoms of pyrexia, but characterised by tumefaction of the Schneiderian mucous membrane. There is not the catarrh of eyes and nose such as one sees in an ordinary cold in the head, but the mucous membrane is swollen and dry. It is usually treated by local loss of blood by puncturing with a needle; but, as the patient generally performs the operation himself, the relief sometimes appears to follow the production of an extraordinarily small quantity of blood. Many practitioners believe it be only a mild febricula, with nasal swelling, and that it would get well in an equally short time even if the pretence of puncture were dispensed with. I am not prepared to come between the disputants.

(*To be continued.*)

THE MICROBE OF YELLOW FEVER (*Medical Record*, January 29, 1898).—At a meeting of the Medical Society of New Orleans, Dr. Paul Archinard, of Louisiana, delivered a lecture on his investigations during the recent epidemic of yellow fever. The experiments were conducted by Dr. P. Archinard, assisted by Dr. R. S. Woodson of Johns Hopkins University, and Dr. John J. Archinard. The lecturer announced that in a large proportion of the cases examined they had found the germ discovered by Sanarelli. They had also successfully applied Widal's method in yellow fever. By means of this experiment, employed also in the case of dengue, they found that occasicnally dengue and yellow fever were produced concurrently in the same individual.

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THE

Journal of Tropical Medicine

DECEMBER, 1898.

MEDICAL MISSIONARIES.

COMMUNICATED.

THE study of tropical disease and the opportunity for supplying the knowledge by which this study may be advanced, seems especially to belong to those who occupy the outposts of civilisation, in Africa or the East, and whose work sets them down among savage tribes or in half-explored countries. None answer to this description more than medical missionaries. For them an acquaintance with tropical maladies is essential, and by them, if they are adequately trained for scientific observation, much valuable material for fuller knowledge may be acquired.

THE JOURNAL OF TROPICAL MEDICINE should prove to these the very organ they require. It will give them the information they need, and offer them a channel whereby their observations and researches may be communicated to the public. The misfortune is that the class of men described is at present a very small one. It has been found, in the past, very difficult to secure anything like an adequate supply of men specially prepared, as well as properly endowed, for this medical mission work. That the want is a real one cannot be questioned for a moment. Applications from Bishops stationed abroad and in the Colonies, and from the great Missionary Societies, alike frequent and urgent, are being continually made. Many of them have been addressed to the Guild of St. Luke, which has at last resolved upon trying to grapple with the problem. The Guild is about to establish a Residential Home for those who desire to enter seriously on the training which will fit them to be medical missionaries. While attending the ordinary course of instruction, at one of the Medical Schools, these students would have facilities given them for special training in tropical medicine and be surrounded with such counsels and influences as would guide and encourage their missionary aims.

In Edinburgh a college of this type has existed for many years and has, unquestionably, done good work. It is the ambition of the Guild of St. Luke to establish a similar home in London.

The demand for medical missionaries is greatly in excess of the supply. In China alone there is scope for hundreds of such men to wage war against disease and to save the suffering Chinese from their native doctors, whose favourite method of treatment is to exorcise the disease with red hot irons. In Corea, again, the demand is great. Five years must elapse before an Englishman can learn the language sufficiently to teach the people, whereas the doctor begins to minister to the suffering directly he arrives amongst them.

The idea that a man can master two learned professions and take simultaneous charge of the bodies and the souls of men is becoming discredited. The engrossing and comprehensive character of a medical education renders it impossible to spare time for pastoral theology, whilst clerical education is apt to suffer if an attempt is made to combine with it an adequate training in medicine. Though it is, of course, desirable for everyone who goes to foreign lands to have some rudimentary acquaintance with the laws of health, such as may be given in an ordinary "ambulance course," it is futile and may be disastrous to supply a man or a woman with a medicine chest fitted with concentrated drugs of dangerous potency without giving him or her that know-

ledge of anatomy, physiology, chemistry, pathology, medicine and surgery which could alone safeguard its proper use.

We have seen in Jerusalem, at the Ophthalmic Hospital, a devoted surgeon and his wife manage practically "single-handed" and without any paid official, a hospital containing twenty beds, with a large out-patient department, and the amount of suffering relieved by this devoted couple, at a cost of 5d. per day per head, it is impossible to estimate. We have seen, too, the doctors at Jerusalem and Damascus, at Haifa and Jaffa, performing prodigies of skill and drawing from the people a like devotion. We have seen the doctors in Egypt and in South Africa showing forth the spirit of loving helpfulness to the leper and to the starving, suffering natives.

Reward does not come in the shape of wealth, but the income of these missionaries is adequate to their needs and the sense of freedom from financial care is far greater than among many of their professional brethren at home.

Such pioneers "have need of patience" and must be resourceful, but they have their reward, not alone in the affection of those to whom they minister, but in the scientific interests which surround them. They can watch the course of undescribed diseases, can estimate the influences of causes and effects, and if they possess a practical knowledge of bacteriology they find new and unexplored fields for investigation.

Some may be the founders of hostelries for the training of native helpers, others collect medical plants or register meteorological and other scientific phenomena. Then, too, there may be some who, by the exercise of sanitary knowledge, may help to arrest the terrible mortality which has affected many mission fields and settlements, who may devise means for the purification of the Stations, and establishing, perhaps, sanatoria on high ground for the hottest season, or organise systematic drainage for less favoured cantonments. Every great Missionary Society should have its sanitary officer, responsible for the arrangements of the Stations, for securing necessary change for the missionaries and for the arrest of all preventible disorders.

We cannot expect another Edmund Parkes, but men are to be found fired by his enthusiasm and furnished with the equipment of expert knowledge he did so much to secure. Take the single case of blackwater fever now so disastrously rife in Central Africa, the unravelling of the mysteries of this one disease may be the means of saving thousands of useful lives. The appreciation of the knowledge lately acquired of the relation of the mosquito to the distribution of malarial disease, may be the means of rendering healthy, places which are now hot-beds of fever.

The capacity to compass such ends and enterprises as these cannot, of course, be acquired by those whose medical education has been partial and incomplete; nor can we expect men to give up the comforts and prospects of life in England unless they are fired with the missionary spirit, and touched with the enthusiasm for humanity.

Those having this spirit, however, need not propose to themselves the double office of priest and doctor. What is needed is a sympathy for the work of missions, and a desire to make professional skill an instrument for the amelioration of the sufferings of the heathen world.

It is no use in the day in which we live to struggle against the overwhelming tendency which makes for specialisation. It dominates industry, art and science. But we may watch against its exaggeration. One palpable disadvantage of this modern rage for specialisation is that a man is apt to throw himself with such ardour into the particular work of his profession that he keeps no thoughts for anything beyond it.

In the Harveian Oration before the Royal College of Physicians, Sir Dyce Duckworth made this most true observation: "We are perhaps too much disposed to commemorate the scientific achievements of our great men, but let us not be unmindful of their characters. We know that genius is not always coincident with the highest moral or spiritual perfection, but when both these qualities are graciously combined in anyone we feel that we are in the presence of a truly great man, of one who becomes a personage and a power for good in his day and generation. In such a profession as ours we can never afford

to lose sight of the preponderating influence of character in all who join our ranks and have to minister to every grade of our common humanity. Yes, character must always be the mainstay and regulator of our conduct amongst ourselves and towards the public, whose servants we are."

THE TSETZE FLY COMMITTEE AND THEIR REPORT.

Two years ago a committee was appointed by the Royal Society to co-operate with Surgeon Major Bruce, in his careful studies relating to the tsetze fly. The Committee entrusted Messrs. H. A. Kanthack, H. E. Durham, and W. F. H. Blandford with the actual experimental work. Their report has now been issued, and though the investigators have been unable to discover either a prophylactic or cure for the disease, they have added considerably to the pathological side of the question, and have been able to confirm the accuracy of Surgeon Major Bruce's discovery, that a particular parasite in the blood causes the malady. The material employed for the experiments was obtained from the blood of a dog which had been infected by the disease on the way home from South Africa. It appears that sewer rats are infected with a similar parasite, so also are African rats. Surra in India is said by Koch to be due to the same parasite as that which produces the tsetze disease, but the statement rests on no experimental evidence. The Trypanosoma Evansi is doubtless the cause of surra, but there is no evidence to show that surra is caused in the same manner as the tsetze disease of Africa. The list of animals liable to infection has been much enlarged by the investigation. Cats, dogs, rabbits, mice and rats are all highly susceptible; hedgehogs and guinea pigs are affected, but the latter are more slowly attacked; hybrids of the zebra with the horse and ass are also susceptible.

It appears that the disease is not communicated by eating the flesh of an animal which has died of the disease, unless there is some lesion in the mouth or alimentary canal. The poison has to reach the blood. The virus quickly loses its power of infection; blood drawn from a living diseased animal will lose its infective properties in three or four days, while blood from a dead animal reaches that stage in the course of twenty-four hours. Nothing in the investigation has been discovered to give immunity; inoculation has been tried, bile from diseased animals has been used, and serum from diseased animals and from those that are immune, but with no result.

So far, then, no cure has been found, nor has any satisfactory preventive been discovered. Happily the bite of the tsetze fly has no effect on man, but its fatality to the horse, ox, and dog, renders it imperative in the interests of Africa that the investigations shall be continued, and let us hope with more successful results.

THE PLAGUE COMMISSION IN INDIA.

THE Plague Commission lost no time in beginning its labours after its arrival in Bombay. Mr. Wingate, Dr. Haffkine, Colonel Weir, Dr. Viegas, Captain Childe and a number of others connected with either the treatment or control of plague, have already been examined by the Commission, and a brief *résumé* of their evidence has been telegraphed to this country. As was likely to be the case, various opinions have been expressed on the several points that have come before the Commissioners. Colonel Weir, the Health Officer, was of opinion that the disease was endemic in Kathiawar, the North-Western Provinces, Bengal and the Himalayas, and inclined to the view that it was not imported. The granaries, he pointed out, were the first places infected in Bombay, and the plague was spread by rats. Mr. Vincent, the Police Commissioner, thought the disease was introduced by Sadhus, religious mendicants who had crossed from Kumaon to Nassick, and thence had come on to Bombay. Dr. Haffkine described his mode of preparing his anti-plague vaccine, and gave statistical evidence in support of his statement that inoculation suppresses in communities 80 to 90 per cent. of plague mortality, the immunity produced by the vaccine developing within

twenty-four hours. On the other hand, Surgeon-General Bainbridge seems not to have favoured inoculation, but was confident of the good results to be derived from sanitary measures. In the social conditions, however, under which the inhabitants live in a large Indian city it is particularly difficult for the sanitary measures which Surgeon-General Bainbridge advocates to be carried out. There can be little doubt that Dr. Haffkine's inoculations are gradually obtaining much favour, not only among the Indian medical profession but also among the general community.

Dr. Galliotti, of the Royal University of Florence, described the process of preparing a curative culture serum which he claims as curing as many as 75 per cent. of the patients treated. The curative serum, whether of Yersin, Galliotti, Lustig, or others, is not to be confounded with the prophylactic vaccine of Haffkine. They are totally different in their composition, mode of preparation, time of administration, and purpose for which they are administered. The serum is a medicine, used when the patient is ill for curative purposes; the vaccine is given to prevent illness. Certainly it would be good news if Dr. Galliotti's claims can be substantiated. For with a vaccine which suppresses the mortality of plague to 80 or 90 per cent., and a curative serum which will reduce the mortality of those attacked 75 per cent., plague will have lost all its former terrors. We are afraid that we are far from this desirable consummation.

PLAGUE.

News of plague reaches us from many places. Excluding India and China, plague is reported as epidemic at Samarcand and Madagascar, and cases have occurred at Delagoa Bay, at Suez and at San Francisco. The three last mentioned were merely cases on board ships, and there is no evidence that at any one place the disease obtained a footing. The Russian Government, however, view with considerable alarm the outbreak at the village of Anzob, some 130 miles south west from the city of Samarcand. A special commissioner, Prince Oldenburg, and a staff of some forty medical officers, have been despatched to the seat of the outbreak, and endowed with full administrative powers. Observation and quarantine stations have been established on the Trans-Caspian railway, on the shores of the Caspian Sea and along the various trade routes in the Bokhara and Amu Daria districts. We may rest assured that if plague can be stamped out, the Russians will have no philanthropic hesitation in dealing rigorously with the inhabitants. How the disease reached Samarcand has yet to be determined. The nearest point in India whence plague could have reached Samarcand is Kumaon or the Jellundur district, some 1,500 miles distant. Moreover, there is little or no traffic between the districts, the Himalayas intervening. It may have come by way of Thibet from Yunnan or Sze-chuen in China.

The Madagascar outbreak is eminently serious and the report that it is a mild variety counts for nothing. There seems little doubt that the disease has been carried by native craft from Bombay to Tamatave. So far as is known, plague has never appeared south of the equator before, but in the squalid surroundings of Madagascar villages the disease may be expected to find a congenial home. At the neighbouring ports in Zanzibar, Mauritius, the Cape, and Delagoa Bay, strict quarantine has been established and careful inspection of crews and passengers insisted upon.

In India plague seems to be travelling in a south-easterly direction, and whilst Bombay city and the Bombay Presidency are emerging from their third epidemic the States of Mysore, Hyderabad, the Central Provinces, and several districts in the Madras Presidency are suffering severely. The cities of Bangalore and Seringapatam are most deeply affected and several riots of considerable magnitude testify to the resentment of the natives towards municipal sanitary efforts. In the north-west and along the valley of the Indus, the third epidemic, which recrudesced with considerable virulence in the Bombay Presidency, seems to have passed lightly over.

"Imperial Penny Postage" is to come into operation on Christmas day next. The list of colonies, &c., to which the penny rates will apply is likely to include: Aden, Bahamas, British Central Africa, British East Africa, British India, Canada, Cape Colony, Ceylon, Cyprus, Falkland Islands, Fiji, Gambia, Gibraltar, Gold Coast, Hong Kong, Lagos, Leeward Islands, Natal, Newfoundland, Niger Coast Protectorate, St. Helena, Seychelles, Sierra Leone, Straits Settlements, Trinidad and Tobago, Turks Islands, Uganda, Windward Islands.

Article for Discussion.

THE POSSIBILITY OF EUROPEANS AND THEIR FAMILIES BECOMING NATURALISED IN THE TROPICS.

II.

In the previous issue of the Journal the question of European naturalisation in the tropics was raised, and the article left off with the query, "How are the Anglo-Saxon dwellers faring in the United States, in Australia, in South Africa, and in other countries to which they have emigrated?"

The United States of America reach to within 100 miles of the tropic of Cancer; Australia is traversed by the tropic of Capricorn; so that its northern half is tropical whilst the southern half is almost wholly sub-tropical. South Africa affords an example of European races living in sub-tropical climates. In all these countries it must be noted that the first settlements were always made in regions furthest away from the tropics—in fact, in regions which came nearest in climatic conditions to those of the birth-place of the immigrants. The northern part of the United States, the most southerly points of South Africa, and the southern shores of Australia are illustrations of the desire for the coolest parts of the selected regions. In these countries the cooler regions are still the great seats of intellectual and commercial activity, as Boston, New York, Philadelphia, and Chicago testify in America; Melbourne, Sydney, and Adelaide in Australia; and similarly, in the Cape the centre of government, &c., is at Cape Town, in the south. Taking British emigrants as an example, they left a country situated between 60° and 50° north, and settled in America mostly in the regions 47° or 37° north of the equator, in Australia and in the Cape between 38° and 30°, and 35° and 30° south of the equator respectively. It would seem therefore that the equator can be approached more nearly with impunity from the south than from the north, as in Australia and in the Cape, Europeans settled and lived in regions some 10° nearer the equator than in America. This is no doubt owing to the absence of land to the south and the presence of cold sea currents from the Antarctic, and the consequent freedom from continental heat. After settling in the cooler and coldest parts of these countries, the descendants of the settlers spread south and north, as the case might be, but always towards the warmer climate. No better example of an European race living in sub-tropical climates can be found than that of the Boers in the Transvaal. Although of purely European blood, they are a mixture of Dutch, German and, later on, of French immigrants. The first settlements of the Dutch in the Cape date as far back as 1652, so that for well nigh two-and-a-half centuries they have lived in a climate within 35° of the equator. Not only so, but the tendency of the Boers has ever been to advance northwards, nearer and yet nearer the equator, until, at the present moment, the countries which they occupy, the Transvaal and the Orange Free State, are sub-tropical and, in fact, tropical in part, for the tropic of Capricorn runs through the northern part of the Transvaal. Practically, therefore, they are living between 30° and 20° from the equator.

Here, therefore, is an example of a pure European race gradually advancing nearer and nearer the tropics until they have become acclimatised. Not degenerating either, for in physique, in diplomacy, in commerce, the Boer is a match man for man for his neighbours, be they black or white. The fierce native tribes the Boers encountered served to preserve their warlike instincts and to keep them a pure-blooded race. More effeminate tribes would have, in all probability, received the European settlers into their midst, and the result would have been a mixed race, with but few of the traits of the whites left. Not that there are no progeny of cross-breeds between the Boers and natives (Hottentots). Half-breeds, or griquas, are the progeny of Boer and Hottentot parents; curiously enough, they separated or were driven off by the Boers, and settled to the westward in the country known as Griqualand. It would seem, therefore, that a race of people from northern Europe became acclimatised in a region between 30° and 20° from the equator; in other

words, in a tropical or sub-tropical, as distinct from an *equatorial* region. A yet further lesson is to be deduced from the Boer emigration. At first they settled in the coolest parts of the country, not only at the point furthest from the equator, but where the coolest sea-breeze from off the Antartic ocean contributed to keep the temperature low. After a thorough acclimatisation for five or six generations, in fact, from 1652 to 1837, they advanced inland 10 degrees nearer the equator. In their new home they were cut off from the sea, and incurred a climate of a continental type in a sub-tropical region. Still did the Boer thrive, and without guessing at the future, it is an established fact that here is an European family in the seventh or eighth generation which finds life possible within a sub-tropical, and in part a tropical, area. The chief features about the Boer emigrants are:

(1) That they settled in the coolest part of the country first;

(2) That after thorough acclimatisation in a region 10 to 15 degrees nearer the equator than their birth-place, they were able to advance still nearer the equator by 10 degrees, and retain their physique and the characteristics of their race. In fact, that from being a man of a temperate climate the Boer has become capable of thriving in a region on the fringe of the tropics.

Turning now to Australia, we find, again, a settlement of Europeans in the furthest south point of the continent, at a latitude of about the same degree as the Boers in South Africa, namely, 38° or 35° south of the equator. In the southern and cooler latitudes the chief centres of activity are still found, but the modern Australian is not content to remain there. He has spread northward, and on the east coast of Australia has founded flourishing towns at Brisbane, 27° south, and Townsville, 19° south of the equator, the latter city being well within the tropics. The bulk of the Australian community may be regarded as being but in the third generation of their native-born existence, so that it is impossible to forecast what the seventh or eighth generation may be, as in the case of the Boers. There seem, however, no signs of lessening of vitality; the population does not increase with the rapidity we are familiar with in the United States of America, but it is quite as rapid as in Canada or South Africa. It is not so much the question of the race becoming unfruitful as it is limiting the death rate of young children. In Canada the difficulty of rearing children is perhaps greater than in Australia, for the intense cold of winter and the fierce heat of a portion of the summer are very hard on the young. In Australia it is heat that has to be fought; not the moist heat of the equatorial zone so deterrent to European vitality, but a dry heat, which whilst it brings with it many physical discomforts, in no way saps the strength of the whites as does the vapour-bath climates around the shores of the Indian Ocean. The Australian in the south is becoming gradually acclimatised, and in time will no doubt thrive further north, and be able to continue his species in a climate 25° nearer the equator than that of the country of his origin. The Australian will no doubt alter, has no doubt altered in physique, with the climatic change, but although altered, that need not, in fact, does not, imply deterioration. The Australian is perhaps more "finely drawn" than his prototype in Britain, but we have evidence of his endurance in the cricket-field, his prowess in rowing, and his courage and soldierly qualities in the Suakim campaign of the "eighties." His commercial capabilities are keen and his intellectual abilities of a high order. In fact, the Australian gives every promise that his race will continue, altered, improved, it may be, but certainly in no sense is there evidence of deterioration. J. C.

(To be continued.)

THE importance of furthering the study of tropical diseases amongst English medical men is being undoubtedly recognised. A few years hence and English people will be surprised that the necessity was so long unrecognised by a nation who sends so many of its population to unhealthy tropical regions. At the annual dinner of the Royal Southern Hospital, Liverpool, Professor Paterson proposed that a scheme such as is proposed at the Seamen's Hospital, Greenwich, should be adopted at Liverpool, and it was announced that money had already been contributed for the purpose.

Recent Literature on Tropical Medicine.

OPHTHALMOLOGY IN ITS TROPICAL BEARINGS.

ACUTE INFLAMMATORY GLAUCOMA INDUCED IN A SUSCEPTIBLE PATIENT BY DENGUE.—Under this heading Dr. J. Lockhart Gibson, of Brisbane, relates a case of glaucoma coming on in the left eye in a lady aged 58 (*Australasian Medical Journal*). The patient was attacked by dengue on January 25, 1898, and next day had pain, watering and dimness of vision in the left eye. She was first seen by Dr. Gibson on the 28th—three days after the commencement of the dengue, two after onset of eye symptoms. He then found general injection of the left eye, cornea hazy, aqueous turbid, anterior chamber shallow, pupil mediumly dilated and quite insensitive to light, tension + 2, faint red reflex only from fundus owing to opacity of media, vision = fingers at two feet, field very restricted. She had been successfully treated (by iridectomy) for chronic glaucoma in the other eye in November, 1892, more than five years before.

Owing to the general prostration due to the dengue, Dr. Gibson postponed operative treatment till February 2—five days after he first saw the patient, seven after the onset of glaucoma—contenting himself in the interim with instillation of eserin into the affected eye and treating the dengue by salicylate of soda; under this treatment circum-orbital pain practically disappeared and vision improved to fingers at six feet. On February 1, temperature fell to normal, and next day a small iridectomy was done. The case did well; vision is now $\frac{6}{9}$, and a few letters of $\frac{6}{5}$; tension normal; field of vision slightly contracted, on nasal side only.

Dr. Gibson thinks the case particularly interesting (1) because the attack was clearly induced by the dengue, and (2) because an iridectomy in the other eye, performed more than five years previously, protected that eye from joining in the attack.

Judging only from the published facts of the case, I cannot help thinking that the conclusion that the glaucoma was "clearly induced" by the attack of dengue is based on very insufficient evidence, and am inclined to believe that the concurrence of the diseases was accidental. In the *malaise* preceding, and the pain and fever accompanying, the onset of dengue, the prodromal and initial symptoms of glaucoma might easily pass unnoticed; and the history of glaucoma in the other eye five years before seems to me to be against the contention instead of in favour of it.

Be this as it may, there can be only one opinion as to the extreme interest of the case and the remarkably successful result.

AQUEOUS EXTRACT OF SUPRA-RENAL CAPSULE IN TRACHOMA.—The fashionable "organotherapy" seems to have invaded the domain of ophthalmic surgery. Dr. Kyle, writing in the *Therapeutic Gazette* (Philadelphia), is enthusiastic in his praises of supra-renal extract as an external application in ophthalmic practice. Used in the form of a 2 or 4 per cent. solution it appears to act as a powerful local astringent, one or two drops instilled into the conjunctival sac causing sudden pallor of the conjunctiva, persisting one to two hours; no effect on the pupil has been observed. Dr. Kyle considers it "especially indicated in cases of chronic trachoma characterised by marked vascularity and lachrymation; in pannus, lachrymal inflammation, acute conjunctivitis, panophthalmitis, and iritis." One learns from sad experience to accept the statements of enthusiastic supporters of new remedies for trachoma with a certain amount of reserve; but a method of treatment which can be of value in "panophthalmitis" and iritis, as well as in trachoma and acute conjunctivitis, would appear to be deserving of trial.

THE CURE OF TRACHOMA.—In a paper with the above title, in the American *Journal of Eye, Ear and Throat Diseases*, Professor Angelucci, of Palermo, gives a detailed account of his method of treating trachoma, a method for which he claims success in the most obstinate cases. Much of this success is doubtless due to the provision made in most Continental eye hospitals, public and private, for the careful and systematic carrying out of every detail of treatment by trained attendants. Professor Angelucci describes the treatment under two heads: (*a*) surgical procedure and (*b*) copious lavage. "Surgical procedure" consists in superficial scarification, and "brossage" by means of the spoon in the proliferous form, and squeezing of the granules in the gelatinous form—a 4 per cent. solution of cocaine being instilled six or seven minutes prior to its application. In addition the lids and eyes are washed thoroughly, copiously, and frequently with 1 in 2,000 corrosive sublimate solution. When surgical treatment ceases to be necessary, tannic acid in glycerine—1 in 10—is instilled twice a day and the "lavage" continued. In cases where the surgical treatment and lavage bring on momentarily irritative symptoms a cold compress of 1 in 10,000 corrosive sublimate solution is also employed.

THE TENSION OF THE EYE IN IRIDO-CYCLITIS.—An interesting communication on this subject was read at the October meeting of the Ophthalmological Society by Captain Herbert, I.M.S. The conclusions arrived at are (1) that in the majority of attacks of irido-cyclitis tension is reduced; (2) cases in which tension is increased owe their origin either to copious exudation blocking the normal outlets of the anterior chamber, or to a predisposition to glaucoma; and (3) there are no grounds for attributing high tension to cyclitis as distinguished from iritis. Captain Herbert's paper was based on 144 cases of irido-cyclitis observed in India.

M. T. YARR,
Major R.A.M.C.

INDIA.

ELEPHANTIASIS IN CEYLON: ITS PREVALENCE IN VILLAGES NEAR THE SWAMPY GROUND OF OLD ABANDONED TANKS. By HAYMAN THORNHILL, M.D., Senior Medical Officer, Northern Province, Ceylon.

1. During 1894 and 1895 I had been engaged in examining day and night-blood from inhabitants of many districts of different provinces of Ceylon, but in none had I found the filaria embryos except in night-blood from inhabitants of Galle and Colombo, in both of which places elephantiasis is endemic and has been so for ages; thus, again, the co-existence of filaria nocturna and elephantiasis was demonstrated and strong support afforded us to the possible, if not absolutely certain, relationship between them as cause and effect.

2. On March 22, 1896, when visiting Batalagoda Dispensary, which is close to the Batalagoda tank, some eight miles from Kurunegalle and some forty miles distant from the sea, Mr. Parker, of the Irrigation Department, who has been resident there for some years, and who has travelled much through the villages, mentioned to me that there were many cases of elephantiasis in the villages.

3. On receipt of this information, I determined to proceed to Batalagoda again as I was anxious to satisfy myself: (*a*) Whether the cases which Mr. Parker had seen were really cases of elephantiasis; (*b*) if so, whether they were endemic or imported; (*c*) if endemic, whether there was any special cause which might be dealt with; (*d*) whether the filaria nocturna or diurna was prevalent in the inhabitants of these villages; (*e*) whether any other forms of filarial disease, such as elephantiasis of the scrotum or labia, chyluria, lymph scrotums, &c., existed; and (*f*) lastly, as to the number of villages and people affected, and as to the effect on the health and labour of the people. I accordingly went there on April 11, 1896, and, accompanied and assisted by Mr. Parker, visited several of the villages and obtained the following information, which yet again shows the co-existence, and thus points to the relationship between filaria nocturna and elephantiasis, though it shows the erroneousness of the idea that elephantiasis is endemic only within ten miles of the sea.

4. (*a*) I saw myself some twenty cases of developed elephantiasis, and heard of many more in ten of the villages immediately about Batalagoda tank, and in six of the villages about Nelligama tank, which is some five or six miles from Batalagoda, and the inhabitants of these villages informed me that such cases exist in other villages beyond theirs, viz., in villages towards Wariapolla, and near to Kurunegalla town.

(*b*) The disease is clearly *endemic* and has been so for ages. Many of the oldest inhabitants say their fathers and uncles suffered from it, and that they told them it existed in these villages from ancient times.

(*c*) There can be no doubt but that the existence of the disease was closely connected with the former condition of the Batalagoda tank. This tank was made in remote ages by the Singhalese kings, but the bund was breached hundreds of years ago and, until recently restored, the bed of the tank was merely a swampy bog containing pools or puddles in several places which were overgrown with rank vegetation; these pools contained tank fish, which the inhabitants of the surrounding villages were in the habit of catching for food. The Singhalese name for elephantiasis is "Barawa" (which means large and heavy), the people recognised that this disease was connected with the water of this tank and of similarly breached and overgrown tanks, which they speak of as "Barawa waters"; they believed, however, that the cause of the disease entered the body through ulcers on the legs of those who stood in the water when fishing; but, as many who did not fish in the tank suffered from the disease, they believed that it was caused also by eating the tank fish; this of course is erroneous, the real explanation being that mosquitos were (and, as I found, are) abundant in the neighbourhood of these old swampy tanks, and that the filaria embryos are thus conveyed to, and are most probably abundant in, the water of these tanks, and in the wells of the villages in their neighbourhood, and that the inhabitants of these villages were and are infected from these village wells, but no doubt those who occupied themselves constantly in fishing in the tank itself would, whilst so employed, drink the tank water, and would thus be additionally exposed to infection.

As I mentioned before, mosquitos of various kinds are particularly abundant. I captured specimens of four entirely different kinds, viz., (1) the ordinary large tiger or striped kind; (2) a large unstriped black kind with a long *snout;* when this mosquito alighted on a wall (or other surface) it stood out perpendicularly thus——/; (3) a very small light-coloured one, more like a gnat; and (4) a small unstriped reddish or brownish kind. I ascertained from the people of several of the infected villages that all these kinds, but especially Nos. 3 and 4, were prevalent at their villages.

(*d*) I got 15 healthy inhabitants of certain of these Batalagoda infected villages (*i.e.*, healthy in so far that they presented no signs or symptoms of elephantiasis), to come to the Batalagoda Dispensary and to sleep there, and about 10 p.m. I obtained *one* slide of blood from each; in 6 of these 15, *i.e.*, in 40 per cent., I found the filaria nocturna in abundance. It is not improbable that had I collected 2 or 3 slides from the other 9, or if blood was taken from them on another occasion, filarial embryos would be found in some of them also. However, that the filaria was found in 40 per cent. of the as yet *healthy* inhabitants of these villages shows how prevalent it is in these villages, and accounts for the great prevalence of cases of developed elephantiasis there. I also examined the night-blood of cases of developed elephantiasis, and the day-blood of 3, but in none of the slides could I find either filaria nocturna or diurna, or any kind of filaria.

(*e*) Though cases of elephantiasis affecting one or both legs, or both legs and one arm were numerous in both men, women, and children, I was unable to discover or to hear of any cases of elephantiasis of the scrotum or labia or breasts, or of chyluria, or lymph scrotum; this, however, may be due either to reluctance on the part of the sufferers to speak to me or to others of disease affecting these parts, or to the fact that they had not recognised these affections as being related to "Barawa," *i.e.*, the elephantiasis of the limbs. It is certain, however, that where elephantiasis of the limbs is so common, the other filarial affections must be more or less frequent also; and I have no doubt but that further inquiries will show this to be so.

(*f*) I have shown above that cases of elephantiasis were known to be numerous in some sixteen villages about Batalagoda and Nelligama tanks, and that it was spoken of as prevalent also in other villages beyond these. As to the serious effects of this disease on the health and labour capacity of these villages and villagers there can be no doubt; nearly all the cases of developed elephantiasis that I saw were unable to work, owing to the constant recurrence of elephantoid (I prefer the term filarial) fever accompanied with pain and increased swelling of the affected limbs; in some this fever and pain recurred every three or four days, in others once a week, and in a few about every ten days; in all it lasted for two or three days, and then subsided. I saw several in which both legs were affected, some in which one leg and one arm were affected, and two in which both legs and one arm were affected. I noticed with surprise that in no case, even in those of ten or fifteen years' duration, was there enlargement or distortion of the leg or foot or thickening of the skin to the extent to which it is so often seen elsewhere in developed elephantiasis; this was particularly noticeable in the case of a woman of Kumbalange village with elephantiasis of one leg of fifteen years' duration; the leg was considerably enlarged, but the skin was soft and not much creased or folded; this woman suffered great pain and distress, and her health was much affected by the recurrence of filarial fever and pain every three or four days. This absence of extreme enlargement and distortion seems to be associated with the constant recurrence in all these cases of this filarial fever and pain. In my experience of cases of elephantiasis attended with great enlargement, thickening and distortion, this fever is by no means frequent, occurring, perhaps, only two or three times a year, the sufferers being thus able to work during the long intervals. The frequent recurrence of the fever and pain seems to be a special feature of these Batalagoda cases, with the result, of course, that the sufferers are practically entirely prevented from working, as they all are more or less constantly sick and suffering; it may be that this is due to the constant presence in them of living adult filariæ discharging swarms of embryos into their blood (this, however, seems to be negatived by the fact that in *none* of these cases of developed elephantiasis did I find embryo filaria in the blood), or it may be that it is due to the malaria from which these people also suffer more or less constantly.

5. These villagers know the premonitory symptoms well; all gave the same account of it, viz., fever lasting for two, three, or four days, attended with pain in the limb which slightly enlarges, then subsidence of the fever and swelling and disappearance of the pain, then some days of freedom, and then a recurrence of the symptoms until at last permanent enlargement results; many who yet have no permanent enlargement of a limb suffer from this fever and pain, and know that they have got the "Barawa" disease; when these primary stage cases are added to those with permanent enlargement, who also suffer regularly every three or four days from these recurring attacks in a severer form, and when it is found that several in one family are affected (I heard of one family of eight, of whom four were sufferers), it can easily be understood how seriously the working capacity of the villagers is affected and what their sufferings are from this cause and from the malaria which is also prevalent.

6. All these villagers came to me anxiously expecting to hear of some remedy to cure the sick and to prevent the disease, but to all I had to convey the dismal intelligence that I was unable to do anything to relieve the sufferers, and but little to check the spread of the disease, as I said I feared they would not act on the only advice I could give them, viz., to carefully boil all water before drinking it. Mr. Parker, who takes the greatest possible interest in these villagers, most carefully explained for me at each village the

cause of the disease and the warning to boil all drinking water, telling them that this would also save them from much malarial fever.

7. The recently completed restoration of the Batalagoda tank, which is now filled with water, will of course in time reduce to some extent the prevalence of this disease by the removal of the swampy places and pools, the water of which was drunk by those employed in fishing there, and the channels from the tank will provide better, *i.e.*, less filaria-infected water, for *some* villages along their banks, but its effect in this way must of course be small and slow; but the indirect benefits of the wise and considerate policy that restored these tanks and thus provided water, which means food, to these miserably poor and suffering people is very great and the results are palpable and most encouraging.

8. As I said before, it is, I fear, hopeless to expect that villagers will boil all water before drinking; but in view of the great prevalence of this disease in these villages, and of the enormous amount of pain and suffering, and hence loss of labour that it causes, *I think that whatever is possible should be done to check or limit the occurrence of these cases in the future.* I therefore proposed and urged that Government should at once undertake the construction at each of these villages, and at any others where similar cases exist, of wells properly sunk, lined and *covered over*. This can, I believe, be done at a very small cost by using the large earthen rings made by village potters, which, when properly set in well puddled clay, constitutes a most effectual lining which lasts for centuries, and as one, or rather the chief of the objects is to exclude filariated mosquitos from finding access to these wells, the top ring should be of iron, or of specially thick, hardened and glazed pottery or cement, and it should project a foot or eighteen inches above the ground, and should be provided with a wooden cap or lid, and special instructions should be given to the village headmen and villagers (and special measures taken to see that these instructions *are attended to*), that the mouths of these wells are to be kept constantly covered over.

9. I here quote the remarks made in paragraph 49 of my Annual Report of 1895, viz.: "I would suggest that all medical officers should be asked to keep a register of all cases of elephantiasis, chyluria, and lymph scrotum, or other filarial disease, showing the name, age, sex, date of first symptoms, where then living, and where living previously; it would thus be possible to construct a filarial map of Ceylon, showing where the parasite was endemic, and of course to take steps for the prevention of the disease by attention to the water supply."—*Indian Medical Gazette*, vol. xxxii. (No. 10, October, 1897).

MALARIA, THE CHIEF CAUSE OF INFANTILE CONVULSIONS IN THE TROPICS. By HAYMAN THORNHILL, M.B. Senior Medical Officer, Ceylon.

The Ceylon Registrar-General gives the following statistics for 1896:—

Estimated Population	Total Deaths	Deaths of Children under 1 year (infant mortality)	Deaths from Infantile Convulsions
3,292,367	81,898	16,374	6,635
	Rate per 1,000 = 24·7		

Thus of the total mortality 8·10 per cent. and of the total infant mortality 40·52 per cent. may roughly be attributed to infantile convulsions, though, no doubt, many of the deaths from infantile convulsions occurred in children more than 1 year old.

The infant mortality rate per 1,000 births for all Ceylon was 158, with the following *highest rates* per 1,000 births in the following nine districts:—

Vavoniya, 386; Mannar, 313; Puttalam, 305; Anuradhapura, 243; Ratnapura, 215; Mullaitivœ, 213; Trincomalie, 211; Matale; 205; and Kurunegalle, 199.

These abnormally high infant mortality rates are thus seen to be coincident with the greatest prevalence of malaria, and thus strongly support the view that malaria is *the chief cause* of infantile convulsions in tropical malarial countries, and indicate that treatment by hypodermic injections of quinine should be resorted to largely *and promptly*.

Unfortunately, the preparations of quinine, viz., the bi-hydrochlorate or the hydrobromate, most suitable for hypodermic use are not usually available, but where they are not the sulphate should be used. The adult maximum hypodermic dose of the sulphate would be about half the internal dose; the maximum hypodermic dose of the bi-hydrochlorate is grs. 3; and of the hydrobromate grs. 2, and the hypodermic doses of these for infants and children would be found by the rule given at page 136 of Burroughs' and Wellcome's most welcome 1898 diary, viz., divide the age by the age increased by 12; thus for a child 2 years old $\frac{2}{2+12} = \frac{1}{7}$; thus the dose would be $\frac{1}{7}$.

The following is from page 469 of Laveran's article on "Malarial Fever" in Wilson's "Applied Therapeutics" (American):—

"Finally, one can employ, for hypodermic injection, quinine sulphate which has been rendered soluble by the addition of tartaric acid. Vinson gives the following useful formula:—

Quinine Sulph.	grs. xv.
Tartaric Acid	grs. viii.
Distilled water	ʒiiss.

"Great care should be taken to plunge the point of the needle well into the subcutaneous tissue.

"The solution of quinine employed should be clear and should contain neither crystals nor spores."—*Indian Medical Gazette*, vol. xxxiii. (No. 3, March, 1898.

CHLOROFORM ANÆSTHESIA IN INDIA.

Dr. Neve read a paper on this subject at the British Medical Association. His statistics went to show the infrequency of deaths from chloroform administration in India. In one series of administrations 2 deaths occurred in 15,800 cases during 1897; in a second series of over 78,000 cases, 3 deaths were recorded; and several surgeons have reported that in 100,000 administrations which came under their individual notice no death has been met with from chloroform. The comparative safety of chloroform seemed to be entirely due to the warm atmosphere, which favours rapid action and elimination of the drug, and it is advised to imitate in Britain the conditions of administration in India.

AMERICA, UNITED STATES.

THE BACILLUS ICTEROIDES.

IN the preliminary report, just published (November 11) by the Marine Hospital Service of medical officers detailed by direction of the President as a commission to investigate in Havana the cause of yellow fever, Surgeon H. D. Geddings states:—

"Experiments had been begun upon the toxins of the bacillus icteroides, both precipitated and liquid, when it became necessary to suspend the work in Havana and to return to the United States on March 15, 1898, since which time further investigation has been prevented by other duties in connection with epidemic work in the Southern States.

"It is here necessary to say a few words in connection with the claim of identity of the bacillus icteroides of Sanarelli and the bacillus "X" of Sternberg, which has recently been made. It is inconceivable to my mind how such a claim can be sustained by any one who has carefully

studied and differentiated the two organisms. The bacillus "X" is coarser, longer, and stouter than the bacillus icteroides; originally quite motile when isolated in Havana several years ago, it is now an organism, hardly as motile as the ordinary colon bacillus; in its growth it produces fermentation in both lactose and glucose agar, with a gaseous product quite similar in composition to the products of the colon bacillus; it produces acid in its growth; its production of indol is well marked and excessive, and lastly, it readily coagulates milk. Indeed, it would seem that the bacillus "X" is simply and solely a well-marked and accentuated colon bacillus. It is pathogenic, to be sure, for rabbits, guinea-pigs, and other of the lower animals, but the time has long since passed when the colon bacillus can be considered as harmless and non-pathogenic.

"It has also been objected that the bacillus icteroides too closely simulates the colon bacillus. To this it can only be said that in its characteristics of growth, as previously detailed in this article, there is a wide difference, nor is its similiarity nearly so great as is that of the bacillus typhosus to the bacillus coli communis. The bacillus icteroides (Sanarelli) produces toxins, precipitable by ammonium sulphate, of well-marked intensity and potency, much more so than the toxins precipitated from bacillus "X" and ordinary colon bacillus.

"The agglutination and arrest of motility experiments of Archinard and Woodson would seem to make the argument in favour of the pathogenicity of the bacillus icteroides all the stronger.

"In concluding this preliminary and independent report, which would indicate that the bacillus icteroides of Sanarelli is the specific agent in the causation of yellow fever, I would beg to recommend that opportunity be given for further experimentation on lower animals with its toxins, and with cultures if necessary, and that both be tested in connection with the antiamaryllic serum prepared according to the methods of Sanarelli."

The two members of this Commission who were forced to abandon Havana on account of the war, and have since been on duty in the Southern States, have now been ordered to return to Havana and continue their investigations, their laboratory having been undisturbed during the war.

"Boo-Hoo" Fever.

Walter F. Robinson, M.D., U.S. Medical Inspector at Honolulu, gives the following account of a disease which it would seem might be termed nostalgia, by a new name:

Shortly after the troops were settled in Honolulu there appeared among them a peculiar complaint somewhat resembling the grip. There were pains in all the bones, especially in the small of the back; generally severe headache, coated tongue, loss of appetite, and some little fever.

It might be thought that these were mild attacks of malaria, more especially as there is malaria on the island. There were seldom chills with the attack, however, nor anything typical about the fever. It would be here to-day and gone to-morrow. The attacks would not last, as a rule, more than two or three days, and then the patient would feel as well as ever.

One symptom, however, was almost constant, namely, that of general depression and discouragement. The soldier would lose all his courage and interest in his duties and in the army in general.

All his good spirits would desert him, and he would only mope and wish to be sent home.

This condition is so peculiar and characteristic that it has received the name of the "boo-hoo" fever, and as such is known all over the island. Strangers are very apt to be attacked with it, and its cause is undoubtedly the new climatic conditions in which the system of the patient finds itself.

On this account the disease is sometimes called climatic fever. The bowels are generally bound up, and it is altogether a different disease from the diarrhœa which is so common in hot climates.

Both quinine and phenacetin are of value in this disease, and as already stated, it is of short duration and yields readily to treatment. It may fairly be compared to the distemper which affects horses when they are taken from one climate to another.

The cases seemed to occur entirely in the first two weeks of our stay, and they are now seldom seen.

CHINA.

The China Medical Missionary Journal, October, 1898, published in Shanghai, gives interesting accounts of the hospital work done in some of the centres.

Hankow.—The statistics for 1897 contained in Dr. Gillison's report are as follows:—

In-patients	501
Out-patients and attendances	4,493

Hung-Chow.—Drs. Main and Kember's report contains excellent full-page photogravures. The number of patients were as follows:—

In-patients	927
Out-patients	43,168
Suicides	211
Operations, major and minor	1,924

Chung-King.—Dr. Wolfendale's report of the work done by the London Mission Hospital during eleven months shows that the number of patients and attendances were 6,350

"The Methodist Episcopal Church Hospital's Report," by Drs. McCartney and Woolsey, shows the total number of patients and visits made to be 39,181

Chefoo.—Dr. Judd gives a short review of the work at Chefoo performed by the Inland Mission since it was opened in 1879, and the following statistics for 1898:—

In-patients	212
Out-patients' attendances	23,242

Foochow.—Dr. Goddard's report of the Female Medical Mission contains the following statistics:—

In-patients	102
Out-patients and visits	5,501

Hinghua.—Dr. Taylor has been instrumental in developing a new hospital at this town, giving accommodation for 120 patients. In the first year of its existence the following were the numbers attended:—

In-patients	677
Out-patients	7,298

Ningpo.—Dr. Smythe gives the numbers for 1898 as follows:—

In-patients	295
Out-patients and visits	7,702

It will be seen that the amount of work done in these several towns and districts of China is enormous. In the reports of the seven districts given above no fewer than 185,845 out-patients and 3,536 in-patients were attended. These represent but a fraction of the hospital work going on throughout the length and breadth of China. The Chinese evidently appreciate to the full extent Western medical science in spite of all assertions to the contrary.

AUSTRALIA.

The Influence of Coitus with White Men in inducing Sterility in Aboriginal Women.

Dr. Sarsfield Cassidy, at the New South Wales Medical Council Meeting in October, 1898, read a paper on this subject. He stated that the aboriginal native women of Australia never can become pregnant to an aboriginal man after she has once borne children by a white man. Dr. Cassidy remarks that this is not owing to disease, as it has been proved, by overwhelming evidence, that a healthy

aboriginal male and female cannot beget children should the female have lived with, and borne children to, a white man. Should, however, the aboriginal female return to her white paramour, she readily conceives. The native male Australian, in common with some tribes in West Africa, does not ostracise the female who has cohabited with the white man; on the contrary, he treats her with greater respect and regard than formerly, and his amorous feelings towards her are increased not abated.

HOLLAND.

European Colonisation in the Tropics.

Professor Stokvis has repeatedly expressed his opinion that it is quite possible for Europeans to found Agricultural as well as Trade Colonies in a tropical climate, and he supports his views by statistics, the only method by which this important question can be decided. The statistics prove that during the last thirty years, thanks to the results of modern sanitation, in the British-Indian as also in the Dutch-Indian Colonial armies, the mortality of the Europeans had so considerably decreased that at the present time it is less than amongst the Natives.

As, however, the life of an agricultural labourer is less exciting than that of a soldier, the conclusion may be drawn, that the European field labourer would not be behind the native worker in power of resistance to deterrent climatic influences. In regard to the capability of endurance of women in the tropics, we have hitherto no reliable data. Experience, however, teaches us that conditions in the tropics are not more unfavourable to the European woman than to the Native female. Finally, in respect to children, the mortality of the latter in India is positively far less than it is in Europe.

It has been asserted that after a long stay in the tropics, a deterioration of race sets in and the same becomes barren. This opinion is founded on the experiences which were made during the first years of the French Colonisation in Algiers. These have been contradicted by the records of later periods. It is indeed true that in India it is rare to find European families of pure race in the third generation. But this may have its grounds in the universal wish to return home as soon as a competency is acquired, and in the fact that when children begin to grow up they are often sent to Europe.

Yet Stokvis gives a series of examples culled from Surinam, Peru, Porto Rico, &c., proving that in the tropics the propagation of the pure, unmixed race is possible, not only amongst the Semitics and Latins, but likewise amongst the Germanic people.

In this respect, the history of a little colony of Dutch agriculturists in Surinam, as related by Stokvis, is interesting. The transition from the temperate zone to the tropics and the acclimatisation, places a great strain on the human organism, which under certain circumstances may be deleterious, and it is the *rôle* of hygiene to diminish the effect as much as possible. Once, however, this period, which may last some years, is happily gone through, the European has become a tropical man.

Mountains and high table-lands where the temperature is low and malaria not to be feared, are particularly adapted for agricultural colonies. Stokvis is distinctly opposed to mass-colonisation. A company of twenty persons, friends or relations, appear to him the ideal number to be supported by Government or Colonial Companies.

In concluding his interesting publication—interesting to every colonist—Stokvis adds the following paragraphs:—

(1) The foundation and prosperity of European trade and agricultural colonies are quite possible in tropical lowlands, as also in tropical highlands.

(2) Tropical temperatures and the races of the colonists play a secondary part in the question of colonisation.

(3) Colonisation in masses, in fact bulk-colonisation, is to be avoided.

New Drugs, Instruments and Surgical Appliances.

Chinosol.—This useful antiseptic, disinfectant and deodorant has now been before the medical profession long enough to allow of a conclusion being come to as to its efficacy. Chinosol is the handiest of disinfectants and the surest of germicides. Chinosol can be used for the hands, surgical instruments, open wounds, and for intestinal, vesical, and vaginal irrigations. Being non-poisonous and non-corrosive it is the safest disinfectant for use by the public.

Review.

Health in Africa; a Handbook for European Travellers and Residents, Embracing a Study of Malarial Fever as it is Found in British Central Africa. By D. Kerr Cross, M.B.C.M., Medical Officer for British Central Africa Protectorate, with an introduction by Sir Harry Johnston, K.C.B. Numerous illustrations. London: James Nisbet and Co., Ltd., 1897. pp. 222.

In a handy volume Dr. Cross has supplied the dweller and traveller in the tropics with an excellent guide to health. The subject is dealt with in sixteen chapters. The first chapter is devoted to the Hygiene of Tropical Life, and it contains hints on food, exercise, clothing, &c., which are evidently based on ripe experience. In the five succeeding chapters the ailments affecting the cutaneous, the digestive, the respiratory, the circulatory, the renal and nervous system, more especially, as the effects of tropical life, are carefully described. Chapter vii. is devoted to the specific fevers, malaria receiving full justice. The last half of the book is taken up with "First Aid to Nursing," of which a clear and practical account is given. We strongly recommend the book to all travellers, not only in Africa, but in every part of the tropics.

News and Notes.

LONDON SCHOOL OF TROPICAL MEDICINE.

The following Advisory Committee has been appointed in connection with the London School of Tropical Medicine:—

Perceval A. Nairne, Esq. (Chairman); Admiral Sir Walter Hunt-Grubbe, K.C.B. (Deputy Chairman); Sir Edwin Arnold, K.C.I.E., C.S.I.; Surg.-Lt.-Col. Oswald Baker, I.M.S., L.R.C.P., L.R.C.S.; Robert Barnes, Esq., M.D., F.R.C.P., F.R.C.S. (Consulting Physician to Seamen's Hospital Society); Sir Chas. Gage Brown, K.C.M.G., M.D., F.R.C.P. (Medical Adviser to Colonial Office, retired); Thos. Lauder Brunton, Esq., LL.D., M.D., F.R.C.P., F.R.S. (Physician to

St. Bartholomew's Hospital); James Cantlie, Esq., M.B., C.M., F.R.C.S. (Editor of JOURNAL OF TROPICAL MEDICINE); James Cleghorn, Esq,, C.S.I., M.D. (Surgeon-Major-General Indian Medical Service); William Collingridge, Esq., M.D. (Medical Officer of Health to the Port of London); S. Leonard Crane, Esq., C.M.G., M.D. (late Surgeon-General of Trinidad); W. H. Crosse, Esq., M.R.C.S., L.F.P.S.G. (late Principal Medical Officer, Royal Niger Co.); Donald S. Gunn, Esq., F.R.C.S.; Rev. Brooke Lambert, M.A., B.C.L.; Herbert H. Lankester, Esq., M.D. (Physician to Church Missionary Society); Allan Macfadyen, Esq., M.D. (Director of British Institute of Preventive Medicine); Stephen Mackenzie, Esq., M.D., F.R.C.P. (Physician to London Hospital); Patrick Manson, Esq., LL.D., M.D., F.R.C.P. (Medical Adviser to Colonial Office); James Laidlaw Maxwell, Esq., M.D. (Secretary to Medical Missionary Association); John William Ogle, Esq., M.D., F.R.C.P. (Consulting Physician to St. George's Hospital); H. J. Read, Esq. (Colonial Office); T. L. Rogers, Esq., M.D.; Major the Hon. W. Rowley; W. Johnson Smith, Esq., F.R.C.S.; H. M. Stanley, Esq., D.C.L., LL.D., M.P.; George Thin, Esq., M.D.; Admiral Sir Richard Tracy, K.C.B. (President of Royal Naval College); Alexander Turnbull, Esq., M.D. (Inspector-General of Hospitals and Fleets, retired); William Turner, Esq., F.R.C.S.

A sub-committee, consisting of Mr. Nairne (Chairman); Sir C. Gage Brown, K.C.M.G.; Dr. Lauder Brunton; Dr. Stephen Mackenzie; Dr. Patrick Manson; Dr. J. L. Maxwell; Mr. Macnamara; Mr. Johnson Smith; Mr. Wm. Turner and Mr. Cantlie has been appointed to draw up a constitution for the School and define the curriculum.

ZULU PRESCRIPTIONS.—The Under-Secretary for Native Affairs in Natal has recently delivered a lecture on "Zulu Social and Domestic Life" in the capital of that colony, in the course of which he gave some interesting particulars concerning certain prescriptions of the Zulu medicine man: "If you have the mumps, go to an ant-bear's hole, look into it, and call out 'zagiga! zagiga!' (let me alone!), and if you return home without looking back, the mumps will leave you. If you are deaf, get a monkey's ear, burn it to ashes, mix the ashes with hippopotamus fat and beeswax, drop some of the mixture into your ears, and your hearing will soon be as good as the monkey's."

A WRITER in the *Leisure Hour*, by an industrious use of Sir Clement Markham's book on Peruvian Bark, has compiled a brief, popular history of quinine, but he appears not to have taken the trouble to read any book on the subject published less than 10 years ago, and this no doubt is responsible for the extraordinary blunder he makes at the beginning of his article in stating that Ceylon and Madras send us more Peruvian bark than all the rest of the world put together. The editor of our lay contemporary may think it only right to correct the wrong impression given his readers, and tell them that Madras and Ceylon are not in it for the supply of bark compared with Java.

SOLDIER COOKS IN INDIA.—The *British Medical Journal* draws attention to the fact that, owing to the uncleanliness of native cooks and their careless methods of dealing with food in India, Colonel Mathias, of the Gordon Highlanders, has for some time past introduced the system of soldier cooks in the battalion under his charge. The experiment gave excellent results; there were, it is believed, no cases of enteric fever among the men, and the work was done in such a manner as to give full satisfaction to the company messes. This system will, doubtless, receive a further trial.

A DEATH FROM YELLOW FEVER AT WASHINGTON.—An engineer, recently returned from the Isthmus of Panama, died recently in Washington of yellow fever.

APPARENT DEATH.—In a paper read before the New York Society of Medical Jurisprudence, Dr. H. J. Garrigues stated, "That there was only one sure proof of death and that was decomposition!"

COLONISATION OF THE TROPICS BY WHITE MEN.—In the *Indian Medical Record* of November 16, 1898, this subject is dealt with at some length. The writer disputes the theory that colonisation by the white man is possible, and refutes the argument that the microbe is the sole enemy.

A FRENCH steamer brought a patient suffering from the plague to Delagoa Bay, but the Portuguese authorities refused to allow the ship to enter the bay. The same proceeding took place earlier at Mozambique. It is reported that the ship in question, the "Gironde," has sailed for Madagascar.

A HOSPITAL for Women and Children has been opened at Jaffna, Ceylon. The funds were collected by the Misses Leitch, and the hospital handed over to the American Mission. At Inruville a hospital has also been opened under the American Mission. This latter institution has been placed in charge of Doctor Isabella Curr. At Galle it is decided to station a hospital ship for the treatment of plague cases.

Correspondence.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—I have much pleasure in sending you my name as a subscriber to your new Journal on Tropical Medicine, and also as an intending contributor, should any observations of mine be deemed of sufficient interest to merit insertion in its columns.

I anticipate that the new periodical will prove a boon to English medical men abroad, either in our own colonies and dependencies, or in situations isolated and far removed from the centres of scientific and intellectual activity. It is surprising, indeed, that the need for a medium of intercommunication between English-speaking investigators and workers in a yet little cultivated field, should not have made itself felt before now, and that the Germans should have been permitted to forestall us in the matter. I therefore wish you every success in your undertaking, and earnestly hope that the advent of your Journal may prove epoch-marking, and give a much-needed stimulus towards a more scientifically co-ordinated and directed investigation of Tropical Pathology. You do not require to be reminded of the difficulties and hindrances which beset a would-be investigator in this field. Difficulties due to

climate and distance from the "base of supplies"; lack of adequate laboratory appliances; postal arrangements; hindrances from the ignorance and prejudices of semi-barbarous or wholly savage inhabitants; impossibility of obtaining collaboration in any special investigation from fellow-workers; lack of advice, direction, and encouragement from the masters of our profession, obtainable only in the centres of light and leading. Such are some of the normal conditions of existence for many if not for the majority of your audience. You are, no doubt, fully mindful thereof. I have not yet seen any prospectus of the Journal, the advertisement in the *Lancet* only having reached me last week; but I venture to hope that one of your objects will be to facilitate the interchange of ideas between, and to direct and co-ordinate the efforts of observers so disadvantageously placed; as likewise to keep them—so far as possible under the circumstances—abreast of the scientific movement of the time in all that concerns the department of medicine within the sphere of which their lifework is cast.

FRAS. C. BROWNE-WEBBER, M.B.
Cargo de los Señores Ripley, Labern y Cia,
Calle de Maximo Jerez, Leon.
Nicaragua, C.A.

THE NEED OF A LIBRARY OF TROPICAL MEDICINE.

To the Editors of "The Journal of Tropical Medicine."

DEAR SIRS,—One of the greatest difficulties that those who work abroad have to contend with is that of access to the literature of tropical diseases. In fact it is not too much to say that a visit to England is in most cases necessary if the scattered references to any given disease have to be studied, and even then a prolonged search in one of the larger medical libraries of the metropolis will be entailed. It would, then, be of the greatest service to tropical medicine if a reference library could be started in connection with THE JOURNAL OF TROPICAL MEDICINE. If the editors would be generous enough to give such books and pamphlets as may be sent to them for review, and workers in this field would present any monographs that they may write, a very valuable and readily accessible collection would gradually be accumulated, and would soon prove to be of the greatest service to labourers in this wide and rapidly increasing branch of medical knowledge.

I am, yours faithfully,
LEONARD ROGERS, M.D., M.R.C.P.,
Indian Medical Service.

Communications, Letters, &c., have been received from:—

A.—Mr. Allan (London); Dr. R. J. Ashton (Kachwa).
B.—Dr. Robert Beebe (Nanking).
C.—Mr. T. Campbell (Kudat); Dr. Charlesworth (Zanzibar); Dr. J. A. L. Calder (Jamaica).
D.—Major P. D. Dant (Jeypore).
G.—Mr. L. M. Griffiths (Clifton).
H.—Dr. G. S. Hirst, Col. Med. Serv. (Wolverhampton); Surg.-Genl. W. R. Hooper (London); Mr. Allan H. Hanley (Bonny).
K.—Dr. William Kirk (Shanghai).
P.—Mr. A. G. Parrott (Lao-ho-Kóu).
R.—Dr. Duncan J. Reid (Shanghai).
S.—Rev. G. A. Stuart (Nanking); Dr. Sydney Senhouse (Dominica); Fleet-Surg. T. M. Sibbald (China Station); Dr. A. Syall (Swatow); Mr. Edw. Sutton (China Station).
T.—Dr. John Thomson (Hankow).
W.—Dr. M. J. Wright (Perak).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Liverpool Medico-Chirurgical Journal.
Medical Missionary Journal.
Pacific Medical Journal.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Therapist.
Treatment.

Births, Marriages and Deaths.

BIRTHS.

AT HONGKONG, on November 1, the wife of James A. Lowson, M.D., of a daughter.

AT WEI HAI WEI, on October 23, the wife of J. N. Case, M.D., of a son.

DEATH.

JENNER.—On the 11th inst., at Greenwood, Bishops Waltham, Hampshire, Sir William Jenner, aged 83.

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Notices to Correspondents.

1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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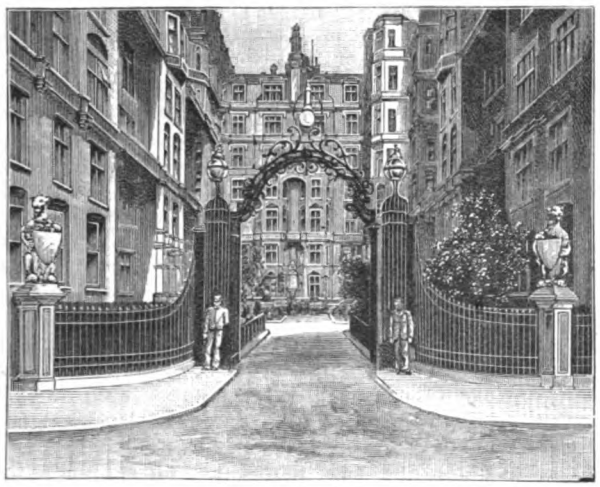

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AN ANALYSIS OF FIFTY-SIX CASES OF BLACKWATER FEVER.

By WORDSWORTH POOLE, B.A., M.B., B.C.Camb.
P.M.O. West Africa Frontier Force.

THERE must be a large amount of material concerning blackwater fever, which, if collected would be of great use in helping us to arrive at some definite conclusions on various points, but which at present is wasted. There are many doctors in the tropics who see a few cases of the disease, but thinking the number insignificant, do not record them. With the idea of drawing, as briefly as possible, some conclusions from a considerable number, the following fifty-six cases, with some details have been brought together and analysed. They are, in all cases, vouched for as undoubted examples of blackwater fever, by doctors of experience in tropical diseases.

Of the 56 cases, 42 occurred in Nyassaland during the four years 1894-5-6-7, and the details of these are drawn from notes by P. MacVicar, Dr. Robertson (whose courtesy in supplying them I would here acknowledge) and myself.

The remaining 14 occurred in members of the West Africa Frontier Force, on the Niger during the present year, 1898.

The following are the statements of some of the facts elicited by an analysis of the cases.

(1) *Death Rate.*

(*a*) Fifteen out of the total 56 died, that is 26·8 per cent.; 13 out of the 42 in Nyassaland died, that is 31 per cent.; 2 out of the 14 in Nigeria died, that is 14·28 per cent.

It will thus be seen that the death rate in Nyassaland is very much heavier than that on the Niger, though it must be admitted that the number of cases in the latter country are too few to afford reliable data; but judging from the cases I have seen in both countries, 22 in all, I believe that those in Nyassaland are of a considerably severer type than those in Nigeria.

(*b*) *Death rate of Second and Subsequent Attacks.*—Of the 56 cases, 10 were second or subsequent attacks. Of these only 1 died, giving a death rate of 10 per cent.

This bears out the accepted view that subsequent attacks are by no means so fatal at primary ones.

(2) *Length of Residence as a Factor in Causation.*

It is generally asserted that the disease rarely attacks people during their first year of residence in a tropical climate. This is not borne out by the cases under consideration.

Leaving out the 10 cases which were second or subsequent attacks, of the remaining 46 at least 17 occurred in men who had been out under one year. The number would probably be greater, for details as to the length of residence have not been available in all the cases.

(3) *Chances of a Second Attack.*

It is the duty of every doctor to warn his patients who have had one attack, that the chances of a subsequent attack, if he returns to a country where the disease is prevalent, is very great.

I cannot give exact figures on this point, but I am unable to call to mind the case of any one who, having suffered once, has escaped a second attack on returning to a blackwater fever country. It is in the interest too, of governments and companies not to re-employ any one who has had one attack. However, the smaller mortality in recurrent cases should always be borne in mind.

These considerations should be put before patients, so that we may find nothing in which to blame ourselves when, as sometimes happens, a person with a large stake in the country, weighs his chances, and in spite of the warning, returns, only again to fall a prey to the disease.

(3) *Frequency of Suppression of Urine.*

The idea seems to be popular, both with the profession and to a greater degree with laymen, that the great risk of the disease is suppression of urine. A layman, considering the state of the urine and the pains in the loins, naturally looks upon the kidneys as the organs at fault. But the frequency of the occurrence of suppression of urine is surely greatly over-estimated.

Of the 15 deaths only 3 were due to suppression of urine, and in my own cases I never observed any probability of such an occurrence. It was found very exceptional to obtain an albuminous reaction when the urine had once become clear. This shows that the damage done to the kidneys must have been very transient. On the other hand, in those cases observed

from the very commencement, it is the rule to find actual red blood corpuscles present in the urine, especially in the first two or three samples when the urine has a rosy tint, in contrast to the port wine or tarry look it assumes later; while the renal casts and amount of *débris* found, of course, point to considerable disturbance of kidney function. But the small percentage of deaths by suppression of urine affords the opportunity of more frequently assuring patients that suppression of urine, an eventuality they have been led to greatly dread, is quite uncommon.

(5) *Microscopical Appearances of the Blood.*

Experience on this point is well worth recording. The extreme leucocytosis and great variety in the sizes of the red corpuscles are well established facts; but on the question of parasites there is much diversity of opinion.

Of the cases I have examined, in a few, which were seen from the beginning, two or three small oval unpigmented parasites were found during the first few days, while on the days following none were found.

In two other cases, a small pigmented parasite was found here and there during the first two days. In three cases older parasites were found, viz:—

(1) A crescent on the second day.
(2) A spherical body on the eighth day.
(3) ditto ditto second day.

These few forms were found only as the result of a great deal of searching. In the other cases which were examined, no parasites whatever were found; but a large amount of experience is required to be sure of the small unpigmented forms, and it is with due diffidence that the inability to find them is recorded. This small find of parasites is significant; and the existence and nature of those found does not appear in any case incompatible with the theory that they belonged to a prior attack of malaria. In two cases, a few days after the blackwater symptoms had subsided, the patients had a rise of temperature, during which I found unpigmented parasites in fair number; but the attack was unaccompanied by any recurrence of the blackwater symptoms. This is a noteworthy fact. Dr. S. C. Rees, of the West Africa Frontier Force tells me that he also has observed the same thing.

In conclusion, I would suggest that your valuable JOURNAL, by taking on the function of a collecting centre, might gather together much useful information on such a debatable subject as blackwater fever, which would otherwise be lost.

SCHOOL HYGIENE IN JAPAN.—An order has been issued, says the *Allgemeine Medicinische Central-Zeitung*, by the Japanese Minister of Education that the medical officers attached to the public schools shall not only inspect the schools as heretofore at the beginning and end of the school year, but must visit them at least once a month during school hours for the purpose of noting any points of defective sanitation.

BLACKWATER FEVER, SOME CASES AND NOTES.

By G. F. REYNALDS, M.R.C.P., L.R.C.P.
Taqual, Gold Coast, West Africa.

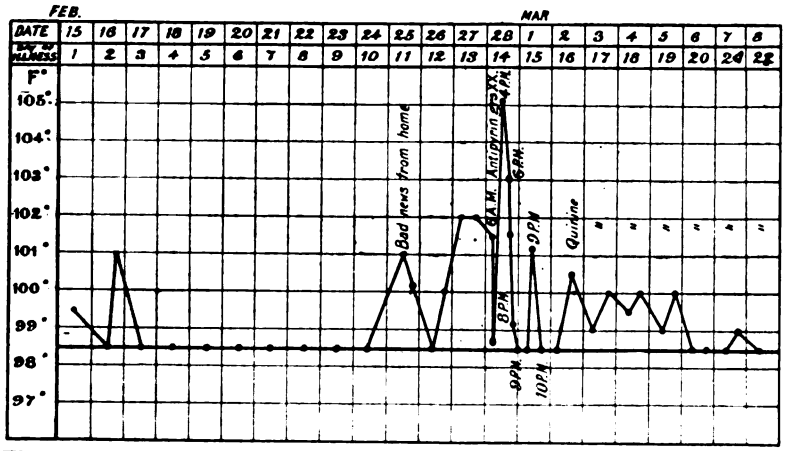
BLACKWATER fever is attracting a great deal of attention just at present, and the following cases may be of interest in showing the clinical features of the malady, as they range from a rapidly fatal attack to a mild, transient one. I have endeavoured in every instance to obtain as much information as possible, and although some of the notes are scanty, I think the course of each case can be readily followed.

Case I.—A. B., a miner, aged 34, a short, well-built man, arrived in this district for the first time in March, 1896, and was invalided home after fifteen months for intermittent fever and mental depression; he stayed in England three months and came back here at the end of September, 1897; had two or three mild attacks of malarial fever, but recovered quickly each time. On the afternoon of March 15, 1898, the patient had a moderately severe rigor, quickly followed by intense frontal headache and general aching pains about the body; during the evening of the same day some very dark blackish-brown urine was passed. I first saw him on March 17, and found him looking pinched and ill; he appeared fairly cheerful, but complained of much headache and great thirst. The temperature in the axilla was 102·4°, pulse rapid, soft, small volume and regular; tongue, slight white fur all over dorsum; lips and mucous membranes pallid; skin and conjunctivæ of a bright yellow colour; examination of the heart, lungs and liver revealed nothing; spleen felt as a soft mass, one finger's breadth below the costal arch, not tender; very little wasting of limbs; urine of a dark brown colour passing in good quantities; bowels constipated. I administered antipyrin gr. x. and ordered a diet of chicken broth and arrowroot, with lime-juice water to relieve thirst. At 9.30 p.m. I gave calomel, gr. v. Early next morning the bowels were well relieved and the urine was of a better colour; as there was no vomiting or digestive disturbance I prescribed a mixture containing tr. cinchonæ comp. ℥ xx., spr. ammon. comp. ℥ x., aq. ad ʒj i., t.d.s. The temperature was normal all day; diet as before. During the next two days rapid improvement took place, the urine became quite clear, the headache and icteric tinge disappeared and the thirst considerably diminished. The temperature did not again rise above normal, the patient quickly gained strength and resumed work in a fortnight, although advised that it was too soon to do so. About a month later he had an attack of sub-acute rheumatism, and as his general condition was unsatisfactory, he was invalided home early in May, 1898.

Case II.—C. D., aged 36 (*circ.*), had been in this district on two previous occasions; was invalided in December, 1897, after a short stay, for intermittent fever, and was extremely ill when he left the Coast; arrived home in cold weather, and was advised by his medical man to remove to a warmer climate, but he sailed for West Africa after a stay in England of a little over two months, and arrived here at the end of March, 1898; almost immediately he had an attack

of malarial fever, although from then he had fairly good health until May, when he had blackwater fever. I will state the case as it was described to me by Dr. Reid, to whom, for the notes of this case, as well as the termination of the former, I am indebted. "C. D.'s case was a typical one of blackwater fever; he was taken suddenly ill in the usual manner with shivering, headache, pains about the body and severe vomiting. The temperature rose a little above 105°, but fell to normal after the patient had sweated freely; the vomit was full of bile and was very persistent and intractable, and blood, both 'coffee-ground' and in streaks, was also present. For about the first two days the urine was quite black, thick, and full of blood and bile; any drop that fell on to his pajamas stained them a dark, brownish-green colour. On the second day icteric discoloration appeared on every inch of his body, quite yellow; in fact, I should say he had almost every symptom of yellow fever, except black vomit. I could do nothing to stop the vomiting for any length of time, and his breath had a strong, sickly smell. On the third day the urine became normal in colour, and about the fourth day there was very little sign of jaundice, except in the conjunctivæ, which were slightly stained. The temperature did not rise again after the first day, except once to 100°, whilst on the boat going down the river. Four days before death most persistent hiccough set in, and would not yield to any form of treatment, although I exhausted all the remedies I could think or read of; the best thing I found was a little hot brandy and water." Death took place on the tenth day of the illness, about seven weeks after the patient had arrived from England.

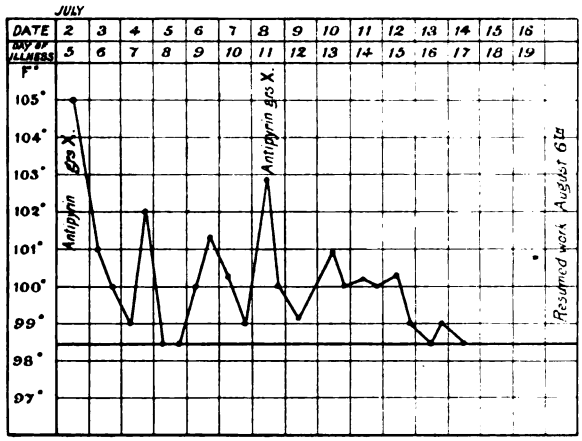
Case III.—E. F., aged 34, engineer, has been on the West Coast nearly nine years, except for short holidays occasionally; has always enjoyed fairly good health, has never been invalided. Arrived in this district November, 1897, and beyond two or three slight attacks of malarial fever, had good health.



The attack of blackwater fever commenced on February 15, 1898, with a slight rigor, the temperature being only one degree above normal; next day a large quantity of dark coloured urine was passed, the temperature rose to 101°, but fell to normal on the following day. There was no discoloration of the skin or conjunctivæ, and no blood in the stools or vomit. On the 19th the urine was clear and did not again become dark coloured. The temperature continued normal for seven days, when the patient had bad news from home, the receipt of which was followed by an intermittent fever, as shown in the chart. The diet throughout the illness consisted of beef-tea, chicken-broth, arrowroot, small quantities of stimulants if required, and milk and soda to relieve thirst. Treatment: at first five-grain doses of quinine were given thrice daily, later on liq. arsenici hydrochlor. ♏ v. was added, but without any apparent beneficial result; five-grain doses of quinine thrice daily were again administered, and the patient made a good recovery, resuming work on March 21, and his health continued good until he resigned his appointment and left the Colony three months later.

Case IV.—G. H., aged 22, Fanti, fitter, was taken ill on June 28, 1898, with shivering and intense frontal headache, two days later diarrhœa and vomiting set in and the patient was troubled with a severe "hacking" cough. I first saw the man on July 2, and his condition was as follows:—Patient looks extremely ill, the face is pinched and sharp, eyes sunken and heavy looking, skin hot and dry to touch; temperature in the axilla 105°, pulse 140, weak, small volume, very irregular; has not slept for the past three nights and is in a restless irritable condition; lies on his left side curled up in bed, avoiding any light, is conscious and answers correctly when spoken to, but wanders a little at times; complains of much headache and thirst; every half hour or less the bowels are opened and a small quantity of dark semi-solid feculent matter, containing bright blood, is passed, almost simultaneously with the passage of a motion vomiting takes place of a thin dark fluid in which red streaks are plentiful, looking like "coffee-ground" vomit with fresh bright blood in it; food and drink are returned almost immediately; the conjunctivæ are of a dirty yellow colour; tongue large, flabby and tremulous, with a thin white fur covering the dorsum; both articulations of the lower jaw are painful and there is some amount of tender external swelling; patient will not separate his teeth more than half an inch; closing the mouth causes no discomfort; the heart sounds are very feeble and irregular, and there is some præcordial pain, varying in character from time to time; small râles are present all over both lungs; the cough is frequent and distressing, patient spits a quantity of thick, white, ropy sputum containing many streaks of dark red blood; the abdomen is concave and tender, especially in the epi- and hypogastric regions, probably largely due to straining of the abdominal muscles by the repeated vomiting; liver not enlarged; spleen can be felt two fingers' breadth below the costal arch; the limbs are much wasted; almost total loss of power in both legs and considerable loss in both arms; knee-jerks absent; complains of a numb feeling in the toes and tips of the fingers; some amount of painful swelling in both elbows and both ankle joints; feet slightly swollen. The urine is passed in quantities of two or three ounces, with

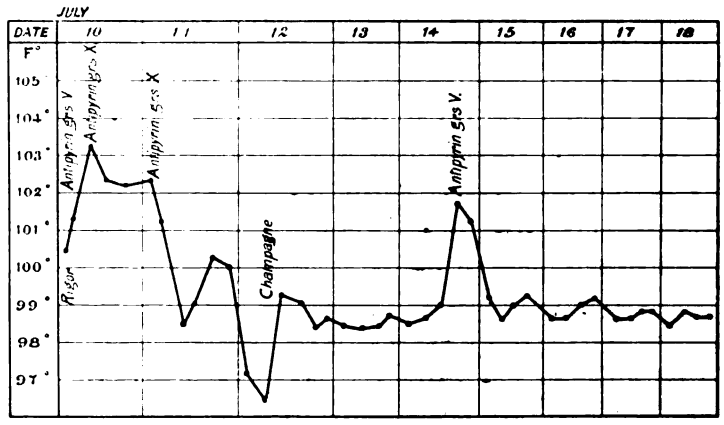
slight pain referred to the bladder; in bulk it is of a dark reddish-brown colour, looked at in a thin layer through glass it appears of a clear sherry colour; on examination the reaction to litmus paper was as nearly neutral as possible; albumen was present to the extent of one fourth of the quantity of urine taken and there was a considerable quantity of blood; no bile could be detected. I first gave the patient a



hypodermic injection of morphinæ sulphat. gr. ¼, and fifteen minutes later antipyrin, gr. x., by the mouth, this was retained. One hour after the antipyrin I prescribed a mixture containing liq. bismuthi et ammon. citrat. ♏ xx., tr. krameriæ ♏ v., acidi hydrocyan. dil. ♏ v., ex. aqua, f. ʒss., at three-hour intervals; the vomiting was completely controlled, the diarrhœa considerably checked and the patient passed a fairly comfortable night with four hours' good sleep. The diet consisted of arrowroot, beef-tea, chicken broth with toast, eggs beaten up in milk, and bread and milk; lime juice in water to drink. Two days after I had first seen the patient I put him on a mixture containing tr. cinchon. comp. ♏ xv., spr. ammon. comp. ♏ x., ex. aqua f. ʒss. thrice daily. During the first eight or nine days of my attendance, the state of the patient's heart caused much anxiety, at times it would be very feeble and irregular and the patient would be in a state bordering on collapse with quick shallow respiration; as the general condition improved, the gradual strengthening and increasing regularity of the heart-beat was very marked. The conjunctivæ began to clear on the 12th day of the illness and the urine was normal on the 15th day; the loss of power in the legs persisted for ten days, although considerable power was gained in the arms before this; knee-jerks were not obtained till the 15th day; the pain in the lower jaw, elbow and ankle joints, did not disappear for nearly three weeks.

Recovery was very slow and there were continual exacerbations of the headache accompanied by insomnia, sleep generally being of a snatchy, restless and unrefreshing character. The patient resumed work on August 6, apparently fully recovered; but on August 23, had to leave work again on account of a gradual loss of power in both legs, which showed itself on August 20; for about a week the patient was unable to walk, the sensation of numbness returned to the toes, the knee-jerks were greatly diminished and there was some loss of power in the arms; the patient, however, felt very well, his appetite was good and he slept well. Power gradually returned to the legs and arms without the aid of treatment beyond rest and warmth; the patient resumed work on September 9, and has kept well since. I take it that the probable primary cause of this man's paralysis was effusion into the theca of the cord causing compression and that the return of the paralysis was due to inhibition of the process of absorption (probably caused by cold and wet) with re-accumulation of fluid, rest and warmth again restoring the normal functions of the cord by favouring re-absorption of the excess fluid. It may be of interest to some to know that throughout this case no alcohol was used except the minute quantities in the drugs.

Case V.—I. J., aged 23, clerk, a stout, pale looking man; had never been abroad before, arrived on the West Coast in October, 1897. For the first four months he had good health, but in January, 1898, he was sent up to this district and had several attacks of malarial fever. Early in June the patient had to go down to the coast, returning up here at the end of the month; on July 1 he informed me that two days previously, whilst on the way up, he had twice passed some "almost black" urine; since then, however, the urine had been "all right," his skin then was of a light saffron colour, but the conjunctivæ were clear; examination of a specimen of his urine revealed nothing amiss. I prescribed a mixture containing acidi nitro hydrochlor. dil. ♏ v. ex. inf. gent. comp. f. ʒs., thrice daily, the tingeing of the skin quickly disappeared and the patient pursued his duties as usual up to



July 9, and on the morning of the next day, except for a slight amount of diarrhœa, he appeared in his usual health; at 1 p.m. on the same day he was seized with a severe rigor, had intense headache, pains about the limbs and a dull heavy aching pain across the loins; he was at once put to bed, five grains of antipyrin was administered, followed by a cupful of hot lime-leaf tea, the patient soon began to feel warm, but did not sweat. At 3 p.m. twenty ounces of dark reddish-black urine were passed, containing blood and a large amount of albumen; an hour later severe vomiting set in, every few minutes retching came on

and nothing could be retained, the diarrhœa had ceased without treatment; great thirst was complained of; this was partially relieved by letting patient swill his mouth out with a weak solution of hydrochloric acid in water (acid hydrochlor. dil. f. ʒi. ex. aqua Oii). At 8 p.m. ten ounces of the same description of urine as before were passed; the vomiting continued, bismuthi subnit. grs. v., acid hydrocyan. dil. ♏ v. ex. aqua f. ʒij. was returned at once; mustard to the epigastrium had no good effect.

July 11.—Patient looks very ill, face and lips blanched, the breath has a smell like that of freshly drawn blood; tongue pale and flabby, edges and tip clean, thin white fur down centre of dorsum; vomiting continues, at first of bright green fluid, later of a dark brownish fluid, probably due to admixture of altered blood, pulse rapid, soft and even; respiration hurried and somewhat jerky in character; heart sounds weak, but clear and regular; lungs, healthy; liver not enlarged or tender; spleen felt as a soft mass just below the costal arch. As the patient could not retain food, I gave him acid hydrocyan. dil. ♏ x. ex. aqua f. ʒi., making him slowly sip a few drops at a time; this materially checked the vomiting. Later on, at intervals of two hours, he took small measured quantities (two ounces) of Benger's food in milk, and Wyett beef juice in milk, at the temp. of the air, 75° F. all of which was retained. Thirst troublesome, and headache very severe; patient is quite unable to sleep, lies on his side in bed with his head thrown back, cannot bear any light and is very restless and irritable. A handkerchief soaked in an evaporating lotion (rectified spirit 3, lavender water 1, water 4 parts) was laid across the forehead and appeared to afford much relief, the patient sleeping for short intervals; phenacetin, caffeine, and a combination of the two quite failed to relieve the headache. Bowels opened once, no pain or straining, motion normal in appearance, no sign of any blood; in the past twenty-four hours thirty ounces of the same urine as before have been passed.

July 12.—Patient looks much worse, appears collapsed and is very weak; the skin is of a bright saffron colour and the conjunctivæ are lemon yellow; this colouration has appeared within less than twelve hours; was restless and sleepy during the night, but only dozed off for a few minutes at a time. Headache is better, much pain present across the loins and over the hepatic region. At 10 a.m. the temp. fell to 96·4° (verified by two thermometers, alternately in each axilla) and the patient seemed to be on the verge of dissolution, lying low in the bed on his back in a dull drowsy state, the pulse was scarcely discernible at the wrist and the respiration was shallow and sighing. Hot water bottles were placed round the patient, a mustard plaster was applied to the præcordium, and four ounces of champagne were given by the mouth with a spoon; in about an hour the patient began to rally and the pulse considerably improved; at intervals of two hours small quantities of Benger's food or beef juice in milk were given, together with one ounce of champagne; everything was retained. In the afternoon as the pain over the liver was getting worse, hot fomentations of mustard water were used for two hours with much benefit, the pain almost entirely disappeared and at 4 p.m. free sweating occurred for the first time since the onset of the illness, having no doubt been induced by the hot fomentations; the patient rallied well towards evening, but vomited a small quantity of food once; thirst and headache not so troublesome; the limbs look shrunken and considerable muscular weakness is present; in the past twenty-four hours 24 ounces of urine have been passed, of a slightly better colour; at 11 p.m., as patient was very restless and tossing about in bed, I hypodermically injected morphinæ sulphat. gr. ¼.

July 13.—Patient had four hours' sleep during the night, very restless towards morning; the colour of the conjunctivæ is less, although that of the skin remains the same. There has been no vomiting, and the thirst is less than yesterday; at noon, passed eight ounces of clear, dark straw-coloured urine, no urine up till then had been passed since 8 p.m. the day before. Pulse is soft and rapid, but decidedly better than yesterday; respiration is still somewhat shallow and hurried. Eggs beaten up in milk added to diet; everything is retained. Thirty ounces of urine passed between noon and 8 p.m.

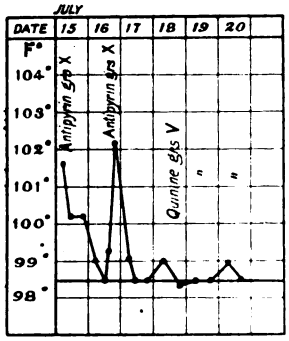
July 14.—The patient passed a restless, sleepless night and appears weaker this morning. The pulse is rapid, soft and regular; the respiration is shallow and uneven. Patient talks in hurried snatches, but articulates distinctly; complains of pain over the liver, which was again alleviated by hot fomentations. Retains everything; thirst again very troublesome, but a little lemon juice, in a tumblerful of slightly sweetened water, slowly sipped, afforded relief; tongue clean and pale over the anterior half, thick white fur over posterior half of dorsum; breath has lost its smell; colour of the skin has begun to fade. During the day the patient has passed a large quantity of dark straw-coloured urine. At 10 p.m. ten grains of Dover's powder were administered.

July 15.—Patient slept for several hours last night and this morning expresses himself as feeling decidedly better; is much more cheerful and less irritable. At 6 a.m. calomel, gr. v., was given, and the bowels were well relieved once in the afternoon; headache is entirely gone, but a slight amount of aching pain over the liver still persists; the colour of the skin and conjunctivæ is much less; thirst is not at all troublesome and food is taken with a relish; pulse is stronger and respiration, although still rapid, is more regular; talking is less laboured; eighty ounces of urine passed in the last twenty-four hours; the colour continues to improve; prescribed pil. Blaud, gr. viii., c. acid. arsenici gr. $\frac{1}{3\frac{1}{2}}$, thrice daily, after meals.

July 16.—Still improving, the urine is now quite clear and the colour has entirely gone from the skin and eyes, leaving the patient looking very pale and anæmic; the spleen can still be felt just below the costal arch; although appearing so thin and wasted, only five pounds have been actually lost, as I had taken the patient's weight the day before he was taken ill; sat out on the verandah and began to take solid food. For the next fortnight the patient slowly but steadily improved, then he had three attacks of remittent fever for which quinine was given in increasing doses without effect, although the attacks invariably yielded to antipyrin. On August 16 the patient went

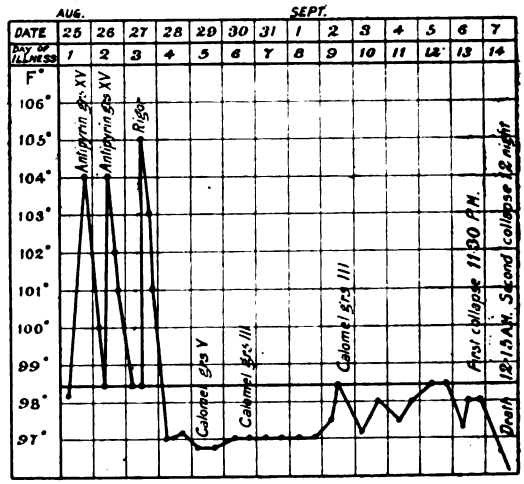
down to the Coast, but again had remittent fever, and was advised to go a sea trip to Sierra Leone and back and has done so.

Case VI.—K. L., aged 33, miner, had been for some time in West Australia, arrived in this district at the end of March, 1897; has not had good health, has had frequent attacks of remittent fever, which always yielded to quinine. For a week previous to onset of present illness, patient had a quotidian fever, the attacks coming on at 7 p.m., with great regularity. Temperature did not rise above 102° at any time. At 4 p.m. on July 15, 1898, whilst sitting in his room, patient had a slight rigor and at once got into bed; one hour later he passed two ounces of prune-juice



coloured urine, containing a quantity of albumen and blood; there was some amount of headache and thirst, but not at all distressing to him; he was already on a light diet, and this was continued; vomiting of some food occurred once; no pain about the body or limbs; the spleen can be felt just at the level of the costal arch; sleeps quietly and well. The next day the urine was clearer and resumed its normal appearance two days later. Five grains of bi-sulphate of quinine were administered once a day for a week; the patient resumed his work on July 25, and appeared to have better health than he had previously had since he came here.

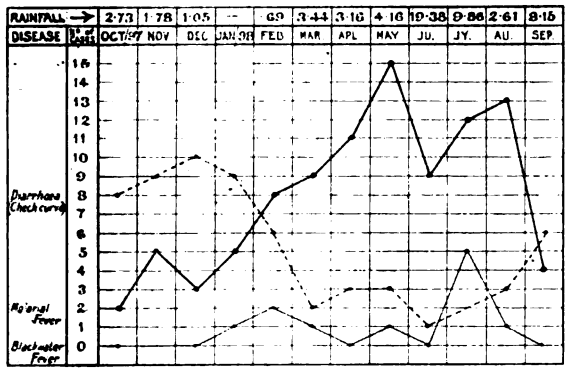
Case VII.—M. N., aged 32, miner, a tall, strong-looking man, had been in this district about nine months, during which period he had two or three moderate attacks of malarial fever, from which he made good recoveries. On Aug. 25, 1898, at mid-day, the patient came up from work stating he did not feel very well; his temperature was normal, but he was advised to turn into bed, and twelve grains of quinine were given to him. In the evening his temperature rose to 104°, and antipyrin, gr. xv., was administered. On the 27th, at 4 p.m., the patient had a severe rigor, the temperature rising to 105°, antipyrin, gr. xv., was given; soon after the rigor some very dark coloured urine was passed; at 9 p.m. antipyrin, gr. xv., was again given; between the rigor and 11.30 p.m., icteric discoloration of the skin appeared, but the conjunctivæ remained clear. Vomiting occurred a few times but was not troublesome, and all food and medicine were retained. No enlargement of the liver or spleen could be detected. Nothing of any note happened for some days; the urine began to clear and was normal on the seventh day and continued so, the icteric tinge had disappeared from the skin by the same day; the bowels were constipated but were relieved by calomel. On the 8th day the patient was allowed to get up and sit out on the verandah, as he was apparently making favourable progress. Two days later, quininæ sulphat. gr. iii., t.d.s., was prescribed, but on the following day, as some amount of vomiting had set in, a mixture containing sodæ bicarb. spr. ammon. comp. and acid. hydrocyan. dil. was ordered and the emesis was controlled. For a short time on the two succeeding days the patient was troubled with hiccough; on the morning of the 13th day he vomited about half a pint of green bile and expressed himself as feeling better afterwards; later on he had some distension of the stomach with eructations of gas. At 11.30 p.m., after he had partaken of some arrowroot,



he got out of bed and sat on the stool although nothing passed; suddenly he showed signs of collapse and was lifted into bed. The pulse could scarcely be felt at the wrist, a hypodermic injection of digitalin, gr. $\frac{1}{100}$, was given, and a teaspooonful of brandy was given by the mouth; the patient rallied extremely well, spoke quite distinctly and said he felt very much better. At midnight he had another severe collapse, and although the same remedies were employed, together with other measures, the patient never rallied, and died at 12.15 a.m. For the notes of this case as well as for those of case iii., I am indebted to Dr. Gardiner.

Blackwater fever, as has been recently pointed out, is widely distributed throughout the world; in many places it is endemic, at times assuming an epidemical character, and is by far the worst type of fever met with on the west coast of Africa, being greatly dreaded by Europeans as well as by natives, the latter being frequently attacked. Whatever relation blackwater fever may have to malaria there can be no doubt that the more malarious a district is the more malignant and fatal is the type of blackwater fever met with there; moreover the curves of the two fevers show a somewhat close resemblance to one another, that is, a rise during the transitional seasons, with a fall in the heavy rains and the dry season. It might be contended that this similarity was the outcome of quinine administered for malarial fever, but I have drawn the curves from my own case book, and I only use

quinine between attacks as a prophylactic, in doses of not more than ten grains in a day, two, or at the most three, days per week. In the causation of blackwater fever the most prominent factors appear to be the same as in malarial fever, viz., residence in malarious districts, especially on or near swampy or low-lying ground, exposure to heavy mists, cold, wet, anxiety, exhaustion from overwork, &c., and perhaps what is most important is the fact that repeated attacks of malarial fever itself often culminate in an attack of blackwater fever; whilst again, each attack of blackwater fever predisposes to another, and the intervals between the attacks tend to become shorter or longer according to the treatment and after care in each instance, much as is the case in paroxysmal hæmoglobinuria; but the immediate exciting cause of the melanuria is still unknown. Despite the failure up to the present to find a specific organism, one is not yet justified in asserting that such does not exist, for taking the analogous instance of yellow fever, it is only recently that Sanarelli discovered and demonstrated the bacillus icteroides, although the latter disease was far better known, and had far more attention given to it than has blackwater fever, so that one is encouraged to think that it is possible and certainly probable that at no distant date either some micro-organism peculiar to blackwater fever will be found, or that its direct relationship with malaria will be definitely established. It may be that blackwater fever is what might be termed a malignant, pernicious or vicious manifestation of malarial fever itself, just as one gets mild and malignant types of variola or diphtheria. It is possible to consider that the amount of malarial poison present in a given case may be so great, the proliferation of an already large number of malarial parasites so rapid, that the slightly increased hæmolysis usually occurring in an ordinary fever may be intensified to a degree that can only be called malignant; the products of this greatly increased hæmolysis are excreted by the kidneys and give rise to the discolouration of the urine typical of the disease, so that blackwater fever could be described as malarial fever plus melanuria, and clinically this is really what it seems to be. The irregular fever corresponding to none of the known types of malaria could be accounted for thus:—the initial fever is caused by the maturation of the parasites (as in an ordinary fever), then, instead of subsiding, is maintained by irritation of the thermal centre due to the profoundly altered state of the blood supplied to it (just as a rabbit's temperature can be raised artificially by chemical irritation of its thermal centre); this is borne out by the fact that the duration of fever varies directly as to the melanuria, that is to say, as soon as the deleterious materials or products of the excessive hæmolysis were eliminated from the blood, irritation of the thermal centre would cease and the temperature would begin to fall. The symptoms and onset are much the same as in an ordinary attack of malaria, shivering, headache, pains about the body, especially across the loins, with a feeling of general malaise and restlessness, but it is not until the typical dark-coloured urine is passed that there is any definite sign by which one can diagnose the case as blackwater fever. The rigor can almost always be taken as an index to the severity of the melanuria; in every case I have seen, whenever the rigor has been severe the disease has assumed a grave aspect, and on the other hand, when the rigor was slight the case has invariably been of a mild type.



In a severe case the pulse soon becomes rapid, soft, of small volume and easily compressible, it is often markedly dichrotic, and frequently intermissions are noted. In case iv., the intermissions at one period were one in three; respiration is quick and shallow; later on it assumes a soft sighing character, whilst at other times it bears a modified resemblance to "Cheyne-Stokes" breathing; the lungs are not often affected, but bronchitis sometimes occurs, usually in bad cases; the impulse of the heart at the apex is weak, fluttering and often very irregular both in rhythm and force; the sounds are muffled and distant although distinct, reminding one of the cardiac condition usually found in a case of typhoid fever. I have never detected any dilatation of the heart; vomiting is common, is generally persistent and exhausting and often is extremely difficult to control; the urine is passed in small quantities, as a rule without pain, still I have heard a patient complain of scalding and another of pain in the bladder during micturition; the characteristics of the urine can best be understood by referring to cases iv., v. and vi.; the bowels are usually constipated, but not infrequently diarrhœa is present; headache, sometimes frontal, at other times vertical or occipital, is one of the most distressing features of the illness, it is very difficult to alleviate, and by preventing sleep exhausts the patient considerably; pain over the liver is sometimes complained of, but the organ is seldom appreciably enlarged; the spleen can always easily be felt extending down to or beyond the edge of the costal arch. About the second or third day in a severe or moderately severe case, icteric tingeing of the skin and conjunctivæ appears; it begins to fade soon after the urine shows signs of clearing and is quite gone about the first or second day after clear urine is again passed. Considerable wasting of the body appears to take place, as well as great loss of power in the limbs, amounting in case iv. to actual paralysis, and the patient for the first three or four days loses ground with alarming rapidity. Considering that the Europeans here are picked and physically sound men, it is very noticeable how a strong, powerful man in a few days is reduced to an

almost helpless condition of weakness with an appearance of having been ill for several weeks at least. Blackwater fever would have to be diagnosed chiefly from yellow fever, bilious remittent fever, paroxysmal hæmoglobinuria, or jaundice with fever; from the first two it could be at once differentiated by its dark urine, this again would have to be distinguished from the urines of the latter two diseases as well as that of acute nephritis; in acute nephritis the history of the case, the state of the pulse, the swelling of the face and feet and the brighter colour of the urine would serve as guides to the nature of the malady; from paroxysmal hæmoglobinuria there are many features of demarcation; the urine here, in bulk, is of a clear reddish colour and not of a dark opaque reddish black or brown as in blackwater fever, moreover paroxysmal hæmoglobinuria is usually very transient, fever is often absent, as are many of the symptoms so prevalent in blackwater fever; the urine with which that of blackwater fever is most likely to be confused is the dark porter-like urine of catarrhal jaundice, but here again a specimen of each in a thin layer shows marked differences of colour, whilst the absence of hæmoglobin and the small quantity (if any) of albumen in the urine of jaundice is in great contrast to the large amount of each present in that of blackwater fever. The prognosis must depend entirely on the type the attack assumes; mild cases almost always recover, severe cases sometimes recover but often end fatally, and it would be wise in every instance to give a guarded prognosis, especially if there is much melanuria or high fever; the prognosis must be considered bad in cases where the urine is scanty or suppressed (one case I know of terminated fatally on the third day through suppression of urine, the patient was unconscious for ten hours before death, but there were no convulsions) in which the health of the patient has been reduced by previous illness or continued alcoholism, or where there is rapid loss of strength, much delirium, excessive and intractable vomiting, severe hiccough, or marked cardiac failure. The effect of blackwater fever on the heart is very grave, and after the kidneys a fatal result is most to be feared from cardiac failure as in case vii.; in this instance nearly three months before the outset of the fatal illness, during an attack of malarial fever, this patient's heart was on several occasions irregular in time and force without apparent cause (beyond the malarial attack itself) although he did not at any time complain of cardiac pain or discomfort.

In addition to the seven cases of which I have here given notes, nine other cases in Europeans have come under my notice, six of which ended fatally, and although sixteen cases are far too few on which to base statistics, a mortality of 50 per cent. is shown counting all cases, or of 53·3 per cent. amongst Europeans only, and I think taking all types of cases this must be considered a fair average death rate. The treatment in our present state of knowledge must be entirely directed to combating symptoms and supporting a patient's powers; briefly—rest, warmth and light unstimulating diet. Attention must be paid to the daily quantity of urine; if this is small the skin and bowels should be made to act as freely as possible to relieve the strain on the kidneys. To induce sweating, hypodermic injections of pilocarpine nitrate gr. $\frac{1}{3}$, once or twice a day, are most useful and calomel in five-grain doses may be given to relieve the bowels. To reduce the temperature, antipyrin, in doses varying from gr. v. to gr. xv., is far in advance of any other drug; it acts quickly, satisfactorily and has no disturbing influence on the head or stomach; should it appear to depress the patient—and I have never seen it do so in these doses—five or ten minims of spr. ammon. comp. may be given at the same time; next to antipyrin I prefer salicylate of soda with ammonia; phenacetin and antifebrin are not nearly so efficacious, and quinine is best avoided during the period of pyrexia, as it often appears to increase any tendency there is to delirium. For the extreme restlessness and insomnia so prevalent in the disease I have used hypodermic injections of morphine sulphate, gr. $\frac{1}{4}$, or Dover's powder, gr. x., with satisfactory results, and provided the condition of the kidneys warranted the use of these drugs, they are of the greatest benefit to the patient by inducing sleep and thus conserving his strength. The heart must be carefully and constantly watched and any sign of flagging at once noted and steps taken to overcome it; for this purpose spr. ammon. comp. ♏ v.—♏ xx. in water is best, or if there has been much vomiting a small quantity of champagne may be slowly sipped; ammonia is, I think, to be prefered to alcohol on account of any baneful influence the latter may have on the kidneys, in fact I think the less alcohol used in these cases the better, although at times it is of the greatest benefit. Convalescence is very slow and great after-care is necessary, especially if the case has been at all severe; the resulting anæmia is best treated with arsenic, and I have given liq. arsenicalis, f. ʒi., thrice daily with good results. Whatever the nature of the attack has been it is always advisable for the patient to have a change of air for at least a month, and the best way is to send him a sea voyage if it is at all possible.

Little can be said of the pathology of blackwater fever until its definite and true cause is known, but there can be no doubt that some profound alteration in the character of the blood takes place, causing the destruction of a very large number of red corpuscles, accompanied by sanguineous exudation from the smaller blood vessels as well as by some amount of capillary hæmorrhages, especially from mucous membranes, and producing intense but not necessarily persistent anæmia. It is probable that an organism develops in a red blood cell (as in ordinary malaria), that every cell so attacked is broken up and its constituents diffused throughout the blood plasma. The next step would be that whilst the liver picked out and stored up some of the hæmatin for future use, it would fall to the kidney to excrete the deleterious matter in the plasma, and the length of time required to effect this completely would depend, firstly: on the quantity of effete material present, *i.e.*, depends on the degree of hæmolysis, or secondly, on the condition of the kidney itself. Omitting for the present the second factor, we may say that the duration and severity of melanuria is in direct ratio to the number of red blood corpuscles in which organisms have developed; that is, blackwater fever varies in type as

to the number of parasites present in a given attack. The existence of chronic interstitial, or any trace of parenchymatous nephritis, would be serious considerations in the illness, for with the additional irritation consequent on the passage of a large amount of broken-down blood, an acute nephritis may be set up, perhaps with total suppression of urine, and thus a great probability of a fatal termination to the case. It is possible to consider that the red corpuscles are infected with the parasites during their passage through the spleen; in those cases of blackwater fever occurring after repeated attacks of malarial fever, it may be that a certain number of organisms become lodged in the spleen in a latent state, that ultimately a point of saturation, so to speak, is reached, then owing to some disturbing influence, say a fresh attack of malaria, the potential energy of the collected parasites is suddenly converted into a condition of great activity, and as I mentioned earlier, an excessive degree of the hæmolysis usually occurring in malarial fever is the result. Melanuria ceases spontaneously and is not at all dependent upon treatment (beyond rest and warmth); this would lead one to believe that all the organisms in the infected cells matured together, that the actual period concerned in the hæmolytic process was a short one, and that as soon as the detrimental substances in the blood had been excreted by the kidneys the attack was over. This is supported also by the icteric tingeing of the skin and conjunctivæ that occurs in some cases, and which is not primarily of hepatic origin at all; a large and sudden increase of a normal process is thrown on the liver cells, and these, unable to meet the increased demand on their functions, allow a certain amount of unattached hæmachrome to pass into the general circulation, probably also with the addition of a small quantity of bile, and give rise to the staining of the skin and eyes. Perhaps this discoloration is due in some measure to the decreased working capacity of the liver and kidneys consequent on their imperfect and diminished blood supply. It is very noticeable how exact is the ratio between the amount and duration of the icteric colouring and the amount and duration of the melanuria; in slight cases with transient melanuria, yellow tingeing of the skin may be quite absent, whilst in severe cases with much melanuria the discoloration of the skin and eyes is very marked. In common with the liver and kidneys, every organ as well as the muscular portion of the body suffers through the defective blood supply; the weak irregular action of the heart is due partly to a badly nourished myocardium and partly to irregular and improperly controlled innervation owing to the anæmic condition of the brain and spinal cord, which also explains the loss of power taking place in the limbs. The presence of blood in the vomit, sputum and fæces, can be regarded as the result of capillary hæmorrhages due to the altered condition of the blood, or it might even be the result of the acute development of anæmia. I regret that I am unable to supply any index of the degree of the anæmia in any of the cases, but in cases iv. and v. it was severe.

With reference to the influence of quinine as a factor in the causation of blackwater fever, although it is known that this drug has a certain more or less well-defined deleterious action on the red blood corpuscles, it is difficult to believe that the disease can ever be wholly due to the toxic action of quinine, especially when used in the moderate and careful manner of British practitioners as a rule; I do not know the German method of administration, but a friend living in French West African territory recently wrote to me that "quinine is piled into you on the slightest pretext by the French doctors, in huge doses, and I dread getting a fever because of taking so much quinine," and it is probably when used in this reckless, meaningless manner that any toxic effect of the drug would be observed. Melanuria occurring after the exhibition of quinine, could, I think, be rightly regarded as an idiosyncrasy on the part of the patient, otherwise after long continued use of the drug one would almost invariably expect the patient to develop hæmoglobinuria, and moreover, that the severity of the attack would bear a direct ratio to the amount of quinine ingested; but this is certainly not the case, for one frequently sees cases that can only be characterised as having a severity in inverse ratio to the amount of quinine taken. Whether excessive or at least immoderate indulgence in alcohol has any effect in helping to establish a "quinine habit," if such can or does exist, I do not know, nor whether it does in any way modify the action of quinine, but it would be expected that by reason of the action of alcohol itself on red blood corpuscles, any hæmolytic influence quinine might possess would be considerably increased. Granting that a drug has different results when administered in the same dose to different people, and also different results when administered in different doses to the same people, and presupposing that quinine, if given in sufficient amount for a sufficient length of time will cause hæmoglobinuria, it would not have been unreasonable to have expected such a result in the following instance, unless, as I have said, a "quinine habit" could have been established:—

O. P., aged 27, had been in Burma and South America, engaged here in road-making; brought out a large quantity of quinine pills, each pill containing six grains of (soluble) bi-sulphate of quinine and dissolving in water in about three minutes; his method was to take six of these pills each morning and evening, *i.e.*, seventy-two grains of quinine in a soluble state, daily, and occasionally he would take three or four of these pills at mid-day as well; this unique dosage was persisted in for nearly six months, when having exhausted his supply, I eventually persuaded him to moderate his daily quantity, but could not make him content himself with less than a daily dose varying from four to six five-grain tabloids, and this went on for another six months, until he left the colony. During his stay of just over twelve months he only had one slight attack of malarial fever, although three or four times he had what might be termed abortive attacks, lasting about one hour or less. Now on the other hand, in cases iii. and v., the patients were very lax about taking quinine, even when ordered to do so, whilst in case iv., to my own knowledge quinine had not been taken for the previous six months (amongst natives generally, this drug is never taken), so that in this case at least, quinine as a causative agent can be absolutely eliminated. Again,

cases iii. and vi. recovered perfectly well under the administration of moderate doses of quinine, but one would think that if the excessive hæmolysis occurring in blackwater fever was in any way attributable to quinine, further dosage with the drug would induce a prolongation of the period of hæmolysis and hence of the melanuria, yet it undoubtedly does not do so; then if quinine, having once caused melanuria, could be said to have a protective influence, so that further dosage with it induced no increase in time or quantity of the melanuria, then the continuance of quinine administration would be quite useless; but it is not so; moreover, succeeding attacks of blackwater fever should be slighter than the preceding ones, but the tendency is rather for each attack to become more virulent.

The failure of quinine to do good in many cases of blackwater fever may be analogous to that of iron in some forms of anæmia, where, although the latter has no beneficial effect, arsenic is of the utmost value; so also it may be that although quinine is useful in ordinary malarial fevers, yet in that particular manifestation of malaria, as I think blackwater fever can be termed, it is as an antipyretic, almost, if not quite useless, whereas other drugs (antipyrin, sodii salicylatis) act quickly and effectually. I think the great drawback to the administration of quinine in severe cases of blackwater fever is due to its probable influence on respiration. This drug, as is known, binds the hæmatin of the red blood corpuscle more firmly to the globulin and retards oxygenation; thus in the anæmia of blackwater fever to enable the red corpuscles, with their lessened number and function (due to the condition of the plasma), to imbibe sufficient oxygen to sustain life, respiration would be quickened, and there would be an increase in the number of heart beats. Together this would tend to further exhaust a patient's strength, and where the utmost conservation of a patient's powers is so essentially necessary, any drug calling for, or having a tendency to produce, greater expenditure of energy on the part of the heart especially, would be strongly contra-indicated. Other reasons, though of less importance against quinine administration, are the headache, tinnitus aurium, and digestive disturbance it causes, or aggravates, if previously existing; and where, as in blackwater fever, headache and vomiting are such frequent and distressing concomitants, in giving antipyretics a course has to be steered and a drug chosen that will allay, and not irritate, the already disturbed condition of the head and stomach in particular. Moreover, quinine, compared with antipyrin, acts very slowly, and the quicker a high temperature can be made to fall in a reasonable time, the sooner does the patient begin to feel comfortable and the better are his chances of natural sleep—all important considerations. It is possible that a dose of quinine given for an attack of apparently ordinary fever, may be followed by melanuria, which would perhaps be credited to the quinine, still it would be safer and perhaps more correct also to regard the previous administration of quinine as an accidental coincidence with, and not as a cause of, the onset of melanuria; this is brought before me in case v. Here at the onset of the rigor, following my usual custom, I gave (five grains of) antipyrin, and I have thought that had I given quinine instead at the time, I might have had to consider the subsequent melanuria as perhaps due to the quinine, whereas now I know it was not so. Amongst Europeans it is never difficult to find evidence of the more or less recent employment of quinine, but it is far harder to do so amongst natives, and as blackwater fever is clinically identical in the two races, one would expect to find some causative factor applicable to every case, as the disease is not contagious, and is not communicated from a European to a native, *vice versa*, or from one to another of the same race. One is therefore led to the conclusion that malaria is in all probability the sole cause, in fact, that blackwater fever is but a severe and pernicious malarial fever. The following description of the disease as it occurs amongst the natives of Ashanti, was communicated to me by a very intelligent, English-speaking native of that country, and I find it substantiated by many Fantis, amongst whom the course and treatment of the malady is practically the same as in Ashanti. Blackwater fever is prevalent at the break up of the rainy season (*i.e.*, August to September); "when the rains come everyone is healthy and well, but when the rains begin to go and the hot weather to come, then plenty of people get this sickness very badly." It comes on with shivering and pains all about the body, the skin is hot and dry; there is often much vomiting, sometimes of bright green fluid, at other times of food or drink taken, and sometimes of blood; the amount of urine is greatly diminished, and sometimes "none at all can be passed." This was accounted a very bad sign, and the patient always died when it occurred; the urine is of a black colour, and sometimes red blood could be seen in it; the bowels are costive, there is great thirst, but a disinclination for food of any kind; "the sick man gets small and shrunken, his arms and legs shrivel up, he is very weak and cannot even stand." The acute illness usually lasts about a week, and recovery is very slow; and whilst in favourable cases the colour of the urine began to improve in four or five days, if melanuria lasted more than a week the man often died. The treatment is worth notice:—When the fever is at its height, the body is washed all over seven or eight times a day with a cold infusion of pawpaw, casada, and lime leaves; the first washing is always at daybreak; "this makes the man's body cool, and the heat of the skin comes off like white smoke"; the patient, after each washing, is rubbed all over with cut limes, some of the juice being squeezed into his mouth, so that he might swallow it; a daily injection is given *per rectum* (through a calabash native enema) of a decoction made by mixing pawpaw root and green peppers ground together, with palm oil; this, in addition to causing the bowels to act freely, is said to considerably increase the flow of urine, this latter effect being due to the pawpaw root. An infusion is made of a species of pale cinchona bark which grows here, by powdering the bark very finely and rubbing it up thoroughly in cold water; about three ounces of this is given to the patient to drink five or six times a day, and three times a day half a pint or so is thrown up the rectum through the calabash enema. This treatment is accounted of great value in the illness, and

reduced in plain terms to cold sponging, purging, and the administration of infusion of cinchona bark, is as rational as one could wish, even if the methods are crude. I was unable to gain any definite statements as to the mortality, as it varied a great deal, sometimes being high, sometimes low, but all agreed that it was the most fatal sickness they were acquainted with.

THE PATHOLOGY AND DIAGNOSIS OF PTERYGIUM.

By M. T. Yarr, F.R.C.S.I.

Major R.A.M.C.; Chief Clinical Assistant Royal Ophthalmic Hospital, Moorfields; Member Ophthalmological Society of the United Kingdom, &c.

Most practitioners in tropical countries are familiar with that peculiar affection of the conjunctiva, or, more correctly speaking, of the cornea, which has been somewhat fancifully called pterygium (πτερύγιον = a little wing): nevertheless some confusion still prevails as to its exact nature, and many confound it with the so-called "false" pterygium or "pterygoid"—a condition to which it bears a superficial resemblance. The ætiology, pathology, and treatment of the two affections are quite different, so that it is of considerable practical importance to be able to draw a sharp line of demarcation between them.

A pterygium affects only the inter-palpebral surface of the eye and is a triangular fold of conjunctiva, the apex of which extends a variable distance into the cornea, while the sides widen out into the bulbar conjunctiva without any very distinct limiting line. In a progressive case this fold of conjunctiva has a reddish colour due to numerous vessels converging from base to apex. The apex or "head" of the pterygium is well-marked and inserted on and into the cornea like a tendon; to this succeeds a contracted portion called the "neck," the edges of which are rounded and incurved so as to permit the passage of a fine probe a short distance (never entirely) underneath them: the remaining fan-shaped portion is known as the "body." The most usual situation is the nasal side of the cornea: I have never seen a *single* temporal pterygium—when one exists in this situation it is always consecutive to one on the nasal side of the same eye; such double pterygia are by no means uncommon. Both eyes are frequently affected, and I remember one case in which four pterygia co-existed, one on the inner and one on the outer side of either cornea. A true pterygium is due to the slow transformation of a pinguecula; so-called pterygia at the upper and lower corneal margins belong to the class of "false" pterygia above alluded to.

Not only is pterygium a deformity, but as it progresses it produces more serious inconveniences, such as impaired motility of the eye, epiphora, and defect of vision. The latter may be due to (1) dragging on the cornea, producing irregular astigmatism; (2) impaired motility with resulting diplopia, or (3) invasion of the pupillary area by the head of the pterygium.

The extent of a pterygium depends on its duration. Commencing at the edge of the cornea, it slowly pushes on, dragging the hypertrophied conjunctiva after it, until it reaches the centre (progressive pterygium); finally, it appears to become stationary and then slowly atrophies to a thin membrane (retrogressive pterygium). A noticeable feature, and one of the many distinctions between it and "pterygoid," is the fact that its progress is unaccompanied by any signs of inflammation.

The disease is comparatively rare in England and in temperate climates generally. In the course of over three years' work at Moorfields I only remember one case, in a patient of Mr. Marcus Gunn's. On the other hand, it is very common in tropical and subtropical countries; I saw a large number in Hong Kong, chiefly amongst Indian residents, but a few also amongst Chinese.

Pathology.

The older writers on pterygium described it as a neoplasm requiring total extirpation. Scarpa first pointed out the erroneous nature of this view; he considered it the result of a peculiar chronic conjunctivitis which he called "varicose conjunctivitis." Middlemoore,[1] writing in 1835, believed the morbid process commenced in the episclera; a belief shared by Rogneta[2] and Manhard[3] many years later. This theory of the episcleral origin of pterygium is still held by a few. Quite recently E. Boeckmann[4] maintained that pterygium is a "traumatic infective episcleritis due to an infected foreign body which has penetrated the sub-conjunctival tissue and which is expelled at the apex of the pterygium"—the foreign body being chased from the conjunctiva into the cornea, so to speak. Unfortunately this ingenious theory is not supported by any evidence, nor is it in accordance with what we know of the behaviour of infected foreign bodies.

Horner[5] was the first to promulgate the now generally accepted view, viz., that pinguecula and pterygium are stages of the same process.

Alt[6] emphasised the anatomical fact that pterygium is really a corneal disease; it is only in the head that peculiar morbid changes are found; the body is merely hypertrophied and hyperæmic conjunctiva. In a large number of microscopical observations he found well-marked and peculiar changes in the cornea at the head, where Bowman's membrane was broken, rolled on itself, and buried in epithelial cells forming a mass between the conjunctival and deeper layers of the cornea. He attributed pterygium to a marginal corneal ulcer, though unable to find any evidence of this in his preparations.

Goldzieber's observations[7] are also of much interest. In addition to the changes described by Alt he notes that the corneal stroma beneath the head is partially replaced by a new layer distinguished from the cornea proper by staining differently with carmine, its thicker fibres, and the presence of sac-like prolongations of epithelium. He also adopts the hypothesis of the corneal ulcer origin of pterygium, though unable to find any proof.

Finally Fuchs[8] made an elaborate study of the subject, based on observations of several hundred pterygia in the living and a large number of necropsies,

which clearly establishes the true nature of the disease, viz., that "it originates from the pinguecula, the degenerative process which exists there making its way into the limbus and then gradully on the cornea itself."[9] Hence the lateral position of genuine pterygium; hence also its occurrence in the middle-aged and elderly, especially in those much exposed to sun, wind, dust, &c. In none of the large number of cases examined did he find the slightest trace of corneal ulceration. The following are the main points brought to light by this distinguished observer's clinical and microscopical researches. Careful observation shows that the head of the pterygium is always preceded and surrounded by a narrow, sharply-defined rim of opaque cornea; beyond this, in the transparent cornea, are little pearly mottlings and occasionally yellow spots resembling those of pinguecula. On microscopic examination the epithelial layer of the pterygium is found to differ markedly in the conjunctival and corneal portions; merely thickened in the former, in the latter it sends down processes into the corneal stroma with here and there invaginations recalling the structure of sudoriparous glands. These observations are confirmed by Panas, who gives an excellent *résumé* of the subject, supplemented by many new facts, in his *Traité des Maladies des Yeux* (Paris, 1894).

One is at once struck by a certain amount of resemblance between the morbid processes of pterygium and epithelioma. In a note in the September number of the JOURNAL, I drew attention to an interesting paper by Dr. X. da Costa, of Lisbon, in which he maintains the possibility of the "epitheliomatous transformation" of the head of a pterygium, and describes two cases in which histological examination showed a distinct epitheliomatous structure. Three similar cases have been recorded by other observers.

Treatment.

The head, neck and a wedge-shaped portion of the body, should be excised, and the sooner the better—if possible before the pupillary area is invaded. The little operation I was in the habit of performing in China resembles that advocated by Fuchs[10] and answered very well; it consisted in dissecting off the head, neck and a triangular segment of the body, and then bringing the edges of the conjunctival wound together with a couple of sutures; for this last procedure liberating incisions are often necessary. I have seldom seen recurrence after this operation and believe the rare cases were due to want of care on my part in closing effectively the gap in the conjunctiva. Panas recommends touching the corneal wound with a thermo-cautery, but I cannot see any advantage in this proceeding; it increases the subsequent nebula and I do not think lessens the risk of recurrence.

Starkey, of Chicago, strongly recommends the treatment of small pterygia, not encroaching far on the cornea, by a weak galvanic current. Details of this method, which seem worth a trial in appropriate cases, will be found at p. 53 of the September number of the JOURNAL.

Boeckmann[11] operates by removal of the head alone, with "reposition and fixation" of the body, which he attaches to the rectus internus or externus, to prevent its sliding over the cornea again. I have never seen this method, and the details in the published descriptions are somewhat difficult to follow.

Pterygoid.

As a result of acute blennorrhœa with marginal corneal ulcer, injuries of cornea by burns, caustics, &c., a fold of chemosed conjunctiva may become adherent to the cornea. This "pterygoid," or false pterygium, presents some resemblance to pterygium, from which, however, it can easily be diagnosed by the history, the inflammation, the staining of the ulcer by fluorescin (if the case be a recent one), the arrest of development on the healing of the ulcer, the absence of the characteristic rim of opaque cornea round the "head," and the variable situation. Often, too, a fine probe can be passed completely under the "neck," showing that the apex of the fold alone is adherent (Fuchs).

Where operative interference with a pterygoid is necessary it usually suffices to "pick" the head out of the cornea; the fold will then retract and become atrophied.

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A NOTE ON ULCERATING GRANULOMA OF THE PUDENDA.

By DR. PATRICK MANSON.

SINCE its first description in the *British Guiana Medical Annual* of 1896, by Drs. Neal, Ozzard, Conyers, and Daniels, this disease has been recognised in India by Maitland (*Ind. Med. Gaz.*, May, 1898), and by Williams (*Ind. Med. Gaz.*, November, 1898). Under date November 1, 1898, Dr. F. Goldsmith, of Palmerston, Northern Territory of South Australia, writes me as follows:—"As regards the disease you term ulcerating granuloma of the pudenda, I had two cases in the hospital of bibras (aboriginal women), aged about 32, suffering from a granulous ulceration of the vulva, very similar to fig. 45, p. 440, in your 'Manual of Tropical Diseases.' I regarded them as probably syphilitic, but was surprised to find that they did not yield to a long course of mercurial and iodide of potassium treatment. After about four months' ineffectual constitutional treatment I left it off and treated the disease entirely locally, with the result that in a month or two I was able to discharge the women comparatively cured. Your work did not arrive in time to try the treatment you recommend;

but with absolute cleanliness, keeping opposing surfaces apart and as dry as possible, and by a free use of a powder containing 1 calomel, 1 boric acid, and 8 powdered oxide of zinc, they healed up, and I have not seen them since (three months ago). One case had the granulomatous condition of the vulva pure and simple; the other had in addition extensive, deep-seated ulcerations in the groin. The excoriations were deep with hard raised edges, and healed comparatively quickly, while the granular vulva took, as I said, months to heal.

"With regard to the causation and history. They both came from a place about a hundred miles from here. About a month before they appeared I received a letter from the same locality telling me that the writer's black boy had an ulceration on his penis, which he thought was cancer, and asking for advice. I gave it and never heard anything more for ten months till two days ago, when I saw my correspondent, who told me that the ulcer was still present and had not commenced to heal. On enquiry I found that the 'boy' used to have connection with these bibras.

"I was consulted yesterday by a white man with an ulceration on the under surface of the glans penis with corresponding ulceration on the contiguous portion of the prepuce. He informed me that he had had connection with a bibra months ago, and that the sore had remained unchanged for the last two months. His sore was neither soft nor hard chancre. It was raised above the surrounding healthy part and had an abundant thin discharge from a granular surface.

"I have now come to the conclusion: (1) that all four cases are granuloma of the pudenda; (2) that they can be transmitted from one person to another by intercourse; (3) that it is not confined to natives; (4) that it is not amenable to antisyphilitic treatment; (5) that it is present in the tropical parts of Australia."

It is interesting to note that my correspondent agrees with Maitland in regarding this disease as venereal but not syphilitic.

Paper read at the Annual Meeting of the British Medical Association.

THE UNCLASSIFIED FEVERS OF HOT CLIMATES.

By A. Crombie, M.D.
Brigade Surgeon, Lieut.-Col. I.M.S.

Specific Fevers.—These are divisible into malarial, non-malarial and mixed. I do not mean to bring the great subject of malarial fevers into this discussion, as their position is now well defined, nor among the non-malarial continued fevers need we spend time over those which are equally well defined, such as relapsing fever, yellow fever, typhus fever, cerebro-spinal fever, and Beri-Beri, all of which are met with in tropical and sub-tropical countries (typhus very rarely, though its occasional presence in up-country jails in India, and among other large bodies of men, seems to be established).

(1) Aphthous fever is undoubtedly communicable to man, and especially to children, by means of the milk of cows affected by foot-and-mouth disease. In man the symptoms are fever, digestive troubles, and vesicular eruptions on the lips, and buccal and pharyngeal mucous membranes. Every one who has practised in the tropics must be able to recall cases referrible to this cause, where there are no laws capable of dealing with the carelessness and cupidity of native dairy-men.

(2) "*Milk Sickness*" is the name given to a disease which prevails in certain districts of Amercia, west of the Alleghany Mountains, and is connected with a disease in cattle called the *trembles* (Osler). The symptoms in man are, after a few days of general *malaise*, pain in the stomach, nausea, vomiting, fever and intense thirst, and usually obstinate constipation, the tongue is swollen and tremulous, and the breath foul with a characteristic odour. Cerebral symptoms are well marked, with irritability, coma, or convulsions, or the patient passes into a typhoid state. The disease may be fatal in a few days or it may extend over a few weeks. It is communicable by the meat as well as the milk and butter derived from the affected animals, as has been experimentally demonstrated in dogs (Osler).

I cannot say positively that the disease has been seen in India; but among the many strange and unusual cases of fever which one occasionally meets with in that country and which refuse to conform to any recognised type, one could not deny that cases of a kind exactly corresponding with this description do sometimes occur. Every one must be able to recall cases that have baffled his diagnosed powers, cases of sudden and pernicious type proving fatal in a few days, or running a malignant course to an ultimately fatal termination. In a country where cattle live under the most insanitary conditions in dark crowded byres, and where hardly anything is known of their epidemic diseases, any infection of this kind is possible, and I feel convinced that inquiry in this direction would be a most fruitful one for a man properly equipped for the task. I have included this "milk sickness" in my list in the hope that it may direct attention to possibilities of this kind in other countries besides America, and because the recollection of such a possibility may lead sometimes to a discovery of the existence of such epizootic causes of disease in India.

(3) I come now to very debatable ground, and I feel that it is round the next five headings on my list that the discussion will centre, and I beg that it will be kept in view that all I am contending for is the recognition of certain clinical types, and that the list I lay before the meeting is only a provisional one to serve as pegs on which those who may take part in the discussion may hang their remarks. That they are clinical types I am prepared to maintain; that they are all separate pathological entities is perhaps a matter on which none of us, in the present state of our knowledge, is in a position to dogmatise.

Urban Continued Fever.—There is a type of continued fever which is prevalent in Calcutta, where it is called "Calcutta Fever," and in Bombay, where it is called "Bombay Fever," and I believe in other

large tropical towns, but is rarely or never met with in the Mofussil. For the reason of its occuring in large towns, and its comparative rarity or absence in rural districts, I have ventured in this paper to call it "Urban Continued Fever," a not inconvenient name, descriptive of one of its peculiarities and more comprehensive than the local names it has hitherto received. It might be called "Bastard Typhoid." It is a continued fever of three or four weeks' duration running the course and describing the temperature curve of a mild typhoid, but without any of the symptoms diagnostic of that disease. There is no rash, no diarrhœa, no gurgling in the right iliac fossa, there is little or no delirium, and it is rarely fatal. The access and defervescence are both slow and gradual, though, as in typhoid in tropical countries, the onset may be sudden and violent, instead of gradual, the temperature running up to 103° or 104° the first evening. But in this respect it does not differ from typical typhoid. If it goes over the 21st day it goes on to the 28th. Sometimes a case apparently of this nature stops on the 14th day, like a case of mild typhoid telescoped into a fortnight, to use Hughes' description of this fever. I do not recall any relapses; the patients make uninterrupted recoveries and convalescence is rapid. The cases as a rule begin as "simple continued fever," then you begin to suspect typhoid, and you look carefully day after day for the various indications of that disease, but you do not find them. As these cases occur most frequently in the cold season or early summer, when there is no prickly heat to obscure the diagnosis, you would have no difficulty in seeing the characteristic spots if they existed; but they do not appear. There may be some tumefaction of the abdomen, from the soups and liquid food the patient is fed on, but there is no pea-soup diarrhœa. If there is any diarrhœa at all, it is of an ordinary bilious kind. Alongside of a case of this kind you will have one of typical enteric, and the difference is very marked.

Natives suffer occasionally from this Urban Continued Fever, and therefore the presumption is against typhoid fever.

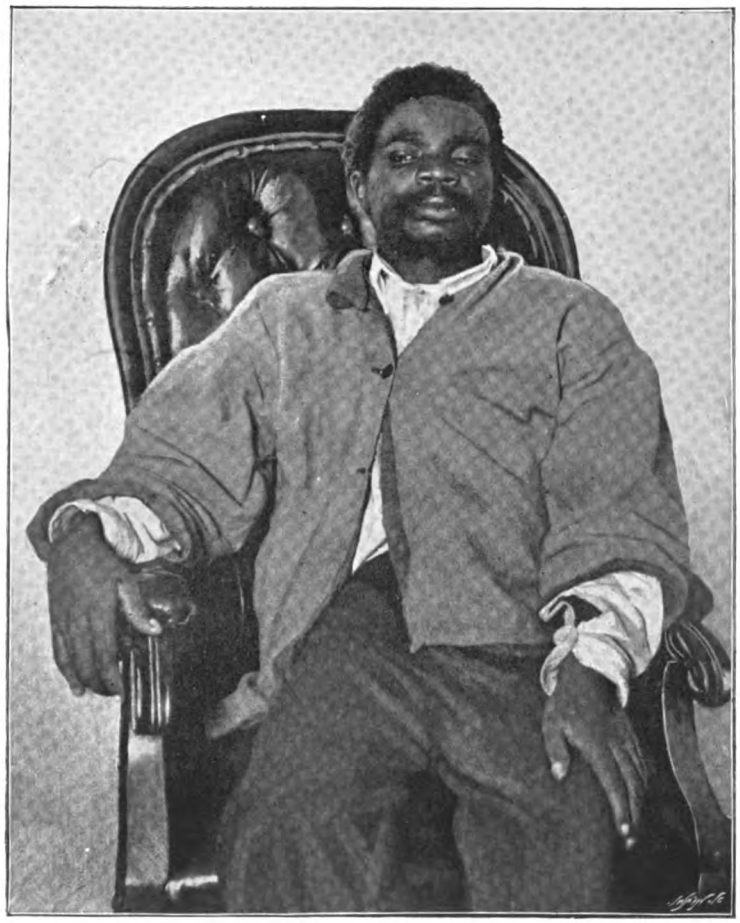
I was in the habit of seeing from twelve to twenty cases of typhoid fever in the European General Hospital and in private practice every year during the ten years I was in Calcutta, and also about an equal number of the cases I am now describing, and there was no room for doubt that they were distinct types of illness, so that one could say this is a case of Calcutta Fever, and this is a case of true typhoid. Hughes has seen this fever in Gibraltar, Egypt and the Mediterranean, distinguished from typhoid on the one hand and from Malta Fever on the other by its course, which is one of definite duration, and without the distressing concomitants of the latter disease. Manson has seen it in China; Billings, of the United States' Army, has seen and recognised its differences from ordinary typhoid. "In the United States" he says, "we have many cases of mild continued fever, lasting from twenty to thirty days, which we presume to be typhoid, but which certainly are not typical, and which shade into malarial forms of fever in a very puzzling way" (quoted from Fayrer in "Allbutt's Syst. of Med.," p. 344).

By many practitioners such cases would be returned as cases of typhoid fever, and there are many reasons to justify such a procedure, such, for instance, as the sudden occurrence of intestinal hæmorrhage in a case of this character, and it may be that some of them are really mild typhoid. But in the absence of all the symptoms of typhoid fever, most of us who see this type of fever among the civil population of large towns, and it is almost exclusively confined to these populations, speak of it by its local names of Calcutta Fever, Bombay Fever, &c., and enter it in our hospital records as Simple Continued Fever. It differs from this, however, in its definite and regular course, which bespeaks a specific cause, and I do not think that the rare occurrence of intestinal hæmorrhage is under the circumstances sufficient alone to necessitate a change in diagnosis. The bacillus of Eberth is not the only one which is capable of causing a continued fever of typhoid type with ulceration of the small intestine. In making *post-mortem* examinations in India it is not a very rare thing to find ulceration of the solitary glands, especially in the upper part of small intestine, in the duodenum especially—a fact noted long ago by Annesley, and Twining, and others, in cases where no suspicion of typhoid fever, or, indeed, of any kind of fever, exists; and we know that a bacillus, normal to the healthy intestinal tract, the bacillus coli communis, is capable, under certain favourable conditions, of producing a continued fever, accompanied with intestinal ulceration. May it not be some such alteration in the nature of the bacillus coli communis which brings about the type of fever I am now discussing, or some other micro-organism as yet quite unknown? We are only on the threshold of bacteriology, and when the door is more fully opened we shall have vistas of the relationships of disease to microbes of which we have no conception as yet.

But it may be asked, why do we not settle the question of the differentiation of urban continued fever from typhoid by the application of Widal's test? This will no doubt be done in the near future; but there are as yet no means in India of keeping up continuous cultures of the typhoid bacillus which are necessary for the reaction; and there are no men with sufficient leisure to do so.

In the meantime we have to trust to clinical distinctions such as those which seem to justify the separation of these cases of urban continued fever from the more typical cases of typhoid with which we are also very familiar, and give them the clinical distinction which their symptoms and course appear to entitle them to.

Non-Malarial Remittent Fever.—The next fever to which I will refer is "non-malarial remittent." I am sorry that I am unable to give it a better name. In the address I have spoken of which I gave at the Indian Medical Congress I pointed out that it was essentially a continued and not a remittent fever, and a negative cognomenn is always open to objection. The name "bilious remittent" has a serious objection to it, inasmuch as it has already been given to an entirely different fever, the hæmoglobinuric fever of central Africa and the West Indies. But it was given to this fever under a misapprehension of its nature, it



SLEEPING SICKNESS.

By PATRICK MANSON, M.D., LL.D., F.R.C.P.

See Vol. I., page 122.

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being thought that the dark colour of the urine, produced in reality by the colouring matter of the broken-down blood corpuscles, was due to bile. The real nature of hæmoglobinuric fever is now known, at least as far as this symptom goes, and so the name wrongly given to it is set free for more legitimate use, and I am not indisposed to appropriate it for the type of fever I am now about to discuss, the more readily as it is the name by which it is universally called by the native practitioners of Bengal.

This is a fever which, though it is sometimes seen in Europeans, is essentially a disease of natives, and is not common after thirty years of age, but is frequent enough in children. Its onset is sometimes gradual, like the preceding fever I call "urban continued," or like tropical typhoid, and more frequently, perhaps, the fever of the first few days is distinctly remittent, or it may be even intermittent in type; but after the third day it assumes its true continued character, which it maintains throughout its course, The temperature by the fourth day may reach 104° and 105°; and I have known it continue at these figures persistently day and night, without any remission for several days at different epochs of its progress; more frequently there is a remission of 2° or 2·5° at some part of the twenty-four hours. With these continued high temperatures, head-symptoms, especially, delirium of a muttering and irritable kind, come on, and the patient may, and often does pass into a condition of coma from which he can hardly be roused. Hepatic congestion and enlargement are early and constant symptoms, but the size of the spleen is not palpably altered. Diarrhœa of a bilious kind is the rule. These signs of early hepatic derangement of the liver functions justify the use of the word "bilious" to distinguish this kind of fever, and slight jaundice is not very rare. We have now a condition which continues in the less unfavourable cases for three, four, or five weeks, without material variation; a persistent high temperature without very marked remission, a distinctly enlarged and tender liver, with bilious diarrhœa and more or less flatulent distension of the bowels, with low muttering delirium, which is generally well marked by the 18th to the 24th day. About this time congestion of the bases and backs of both lungs will be detected, the delirium passes into coma, and the patient dies in fatal cases about this period. In more favourable cases, the symptoms are less severe, they continue a week or two longer, the usual duration of the illness being six weeks.

These cases form a very consistent picture which is often repeated in the experience of any one who sees a good deal of native practice, though this type of fever is by no means confined to natives. It is not easy to say what part malaria plays in bringing about the *tout ensemble* in these cases. They are probably more common during the early months of the cold weather, when the effects of malaria are most in evidence; and the symptoms of the first few days are often distinctly malarial in their character. Indeed, some practitioners of large experience say that "during a long residence in tropical climates they have never failed, by making the proper enquiries, to trace back a case of remittent or continued fever to its original intermittent type" (Sullivan, quoted by Moore, "Diseases of India," 1880, p. 252). I think this statement is too sweeping, and there are several difficulties in granting an essentially malarial nature to these cases.

In the first place, you may search the blood every day for the malarial parasite in these cases without finding them. I do not consider this result to be by any means conclusive, because the same difficuliy is often experienced in continued or remittent fevers which are unquestionably of malarial origin and character, and which yield quickly to quinine. I know nothing more dissatisfying than the pursuit of the malarial parasite in the irregular remittents of Lower Bengal, an experience which is not confined to that region.

Secondly, even if the parasite were found in the first few days of the illness, this would not prove that the case was essentially malarial in character, though its presence might give a malarial type to the early symptoms, as so often happens. Laveran notices this fallacy when discussing the diagnosis of typhoid when it supervenes on a malarial fever. In such circumstances the two maladies co-exist, and one can only ask which at a given moment predominates. He has several times seen the micro-organisms disappear from the blood of a patient in whom this sequence of events had occurred, and reappear during convalescence when the patient had a relapse of intermittent fever, and I may here mention that very frequently indeed, in our most typical cases of typhoid fever in India, convalescence is delayed for the best part of a week by the supervention of fever of an intermittent type, for which quinine has to be given freely.

Thirdly, we are quite familiar with malarial continued fever, but it presents a very different aspect from that of the fever we are now considering. You may have the same continued high temperatures, but there is not the same tendency to head symptoms, and the cases are irregular in duration and are readily amenable to quinine.

Fourthly, the cases of bilious remittent I am speaking of resemble specific fevers in their regular course and duration, so that it is possible to predict their determination, which no treatment will enable one to anticipate.

And, in the fifth place, we have the evidence of the inefficacy of quinine. Of the use of quinine in distinguishing a malarial from a non-malarial continued fever, Laveran speaks in no uncertain terms. This is what he says: "Quinine offers a valuable means of diagnosis; if treatment with quinine cause the fever to fall at the end of two, three, or four days, we have probably to do with a continued malarial fever; if the fever persists beyond the fourth day, when the patient has consumed 5 or 6 grammes of quinine, one may conclude, almost with certainty, that one has to deal with a typhoid fever, or some other fever foreign to paludism" (Laveran, *Paludisme*, 1897).

Five or 6 grammes, 75 to 90 grains, in four days! I have treated these cases with 90 grains of quinine a day for a week, till they have fled from me to the arms of some milder villain (so my friends tell me), without producing any effect on the fever, beyond aggravating all its distress. Latterly I have only used quinine to establish the diagnosis on Laveran's

lines, and then I conduct the cases to their termination on general principles, regarding them as exemplifying some as yet unknown specific intoxication whose duration is definite and which I cannot influence.

Now are these cases of fever examples of some fever foreign to paludism (to use Laveran's expression) whose specific cause is as yet unrecognised, or are they representative of the malignant type of Malta fever? I admit that this is a legitimate contention if individual cases alone are considered. The symptoms of the malignant type of Malta fever are apparently those of the fever I have been describing, as they are detailed by Hughes. There is the same rapid assumption of temperatures of 104° and 105°, the early head symptoms, the pulmonary congestion, the diarrhœa and the possible fatal termination during the second and third weeks. The resemblance so far is very close, but the further progress differs. Malignant cases of Malta fever pass in a certain proportion into the ordinary undulatory type, with its indefinite duration and its anthritic and neuralgic concomita, whereas the duration of the bilious intermittent of Bengal is definite, is limited to six weeks, and there succeeds usually an uninterrupted convalescence, unmarked by the concomitants or sequelæ which are so frequent and distressing in Malta fever. It is true that sometimes, and especially is this so in children, after a few days or a week of apparent freedom from pyrexia there is a recurrence of the fever with all the previous symptoms in a milder degree; but this is clearly of the nature of a relapse such as we so often see in typhoid, and is a succinct attack similar to the first, but of a less severe type and shorter duration, which does not conform to the descriptions given of the recrudescences and partial abatements of true Malta fever with its indefinite duration and sequelæ. I would again say that it would be impossible to deny that a given case of the bilious remittent fever of Bengal does not at a certain stage very closely resemble one of the types of Malta fever, but a group of such cases is distinctly differentiated from a group of cases of malignant Malta fever by their more definite period, the infrequency of relapses and undulatory progress and the entire absence of the sequelæ of Malta fever. If it is Malta fever why do we have only the malignant form? Why do we never see the undulatory phenomena which are so marked in true Malta fever, so much so as to induce Hughes to give it its name of "*febris undulans*"?

I, however, grant that the resemblance is sufficiently great to make the search for the microcosms of Bruce, or some other related organism, in the spleens of fatal cases of Bengal bilious remittent imperative on those qualified for the task. I am, however, no more prepared to admit in the meantime, on the evidence before me, that the philosophy of Bengal non-malarial remittent fever is to be summed up in the words Malta fever than in that of typhoid. There is probably a great deal more to be understood in these fevers than we have yet any conception of.

I am aware, with regard to Malta fever, that the question of its occurrence in India has been recently raised by Dr. A. E. Wright and Surg.-Capt. Smith in the *British Medical Journal* of April 10, 1897. Up to that time its occurrence in India had not been suspected. These gentlemen examined the blood of ten invalids from India, of whom several were suffering from symptoms coinciding with the sequelæ of Malta fever, and found that on applying the serum-sedimentation test they all gave a positive reaction, which they regard as characteristic of Malta fever. Of these ten invalids seven had served at Sabathu, and Drs. Wright and Smith conclude that Malta fever is of frequent occurrence in India, and especially at Sabathu. Brigade-Surgeon Lieut.-Col. Macartney (*British Medical Journal*, May 29, 1897) who was evidently impressed by this observation of Wright and Smith, thinks that enteric fever and Malta fever occur side by side at Sabbathu, as they do in Malta and Gibraltar; but Surg.-Major S. F. Freyer (*British Medical Journal*, May 22, 1897) and Surg.-Col. Welch, (*British Medical Journal*, June 2, 1897) both scout the idea that Malta fever is prevalent in Sabathu.

Not having had an opportunity of observing the types of fever which prevail at Sabathu, I am not in a position to give an opinion on the subject of its occurrence in that hill station; but with regard to the wider question of the occurrence of Malta fever throughout India generally, I would like to submit the following observations:—

Malta fever is a fever which apparently is capable of manifesting itself under the most divergent clinical aspects, of which however, the second or undulatory type is the most common, so much so that Hughes, recognising the inadequacy of the popular title in the face of its extended geographical distribution, proposes for it the title of "*febris undulans*" in place of the popular name of "Malta" fever. This is the typical character the fever most commonly assumes, and the other types, if not quickly fatal, at some time or other are likely to assume the characteristic undulating progress during some part of their prolonged course.

Now I freely admit that it would be quite an easy task to select from the records of any hospital in India temperature charts having the same general character as those given by Hughes in his recent work on *Febris Undulans* as characteristic of Malta fever. Instances of continued fever are seen lasting a month or six weeks, with variations in temperature occurring more or less in waves such as his temperature charts show, cases which are not malarial, and whose progress is not influenced by quinine. Neither would it be possible, I think, at certain periods of certain cases of continued fever in Lower Bengal, judging from the temperature chart and the febrile symptoms alone up to a certain point, to prove to a critic that such an individual case was not one of Malta fever. A fever which is so Protean in its clinical characters as the description of its three types shows Malta fever to be is one which may present the characters of any fever which one may name. Indeed, this diversity of phenomena has been one of the reasons which delayed so long its recognition as a separate entity, and which gained for it the many titles which it had before the discoveries of Bruce, Gipps and Hughes, fixed its true nature.

It is probable that Laveran himself in Algeria was deceived by the intermittent type of Malta fever, for he speaks of cases of typhoid fever in which the morning temperature was normal throughout their whole course, and also of sudoral cases of typhoid in which the febrile paroxysms are very marked, and accompanied by abundant perspiration, resembling the phenomena of intermittent fever, the nervous and abdominal symptoms being of little intensity, and the duration of the malady about five weeks. These were probably not cases of a typical typhoid as Laveran supposed, but of Malta fever. But as Laveran says, the evolution of the malady distinguishes Malta fever from all other forms of fever. It may not be easy at a given moment to distinguish a case of another kind of fever from one of Malta fever at a similar period, but the further progress of the case will soon make the diagnosis easy, and I think there can never be much difficulty in distinguishing a group of cases of Malta fever from a group of cases of fever of another type. The prolonged course of the majority of cases of Malta fever extended by frequently recurring undulations of fever during several months, together with the rheumatic affections of the joints, which, according to Hughes, occur in 40 per cent. of the cases; the neuralgias, the orchitis which is so common, produce a clinical picture which is unique, and which I must say is unknown in the parts of India where I have practised. I speak with the more confidence on this point from the fact that it has been my fortune to have had some twenty cases of Malta fever under my own observation and treatment for several weeks. They were landed from a ship of war which brought them straight from Malta to Calcutta, and dumped them down, if I may use the expression, in the middle of our ordinary cases of fever, in the wards of the European General Hospital at Calcutta, of which I was superintendent. These cases naturally were the subject of much excitement and interested observation among the staff of the hospital, and there was only one opinion, that the clinical picture they presented was perfectly new to us, and that there was no possibility of confounding such a group of cases with anything we had ever seen before. The whole aspect of the patient, the progress of the fever, the neuralgias, the swollen and painful joints, the orchitis from which some of them suffered, marked them off sharply from the cases of continued and remittent fever lying in the adjoining beds, and with which we were so familiar.

So strongly do I feel on this question that Malta fever is not prevalent in Lower Bengal, that if it were found that the blood of any considerable number of cases of fever from that part of India gave the reaction alluded to by Wright and Smith, I should regard it as seriously discrediting the value of the serum-sedimentation test, and I believe that I have had exceptional opportunities of forming an opinion on the subject.

Double Continued Fever.—The remaining fevers on my list will not detain us. Manson ("Tropical Diseases," p. 216), in addition to the types of continued fever which I described in 1894, recognises as occurring in China, and he believes as a distinct clinical entity, a form which from its peculiar feature he calls "double continued fever." This he met with in South China, both in Amoy and Hong Kong. It was characterised by an initial pyrexial stage of from ten to fourteen days' duration, followed by a stage of from three to seven days' relative or absolute apyrexia, which in turn was succeeded by another spell of about ten days' duration of smart fever, and then by convalescence. Both in the primary and terminal fever the evening temperature may rise to 104° and 105°. He has seen on at least two occasions the same succession of events occurring almost simultaneously in two patients living in the same house; once in a husband and wife, and once in a brother and sister; and he therefore thinks that this is a special form of disease, and that the double character of the fever is a constant and characteristic feature. In the case of the brother and sister the march of their fevers was strictly simultaneous; the primary fever, the apyretic interval, and the terminal fever occurring in both patients on the same days. There are no special symptoms, nor any apparent complications, and it is of little gravity as regards life.

I cannot recall any experience of a fever having this peculiar double character, at least as occurring synchronously in two patients; but now that attention has been called to its occurrence, other cases may be notified as being met with in other parts of the world.

Acute Febrile Uterus (Weil's disease) has been met with in India in epidemic form, though its true nature was not recognised. The native doctor who reported and described the cases returned them as yellow fever. It is an acute infectious disease characterised by fever and jaundice. It sets in abruptly, often with a rigor, and is charactised by marked remissions. The liver and spleen are enlarged and the former is tender; jaundice is early and may be severe, with clay-coloured stools. There are no gastro-intestinal symptoms and the duration of the attack is about fourteen days. From observations and experiments which have been made, it is not improbable that it is due to a bacillus, the *bacillus Proteus fluorescens*. It is an interesting entity which, as it has been reported in India, may be met with again, though it has not been so in my own experience, at least as an epidemic.

III.—*Fevers of Compound Origin.*—A "typho-malarial" fever has been much written of, especially by American authors, but there is a pretty general consensus of opinion that it is not a specific fever, but rather that its peculiarities are due to the concomitant action of the two organisms, the typhoid and malarial, working together in the same person. An epidemic of an atypical form of enteric fever was observed by Karlinski in Bosnia in those who had recently suffered from malaria, which was called dog-typhus (Hunde-typhus) and typhoid bacilli were found in the stools (Dreschfeld). In India this combination of malaria and typhoid serves to obscure the early symptoms of typhoid, giving us in a great many cases, I think the majority of cases, a sudden and violent access instead of the usual gradual step-like rise of temperature in the first few days. This, a typical character of the onset, greatly increases the difficulty of diagnosis in the first week, and I do not think we

would get any great assistance from the microscope or from Widal's test. The discovery of Laveran's organisms in the first day or two would only make the error in diagnosis more likely if there were a malarial element in the case, and, on the other hand, Widal's reaction is not often present before the sixth day and sometimes much later. Beyond giving rise to this increased difficulty in diagnosis, and by leading to a delay in the defervescence by often introducing an intermittent character into that period of the fever, I do not think that the moderate presence of the malarial habit has any appreciable effect on the progress of an ordinary case of typhoid fever, if I may judge by my own experience.

Dreschfeld ("Allbutt's System of Medicine") mentions a type of fever which he calls spleno-typhoid, in which there is excessive enlargement of the spleen, and an absence of intestinal symptoms, and in which the affection of Plyer's patches is not well marked, only congestion and swelling being noticed. Such a condition is often described by the older writers on Indian fevers, and as long ago as 1876, in a report on the fevers of Burma, I recorded several cases of typhoidal fever in which the intestinal ulceration was not longitudinal as in typhoid, but transverse, as in a case recently reported by Snell, and commented on by Sir William Broadbent (*Lancet*, July 30, 1898). These cases would all seem to show that we have not yet heard the last word regarding the bacterial pathology of typhoidal fevers.

(*b*) *Kála-Azar*, a fever which is depopulating large tracts of country in Assam, is one which I have not had an opportunity of observing, and I do not venture to enter on the controversy which it has raised. It is an epidemic fever which spreads slowly from village to village, gradually enclosing a large area. It presents all the phenomena of the malarial cachexia in an aggravated form, but the difficulty of regarding it as purely malarial lies in its obviously infectious nature. It is true that malaria has occasionally behaved like any other infective disease, and apparently spread from person to person; witness its rapid prevalence when first introduced into the Mauritius, where it was previously unknown; but the conditions of its slow progress in Assam are hardly satisfied by that hypothesis. Much importance has been attached to the presence of enormous numbers of the ankylostomum duodenale found in cases of kála ázar. Dobson, however, found this parasite to be present, often in large numbers in coolies passing through Dhubri on their way to Assam, and who exhibited no symptoms of ill-health. I think that the pathology of kála-ázar requires still more elucidation before it can be stated with precision, and that it needs for its manifestation a combination of two or more parasites, probably the malarial in a particularly virulent form, or of some other hæmatozoon, along with the debilitating effects of ankylostomiasis, to explain the pathological phenomena as well as the slow epidemic procession of the disease.

(*c*) *Hæmoglobinuric Fever*, to which the name of bilious remittent fever was formerly given, is practically unknown in India. Though a few doubtful cases have been reported by Nother, Firth, Wenyon and others from India, Assam and Cochin China, it is confinded to tropical and sub-tropical regions of Africa and America. Personally I have never met with it. I have only seen the symptoms of hæmoglobinuria in two cases of malarial poisoning, one in the person of a prisoner in the Dacca jail, and another in that of a tea-planter from the Terai. Koch seems to favour the idea that this symptom is in some way connected with the action of large doses of quinine in malarial fever. Taking into account the enormous doses of quinine given daily in India under all conditions of malarial poisoning, and the absence of hæmoglobinuric fever in that country, it is not possible but to demur to such an opinion of its causation. Its limitation to certain geographical regions in Africa and America would rather seem to indicate that there is some other hæmatozoon at work there, which may be related to the amœba of Laveran or associated with it, and possesses properties which enable it to give rise frequently to a symptom of which Laveran's parasite is incapable alone.

A Bacteriological Laboratory for Johannesburg.—No worthier end could be served by the recent Medical Congress in the Golden City than the establishment of the laboratory for which Dr. Geo. Murray put in so strong a plea in returning thanks to Mr. Eckstein for his sumptuous entertainment at the Robinson Mine. The President of the Congress clearly brought out what a great saving a small initial outlay for this purpose would effect in the expenses of mine management, by teaching how to preserve the health of the great industrial army, how it would foster the working enthusiasm of the medical officers, and how it would assist in the development of medical science in South Africa by providing a scientific centre for the study of South African disease problems, and the many unstudied natural remedies we possess. He also demonstrated in how large a measure it might be self-supporting, and, finally, he expressed his belief that it would prove a powerful lever to raise the professional level of the practitioners in contact with it. All medical South Africa will endorse this.—*South African Medical Journal.*

Infection of Healthy Areas by Diseased Troops.—A correspondent in the *Journal of the American Medical Association* calls attention to the real danger there is of the troops returning from Cuba scattering broadcast in America the germs of tropical dysentery and tropical anæmia. Soldiers travelling home along the various railroads are liable, he points out, to infect the entire country with the amœba and with anchylostoma—the parasites causing the two diseases in question. He proposes that in order to prevent this all returning soldiers suffering from dysentery, tropical anæmia or typhoid fever, should be confined in suitable hospitals, and kept there as long as the microscope shows them capable of being a source of infection to others.—*Indian Medical Gazette.*

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MALARIAL NEURITIS.

(Communicated.)

WHETHER he be right or wrong in calling the cases of polyneuritis, described in the November issue of THE JOURNAL OF TROPICAL MEDICINE, "Malarial Neuritis," Dr. Highet has done a distinct service to tropical medicine by drawing attention to a subject which, so far, has been too much neglected. Besides those forms that are common to all latitudes, such as are connected with diphtheria, gout, alcohol, the metals, &c., there are at least two types of polyneuritis more or less frequent in, and peculiar to, warm climates, namely, beriberic neuritis and leprous neuritis. Are there other forms? If so, what are their clinical features, epidemiology, and etiology?

It can generally be taken for granted that when some phase of tropical disease is recognised for the first time, in the absence of any very evident cause, that disease at the outset is nearly certain to be attributed to malaria. This is the history of most tropical diseases. Thus it has happened that at one time or another cholera, dysentery, Malta fever, scorbutus, liver abscess, elephantiasis, black-water fever, sprue, and a host of other diseases have had such an etiology assigned to them. In most instances the only, or at all events the principal, reason for such an etiological assumption has been the fact that the diseases in question may, and do, occur in patients who have at one time suffered from malaria, or who have visited places in which malaria is endemic. In not a few instances the reason for the assumption is not so well grounded as this even; the fact of their occurring in a warm climate being held as amply sufficient to justify their being attributed to malaria.

A moment's reflection, however, should put us on our guard against jumping in this way to so unwarrantable a conclusion. The human mind is so constituted that so soon as it perceives a phenomenon it seeks for cause and explanation. When these are not on the surface and at once forthcoming we are apt to pitch on some concurrent or antecedent event or fact, notwithstanding that such event or fact may be related to the phenomenon in question simply as an accidental, or, at most, as a predisposing circumstance. This is obviously highly unscientific. Not only is it unscientific, it may be damaging and even dangerous; for when such a conception crystallises into a nomenclature it is apt to satisfy the mind, and thereby to arrest search for the truth, and even to influence injuriously methods of treatment.

In illustration we would refer to Colonel K. Macleod's interesting and valuable article on "Malarial Scurvy" in the September number of this journal. Such an expression as "malarial scurvy" is apt to convey the impression that scurvy is caused by malaria. That scorbutus may occur in a malarial is certain, and that malaria may occur in a scorbutic is also certain; but to say that malaria can cause scurvy is just as erroneous as to say that scorbutus can cause malaria. We do not for a moment mean to say that Colonel Macleod affirms that the plasmodium causes scurvy; but we do mean to say that to many the expression "malarial scorbutus" will convey and lead to the adoption of this idea. It is extremely desirable, therefore, that the adjective "malarial" be used with caution, and only in

connection with such pathological conditions as can be distinctly shown to be directly dependent on, or connected with, the plasmodium.

It is true that the fact of the concurrence of two phenomena, although not conclusive, is to some extent in favour of the existence of a cause and effect relationship between the concurrent phenomena. But when two facts are separated by a long interval of time, unless they are very frequently or invariably associated, an argument based solely on their occurrence in the same individual is very weak indeed. It is manifestly illogical to conclude that because a man had at one time been in the tropics any disease he subsequently suffered from was caused by malaria. It would be just as logical, should that man break a leg, to say that the fracture was the result of malaria. Nevertheless, illogical though such a style of argument certainly is, we constantly meet with it. Thus in an article on Malarial Neuritis, in the *British Medical Journal* of May 8th, 1897, referred to by Dr. Highet, the author confidently attributes two cases of polyneuritis to malaria, and for no better reason than that in one of the instances the patient had had an attack of ague 17 years before, and in the other instance because the patient had merely visited the tropics!

To quote the author's words: "In the first patient, you will all, I think, be disposed to admit that to ascribe his malady to malaria is justifiable enough. He had ague some 17 years ago, and although he has had no attacks since, and although there are now no malarial changes in his spleen or other organs and tissues, we have to remember that the malarial poison, having once gained an entrance into the body, may yet manifest its presence at any time subsequently. . . . In the second patient, W., the acceptance of the malaria as the origin of his symptoms might seem a little more difficult, for not only does he now present none of the ordinary pathological changes of malaria . . . . but he tells us positively that he has never had malaria at all. He has, however, as a sailor, been much in malarial districts." The premisses do not justify this writer's conclusion; he has no more justification for calling these cases "malarial neuritis" than he would have had he called them "nautical neuritis."

When, however, as in Dr. Highet's cases, we find definite evidence of active malaria, and the patient is at the same time the subject of polyneuritis, there is some justification for regarding the neuritis as a direct result of the malarial infection. At the same time such an inference must not be regarded as necessarily representing the exact truth, unless the fact of concurrence be supported by additional evidence. We must bear in mind that there are such things as double infections; in fact, one infection very often predisposes to the reception of another and totally different infection. This, indeed, is the view we incline to take of Dr. Highet's cases; at all events of the two cases of which he has favoured us with the details. Dr. Highet himself suggests that they may have been the outcome of such a mixed infection. Neuritis is certainly far from being a common effect of malaria. Were it a common effect we would see it much more frequently than we do. It is most significant that, in his collection of cases, Dr. Highet observed no fewer than eight of his examples of combined neuritis and malaria in the course of one very circumscribed epidemic. This certainly suggests something over and above malaria. Considering that this little epidemic occurred in Singapore, and considering the well-established reputation of Singapore as a beri-beri centre, the localised nature of the epidemic and the symptoms, we believe that his conjecture of a mixed infection is well founded, and that these cases were examples of combined beri-beri and malaria. It may be quite true that all the classical symptoms of beri-beri were not present in every one of his cases; nevertheless they were present in some of them, and we know that beri-beri, like many other diseases, very often presents remarkable variations in the severity and grouping of its symptoms, and that it is prone to attack those predisposed by any debilitating affection such as malaria.

Some time ago, Dr. Strachan, then of Jamaica, published ("Salous Annual," vol. i., 1888, and *Practitioner*, 1897) an admirable account of a type of neuritis remarkably prevalent in that island.

Other medical men possessing practical familiarity with the disease of Jamaica have, in conversation with us, confirmed Dr. Strachan's account. The subject is so important that we need make no apology for quoting Dr. Strachan's article in the *Practitioner*.

After mentioning that the form of multiple neuritis he describes is very common in Jamaica, occurring there both in blacks and whites, Dr. Strachan writes :—

"A patient presents himself complaining of 'numbness and cramps in his hands and feet, dimness of sight, and a tightness round the waist.' . . . . If the case be somewhat more advanced, he may add to this the statement that he fears he is getting 'hard of hearing.' He goes on to say that he suffers from severe burning in the palms of the hands and soles of the feet, and that very often this is worse at night than in the day, and that the pains and the burning heat prevent his resting.

"On examination it will be seen that there is slight excoriation, with fine, branny desquamation of the edges of the eyelids, margins of the lips, and around the margins of the nostrils; the palpebral conjunctiva may be hyperæmic, as well as the lips. The heat in the hands complained of by the patient will be found to be not merely subjective but appreciable to the touch, and due to a hyperæmic condition of the palms; the acuteness of vision for form will be found to be more or less impaired, according to the stage to which the malady has progressed; examination of the main nerves to the extremities will show that they are very tender on pressure, especially the ulnar nerve, and along the distribution of their terminal filaments they may be tracked by lines of fine herpetic vesicles.

"On admitting such a case to hospital, and watching its further progress, it will be noted that at night the patient will be awake for hours, rubbing his feet and legs most probably, and moaning with pain. The loss of vision will proceed until he can with difficulty distinguish a large object immediately in front of him, and cannot recognise individuals. The muscles of his limbs will waste until the 'claw' hand and foot are features, and this wasting of muscles and the disappearance of fat will produce an emaciation which is very noteworthy in advanced cases. There will be found to be no alteration in the reaction of the pupil to light and accommodation, no falling when the eyes are closed, and the sphincters will not be affected.

"Should the disease make further headway, the patient may become a mere helpless skeleton, unable even to feed himself, his breathing laboured from implication of trunk muscles in the general muscular atrophy, almost blind, and with, perhaps, an ulcer on the cornea, quite deaf, and with, possibly, small bullæ on the extremities. There may also occur during the course of the malady monoplegias, as facial palsy, palsy of some of the external muscles of the eyeball, and (but very rarely) of some group of muscles of an extremity. The temperature chart will show a subnormal condition in the mornings, with evening rise of one or two degrees. A fatal termination is fortunately rare. When it occurs it is due to dyspnœa and the riotous action of the heart, resulting from vitally important nerves becoming involved in the now almost universal nerve change.

"As a rule, however, under appropriate treatment recovery gradually—with perhaps, from time to time, slight recurrences of nerve inflammation —takes place; the patient becomes stronger, can help himself a little, assimilates food well, and puts on fat again; then is able to walk a little, first with help, and afterwards alone; his grasp, measured with the dynamometer, shows daily increase of muscular power; his sight clears up, and his deafness gradually passes away (though if this has been extreme it is usually one of the last symptoms to disappear)."

Did space permit, we would fain quote further from Dr. Strachan's interesting paper. We have given enough to show that he is dealing not with beri-beri (the absence of œdema, the paresis of the head muscles, the affection of sight and hearing, and the trophic skin lesions, prove this), but with some as yet unrecognised toxic disease of the tropics. At one time in Jamaica this affection was regarded as being malarial in

nature; but such a view, considering the peculiar geographical limitations of the disease, cannot be upheld. Clearly it is a special disease, a special type of multiple neuritis, about which we would like to obtain further information, not only as regards its clinical feat .res, but also as regards its etiology, geographical distribution, and treatment. P. M.

THE INDIAN PLAGUE COMMISSION.

THE evidence given by Dr. Lawrie before the Indian Plague Commission at Hyderabad seems to have raised some considerable interest in India. If the reports received in this country are correct, this was due to Dr. Lawrie's attitude towards M. Haffkine's anti-plague inoculation. We are not surprised at the attitude, and it is impossible to be otherwise than amused at the reasons set forth. It appears that, notwithstanding the fact that thousands of persons, young and old, have been inoculated in different parts of India by medical men, that the inoculated have been carefully watched, and not a single instance of blood-poisoning discovered, Dr. Lawrie is of opinion that the vaccine is a putrescent organic matter occasionally containing putrescent organisms, and declares that the use of it is opposed to modern antiseptic surgery, because of its likelihood to cause blood-poisoning. No one will gainsay Dr. Lawrie's views, that the injection of putrescent liquids is opposed to modern antiseptic surgery, and that it will cause blood-poisoning. This is an axiom established by Lister many years ago. But before bringing this accusation against a treatment which has uniformly been attended by excellent results, we would, at least, have expected in support of it some instructive and convincing facts. None appear to have been given, and until they are forthcoming it is impossible to consider the opinion in any more serious light than those crude and unintelligible notions propounded by the same author on the value of the malarial parasite and on the nature of the cholera vaccine, which gained a certain local popularity for the time but did not in the slightest degree affect the progress of truth.

A number of medical men have given evidence in Calcutta on the plague. Major Evans believed that the plague was introduced into Calcutta by infected articles arriving by rail and sea, the first patients affected in India being old residents. Cases of fever with glandular enlargement were common and undistinguishable from a mild form of plague, except by bacteriological examination. Major Charles, Professor of Anatomy at the Medical College, believed that in a case of plague, in October, 1896, in a child, the contagion arrived in bales of goods from Bombay, among which the child played. A rat caught it at the same time, and the plague bacillus was found in its blood. In 1898, he considered the plague was imported by sea and spread by rats from the landing jetties. There had been a great improvement in the sanitation of Calcutta in the last few years, and the city was better able to meet an epidemic. Dr. Clemow considered plague a filth disease. Rats might produce disease in a house, but not in a city; no second cases had occurred in houses disinfected with perchloride of mercury. He had treated 50 cases of plague with Yersin's serum and compared them with 50 in which no serum was employed. In each group 40 died and 10 recovered; 13 cases of plague were treated with Lustig's serum, and of these 10 died; he also mentioned 2 cases of a second attack of plague, both of whom died.

Dr. Cook, the Health Officer, stated that the deaths from plague in Calcutta, from April 14 to September 24, had been 142; that he had observed no ill effects from inoculation; that he had tested Haffkine's fluid and had invariably found it sterile; that he had inoculated 1500 persons, and none had been attacked with plague; that the plague stopped in Calcutta of its own accord; that it was not affected by the measures taken, and that he had been unable to discover the original source of infection. Major Green, Extra Health Officer to the Corporation, stated he had been in the employ of the Bengal Government since 1891, treating the poorest natives, and he had never seen among them, previous to the plague in

India, symptoms of a similar nature. There was no reason to believe that the plague was endemic. Dr. Justice described an importation of plague from Calcutta into two villages in Bengal which caused a circumscribed outbreak. He believed that the plague had not spread in Calcutta because the native huts were better ventilated and lighted than the chawls in Bombay. Dr. Bose was of opinion that plague was brought into Calcutta from Bombay in February and March by men who had evaded medical inspection at the inspection camps. He had never seen other diseases clinically resembling plague. Dr. Hossack considered that there was an unknown factor in the environment of Calcutta inimical to the spread of the plague epidemic. He had seen mild cases of fever with enlarged glands at Poona which he did not believe were cases of plague. He had not made a bacteriological examination of the latter cases. Dr. Banerjee said that he had attended 14 cases of plague, and had seen cases of fever with glandular enlargements, but the symptoms were not similar to those of plague.

Article for Discussion.

THE POSSIBILITY OF EUROPEANS AND THEIR FAMILIES BECOMING NATURALISED IN THE TROPICS.

III.

The Jews, perhaps, afford the best example of a people capable of living in any climate and continuing to preserve their physical power and mental ability. When they came into prominence in the world's history they were settled at the eastern end of the Mediterranean, now in Palestine, then in Egypt, Arabia or Syria. Whence they sprang is unknown; the contention that they were a northern people is favoured by the fact that fair skins, fair hair, and blue eyes are common features, even at the present day, and by many it is held to be the original type. Of the four primitive sects, which blended together to form the original Hebrew tribe, some may have been fair and others dark, or by admixture with native races the colouring and feature may have altered. Time and climate, on the other hand, may have succeeded in determining a local character to the appearance of the Jew. In the sweltering heat of Bagdad it is but natural that, in the process of evolution, a darker type would prevail, and that in the snows of Poland a fairer complexion would be engendered.

There are, however, black Jews to be met with in several parts of India. But the black Jews of Cochin and elsewhere on the Malabar coast of the Madras Presidency, in Goa, and the black offshoot from the Beni Israel class in Bombay, are regarded as proselytes and the emancipated slaves of the white Jews, with whom they have no inter-marriages.

The Jews, therefore, are essentially a white people, and any intermixture with their dark coloured neighbours is held to be a contamination, and the offspring a race apart. In almost every country of the world is the Jew to be found. His progeny is capable of living and multiplying for generations in every Continent and in any part of every Continent, be it torrid, temperate, or tropic. The power of the Jew to adapt himself to climate is remarkable and well known. He is supposed to be able to stand tropical heat better than the European, whilst at the present day the majority of his race inhabits cold climates. The Jew would seem to break down the barrier we are inclined to apply to the possibility of white people dwelling in any climate; granted that he is of northern origin, then it is evident he can live and continue his race in tropical countries; or if the argument places his cradle within the tropics, then it is evident that he can live in cold as well as in hot climates. We are apt to consider the possibility of a race living in altered conditions only from the point of travelling from colder regions to warmer; but we have examples of the opposite. The negro from the equatorial regions of West Africa finds a home in which he thrives and multiplies in the United States of America, and not in the south only, but in the more northerly States, he flourishes in health and numbers.

Several further examples might be cited; but space does not permit of further discussion. It, however, would seem, that as the red man can live anywhere in the American continent, as the Jew can continue his species in all parts of the world, as Europeans can colonise tracts within the tropics as in South Africa and Australia, and as the negro can live and multiply in temperate or even cold climates, that the human being can adapt himself to any climate on this globe, and that it is disease and not climate which determines the possibility.

J. C.

THE SIESTA.

Gold Coast Custom.

In regard to the usefulness or harmfulness of the Siesta, a discussion on which has been opened in the columns of the Journal of Tropical Medicine, I may be permitted to record the result of four years' residential experience in West Africa.

On the Gold Coast the mid-day meal is taken between 11 and 12 o'clock, and it is the custom for work in the offices of the Government and of the mercantile community to cease at 11 a.m., and be resumed at 1 p.m. Indeed, the same rule is adopted by all classes of the population, Native and European, and the artisan or labourer has his mid-day "chop" at the same hour as his master, and, like him, enjoys a post-prandial "forty winks." The prolonged siesta of certain Eastern and South-American countries is adopted by very few of the European residents in the Gold Coast colony, except on Sunday, when the mid-day meal is often later than on week days, and is in many cases followed by one or two hours' sleep.

I think I am correct in saying that it is only "the old coaster" who systematically indulges in a two hours' sleep in pyjamas during the hot part of the day. On the other hand, the tendency among the more recently arrived Europeans is to a light luncheon, followed by a pipe and a short nap before returning to business, and I cannot say that I consider this habit to be injurious. The natural tendency of all animals in the tropics is towards repose during the hottest hours of the day. Man is no exception to this rule, and it is a question how far this prompting of nature should be obeyed or resisted by the European residing in a tropical country.

The inclination to sleep after a meal consumed during the heat of a tropical day appears to me to be a natural and physiological phenomenon. At a period when the cutaneous circulation is most active and when the portal system is the seat of an active and physiological congestion, the cerebral centres are necessarily depleted and mental apathy and drowsiness the natural results.

It is not the afternoon sleep *per se* that is objectionable on hygienic grounds, but rather the excessive indulgence in it, and the conditions which lead to this excess, the most potent and injurious of which is the ingestion during the hottest part of the day of a full meal of animal food accompanied by alcohol. The point really at issue, as it appears to me, is not the mid-day sleep but the mid-day meal. To gorge the portal system with nitrogenous food and alcohol at that period of the day when the greatest demand is being made on the cutaneous circulation is obviously to stray from "the ways of physiological righteousness," and the sleep which almost certainly follows is equally certain to prove unrefreshing or even positively injurious. On the other hand, the system requires to be nourished and the strength renewed between the early hour of breakfast and the late hour of dinner, and if a light and wholesome luncheon be followed by a light refreshing sleep, I do not see that any objection can be taken on purely physiological grounds to the siesta in this modified form. For a European, work of any kind in the tropics is of necessity more exhausting than in temperate climates, and if the native clerk or native carpenter finds that nature sends him to sleep after his morning's work and his 12 o'clock "breakfast," it does not appear reasonable to me that a European after his work of the forenoon should resist what would seem to be a natural inclination to repose, provided, as already suggested, such inclination be not artificially stimulated by over eating and drinking.

W. M. E.

Recent Literature on Tropical Medicine.

OPHTHALMOLOGY IN ITS TROPICAL BEARINGS.

RETINAL CHANGES IN PERNICIOUS MALARIAL FEVER.—Professor Guarnieri, of Pisa, has recently (*Arch. p. le Scienze Mediche*, vol. xxi., No. 1) published a valuable paper describing the results of the microscopic examination of the retinæ in ten fatal cases of pernicious malarial fever. Punctiform hæmorrhages abounded in the substance of the retina, mainly in the inner nuclear, outer molecular, and outer nuclear layers, none as deep as the rods and cones; they could be traced to thrombi of the vessels of the external retinal plexus. The retinal vessels were gorged with plasmodia and pigmented leucocytes. A large number of pigmented elements (parasites ?) were also found in the choroidal vessels, producing thrombi here and there. These observations are of great interest, and carry our knowledge of the pathology of malarial eye affections a step farther than Poncet's descriptions (quoted in my paper on Malarial Eye Affections in the September number of the JOURNAL). Professor Guarnieri does not describe any changes in the coats of the vessels, but it seems probable that the thrombi in malarial retinitis are the direct result of changes in the endothelial lining, as is the case in albuminuric retinitis.

CONJUNCTIVITIS IN ALGERIA.—Dr. H. Gros, of Lourmel, in Algeria, describes in the last number of *Janus*, a peculiar form of conjunctivitis ("seasonal" conjunctivitis, *conjunctivite végétante*), which he considers a disease *sui generis*, with a special form of diplococcus in the secretion. It bears some resemblance to vernal catarrh and trachoma, but differs from the former in sparing the cornea and bulbar conjunctiva, and from the latter in never causing paunus and in attacking children almost exclusively. The changes in the palpebral conjunctiva described by the author seem indistinguishable from those seen in trachoma, but the disease is very amenable to treatment. On the whole it seems probable the disease is a mild form of trachoma, and that its prevalence at particular seasons is due to some exciting cause or causes peculiar to the locality.

THE OCULAR MANIFESTATIONS OF LEPROSY.—MM. Jeanselme and Morax contribute an elaborate monograph on the ocular manifestations of leprosy to the November number of the *Annales d'Oculistique*, passing in review successively the leprous diseases of lids, conjunctiva, cornea, sclerotic, iris, choroid and retina. This classification, according to site, is purely one of convenience, as the bacillus never localises itself in one part of the eye to the exclusion of others. Tubercular leprosy is that which chiefly attacks the eye; the percentage of tubercular lepers in whom ocular lesions are found being no less than 98. The authors refer to Trantas' case (*vide* October number of the JOURNAL) as the only published description of fundus lesions seen during life. They found Hansen's bacillus in abundance in all forms of leprous infiltration of the eye.

The mode of infection of the eye in leprosy remains to a certain extent unexplained. The probabilities are in favour of the bacilli reaching the eye by means of the blood-vessels, and localising there through embolisms; but this does not explain the fact that localisation in the anterior half of the eye is almost the rule; why should emboli be so rare in the central artery of the retina or its branches, while so common in the anterior ciliary arteries? Ocular tubercle presents a similar problem. Poncet (*Bull. de l'Acad. de Méd.*, January and June, 1888) and Babes ("Histology of Leprosy," Berlin, 1898) believe the ocular infection to be from without, and suppose the conjunctiva to act as one of the gates of entry of the bacillus into the body. It is difficult to understand how they can reconcile this hypothesis with the known facts that leprous eye-lesions are always secondary, that when the conjunctiva is attacked it is always as an extension from subjacent changes, and that the invasion of the cornea by tubercle may be arrested by cauterising around the tubercle (Bull and Hansen).

M. T. YARR,
Major R.A.M.C.

AUSTRIA.

ON VISCERAL LEPROSY. By MAX JOSEPH.

The author examined the liver, spleen and tongue of a case of Lepra tuberosa, which had been extant about twenty years. The liver, kidneys and tongue showed no trace of lepra-bacilli; on the other hand, in the spleen, which microscopically was normal or nearly normal, there was an enormous number of bacilli, and the following were found:—(1) Smaller or larger heaps situated in the vicinity of small arteries (Malpighian corpuscles), forming in cells (mostly in vacuoles) and vesicular non-nucleated forms, which Joseph took to be vacated cells; (2) in the lymphatic vessels and cavities, and (3) free in the splenetic tissues. On the other hand, none were found in the blood vessels.

As an annex to his communication, the author passes in review the conditions of the spleen as found by other writers, which represent the most constant appearances of visceral leprosy.—*Janus*, December, 1898.

SIERRA LEONE.

In a letter addressed to the editor of "Public Health," of December, 1898, Capt. Smith, R.A.M.C., describes the following interesting experiment:—

Four Petri dishes were arranged thus:

No. 1, containing sterile coagulated serum.
No. 2, ,, culture of diphtheria on serum.
No. 3, ,, sterile coagulated serum.
No. 4, ,, ,, ,, ,,

A common house-fly was made to walk over the four surfaces of serum in the order given. Dishes 1, 3 and 4, were now placed in the incubator. No. 1 next day showed some cocci only; Nos. 3 and 4, showed colonies of diphtheria bacilli in the tracks of the fly.

We have here a simple explanation of one method of contaminating milk and other matters. In that flies, however, are absent in winter when diphtheria prevails, they cannot be regarded as a great factor in the spread of the disease, but in all probability they have some influence in helping to swell the autumnal rise in the prevalence of diphtheria.

News and Notes.

THE Java cinchona controls the market for this drug the world over.

TYPHOID FEVER AT HONOLULU.—Typhoid fever prevails extensively amongst the United States troops now at Honolulu. Three hundred cases were reported in dispatches received November 23, 1898.

PLAGUE BACILLI.—Dr. Haydon, of Melbourne, who visited India during the plague, took back with him some cultivations of the plague bacilli. Fearing an outbreak similar to that which occurred in Vienna, the Victorian Government demanded their surrender. This Dr. Haydon refused, unless compensated, whereupon the Government officials seized and destroyed them.

TREATMENT FOR LEPROSY IN THE FIJI ISLANDS.—There is a tree indigeneous to this country (the

Exæcaria agallocha), the venomous juice of which is supposed to possess the property of curing this terrible disease. Professor L. Lewin has received information on this point and on the manner of procedure, which is as follows:—The patient is placed in a narrow hammock made from the branches of the tree mentioned, rubbed with the fresh leaves, and afterwards enveloped in the same. Thus covered, his hands and feet are tied and he is dragged near to a little fire made of the branches of the famous Exæcaria. The patient's head is raised a little, but is in the midst of a dense black smoke. In short, he is made to take an aromatic (?) vapour bath.

Notwithstanding his despairing cries, the invalid is suffered to remain for several hours thus exposed to the smoke; the microbes of the leprosy are cooked (!) and, if the patient survives this desperate remedy, some days afterwards he is cured.—*Janus*, Dec., 1898.

ITALIAN investigators have proved and accepted Surgeon-Major Ross's observations on the life history of the malarial parasite in the mosquito, in all its details, as an established scientific fact.

HOW TO COLLECT MOSQUITOES (*CULICIDÆ*).[1]

List of Articles Required for Collecting and Preparing Mosquitoes.

ONE entomologist's collecting-net of book-muslin (one or two spare net-bags should be taken in case the one in use gets torn).

One dozen glass-*bottomed* pill-boxes (1½ to 2 in. in diameter is about the best size).

A cyanide killing-jar, or materials for making same, as follows:—

¼ lb. of cyanide of potassium (in lumps).

1 lb. of plaster of Paris.

A glass jar with wide mouth and closely fitting lid.

Entomological forceps (two pairs), with curved ends, for holding pins.

One ounce No. 20 entomological pins (D. F. Tayler and Co., New Hall Works, Birmingham. These pins are sold in boxes at 7s. 6d. per ounce, and as the pins are exceedingly fine, an ounce will go a very long way).

Common pins (three or four packets).

Gun-wad punch, No. 20 bore.

Cards (2-sheet Bristol board) from which to punch discs; a supply of the latter should be prepared ready for use.

Needles (two or three) mounted in handles, for arranging legs and wings.

A Platyscopic lens:—this is indispensable.

Cork-carpet or pith—one or two sheets about 6 in. square, on which to perform the operations of pinning, &c.

A strongly-made wooden box (a cigar-box will do), in the bottom of which is fixed a layer of cork-carpet or pith (if the latter is used it should be not less than half an inch thick).

[1] Issued by the Natural History Department, British Museum.

Importance of Sending Home Specimens for Determination in the Best Possible Condition.

It should be borne in mind that, for the purpose of the scientific determination of species, *mosquitoes cannot be collected with too great care.* As important specific characters are furnished by the *wings* and *legs*, it is of the utmost consequence that these should not be denuded of their scales, or otherwise injured; *unless attention is paid to this point the specimens will probably be quite worthless for determination.*

Spirit Not to be Used.

Specimens for determination must *on no account* be placed in spirit.

Specimens to be Pinned Immediately they are Dead.

Mosquitoes should in all cases be pinned, and that as soon as possible after death; duplicate specimens for dissection can, of course, be preserved in spirit, but if this is done care must be taken, by the use of corresponding labels or numbers, to prevent confusion between species.

Number of Specimens of Each Species Required.

In collecting specimens of a species of mosquito for determination some *half dozen* examples of *each sex* should, if possible, always be obtained.

How to distinguish the Sexes.

The harmless male mosquitoes can be distinguished from the females (which alone bite and suck blood) by the possession of plumose antennæ and palpi, forming tufts in front of the head; in the females the antennæ, though long, are nearly bare (having whorls of only short hair at the bases of the joints), while the palpi in the case of females of the typical genus *Culex*, to which the majority of the described species belong, are quite short.

Method of Collecting and Killing.

For capturing mosquitoes in the open an entomologist's collecting net is necessary, from which the insects can be transferred to glass-bottomed pill-boxes; in doing this great care must be taken not to pull off the legs; inside buildings it is possible, with care, to capture mosquitoes on walls and windows in the pill-boxes themselves. In any case mosquitoes should be collected alive in the glass-bottomed pill-boxes[2]; if care is taken, several specimens can be got into one pill-box. To kill the mosquitoes the box is opened a fraction of an inch on one side, and placed for a few minutes in a cyanide killing-jar[3], which must of

[2] These boxes can be obtained from any dealer in natural history apparatus, but care should be taken to see that the *bottoms*—and not the tops, as is often the case—are made of glass. Since the boxes are constructed of cardboard, they are liable in tropical countries to go to pieces in the rains, and to prevent this they should be covered with glazed jaconet (stuck on with liquid glue) and then coated with Aspinall's enamel.

[3] A cyanide killing-bottle can be procured ready for use from Hinton and Co., Bedford Street, London, W.C., or any other chemist will prepare one to order, but when mosquitoes (or indeed *any* Diptera) are collected in the manner here advised it is preferable to make a large-sized killing-jar for oneself as follows:—Take any fairly large glass jar (such as a pickle-bottle) with a wide mouth and closely fitting lid, and cover the bottom with a layer of dry plaster of Paris to the depth of half an inch; pour in above this a layer equal in depth consisting of *powdered* cyanide of potassium, mixed with rather more than its bulk of dry plaster of Paris; cover this mixture with a layer of dry plaster of Paris to the depth of a quarter of an inch or

course, be closed. As soon as the insects are quite dead (if the mixture in the jar is of reasonable strength from three to five minutes is sufficient, and mosquitoes should not be allowed to remain exposed to the effects of the cyanide longer than this) they should be turned out on to a sheet of cork-carpet or pith; they should be touched as little as possible, the manipulations necessary in arranging the wings and legs being performed with a needle.

To Pin a Mosquito.

Take a card disc and write on it all the data connected with the specimens to be pinned, as follows:—(1) Name of *locality*, including *altitude* if necessary; (2) *Date*—day, month, year—thus, 9, 11, 98; (3) *Collector's name*; (4) Any *remarks of interest, e.g.*, "Most troublesome species in district"; "Abundant in bamboo-jungle"; "Uncommon," etc. Place the disc on a sheet of cork-carpet or pith, and picking up with the entomological forceps one of the fine No. 20 pins, thrust about one-third of an inch of it through the centre of the disc; in doing this the pin should be held by the forceps below the middle, otherwise, owing to its fineness, it may bend and fail to pass through the card. Lay the specimen *on its back* (turning it over with the aid of a needle or one of the No. 20 pins held in the forceps), and thrust the pin, which now carries the disc, through the centre of the thorax, between the bases of the legs, until the tip of the pin projects one-sixth of an inch beyond the dorsal surface of the thorax[4]; invert the disc (the specimen will then be right side up), and thrust an ordinary pin through the disc near the margin for the purpose of carrying both disc and specimen. The next and last thing to be done is to arrange the legs and wings as far as possible; *i.e.*, the wings must be made to project at an angle from the body, and not allowed to remain closed, and the legs must be disposed symmetrically on the card disc so that all parts of them can be readily seen, instead of being left crumpled up beneath the body. These operations must be performed as gently as possible with the help of a needle mounted in a handle, or by the aid of a No. 20 pin held in the forceps, and care must be taken that hairs and scales are not rubbed off in the process. As the tissues contract in drying, the legs and wings are very apt to get pulled out of place, and, to correct these changes, the specimens should be examined once or twice during the next day or two after being pinned.

Transmission of Specimens to England.

Pinned specimens of mosquitoes, like those of other insects, rapidly develope mould during the rainy season in tropical countries, and since mouldy specimens are practically worthless for purposes of scientific determination, the insects should be sent home as soon as possible after being collected. To contain the specimens, if a proper entomological store-box is not available, any small strongly-made box (such as a cigar-box) will serve, in the bottom of which a layer of cork-carpet or pith (not less than half an inch thick, in the case of the latter) is firmly fixed. The greatest care must be taken to prevent specimens getting loose and rolling about in transit, since in this way a single loose disc might easily destroy or hopelessly damage all the other specimens in the box. To prevent this the pins supporting the cards should be inserted as tightly as possible into the cork-carpet or pith, and they should all be driven in to the same level; if this is done a sheet of soft paper (newspaper does very well) can be *fixed* into the box, resting on the heads of the supporting pins, in order to minimise the damage should a disc happen to get loose. The box containing the specimens should be well wrapped in cotton-wool, or similar material, and firmly packed in an outer box for transmission (by Parcel Post) to England.

N.B.—Not only mosquitoes, but all diptera should be collected, prepared, and sent home in the manner above described.

The above instructions, though drawn up with special reference to mosquitoes, are equally applicable to the collecting of diptera in general, except that in the case of the large forms, such as horse-flies (Tabanidæ), robber-flies (Asilidæ), &c., it is not necessary to use so fine a pin as a No. 20 (D. F. Tayler and Co.'s entomological pin No. 5—price 1s. 6d. per ounce—would do instead).

[Should collectors wish to have their specimens examined and reported upon, they can secure the same being done by forwarding them to the address of this Journal, when they will be submitted to competent authorities.]

so, and pour in above the whole a layer, half an inch in depth, consisting of plaster of Paris mixed with water to the consistency of cream. As soon as the top layer of plaster is dry the jar is ready for use. To obviate the risk of cracking the jar owing to the heat evolved when plaster of Paris is mixed with water, it may be advisable to stand the jar in warm water before adding the final layer. The exact amount of cyanide of potassium to be used is of no great consequence, but in the case of a properly prepared jar the odour should be readily perceptible on removing the lid; if it is not, the reason may be that the mixture is too dry, when a little water poured on to the top layer will probable set matters right. After some months' use the cyanide loses its efficacy (to obviate this so far as possible the jar should never be allowed to remain open), and the mixture must then be renewed.

[4] Should it be found impracticable to proceed in the manner here prescribed, owing to the difficulty of making the specimen lie in the required position on its back, it may be pinned in the ordinary way through the middle of the thorax from the *dorsal* side; in this case, however, the specimen must be *pinned first* (*i.e.*, before it is mounted on the card disc); it should be drawn two-thirds of the way up the pin, and the latter should then be thrust through the disc, holding the pin with the forceps *below* the specimen; mount the disc on a common pin, as in the first method.

Correspondence.

MEDICAL MISSIONARIES.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—I have read with interest the article in the December number of THE JOURNAL OF TROPICAL MEDICINE under the above heading.

The number of Medical Missionaries is said to be "very small," and so it is, relatively, to the number of men and women in the profession, but possibly some of your readers will be surprised to learn that at the present time there are 268 Medical Missionaries holding British qualifications; quite a sufficient number to make valuable observations and researches in different parts of the world.

The writer of the article refers to the proposed Residential Home for would-be Medical Missionaries, and also to the work of the Edinburgh Medical Missionary Society. No mention is, however, made of the work of the London Medical Missionary Association, which has had a Residential Home in London for many years past, and I believe at the present time it has some thirteen or fourteen inmates. It also ought to be known that some of the great missionary societies, such as S.P.C.K. and the C.M.S., make grants towards the cost of the medical education of men judged to be suitable for missionary work.

There are certainly very few men that can "master two learned professions," and therefore many hold that the Medical Missionary should not be ordained. No definition of the term "Medical Missionary" is given, but I hope that the writer of the article does not wish us to infer that the Medical Missionary is to give the whole of his attention to strictly medical work; if so, where does the "missionary" come in.

I do not think we should find a dearth of medical men, who would be willing to go abroad in order to care for the bodies of men, while clergy and others attend to the spiritual needs, but the *power of the Medical Missionary lies in his dual capacity*. There is not a sufficient supply of such doubly qualified men. The natives learn to love and to respect the knowledge and power of the man or woman who may have healed them, and they will listen to the healer about spiritual things when they will not listen to anyone else. The Medical Missionary must therefore have a good, simple, working knowledge of theology if he is to be efficient. In the same way the clerical missionary will almost always find that he has to do a certain amount of "medical" work, although he is not a Medical Missionary.

We are told "It is futile and may be disastrous to supply a man or woman with a medicine chest fitted with concentrated drugs of dangerous potency, without giving him or her that knowledge of anatomy, physiology, chemistry, pathology, medicine and surgery which could alone safeguard its proper use." Surely this is going much too far; the question is simply this, are missionaries going to out of the way places, far from any skilled medical attendance, to be left to chance, or are they to be supplied with such drugs as their own simple knowledge and the information given in simple books will enable them to utilise? Are they to have "tabloids" of quinine, tonics, aperients, simple sedatives, anti-diarrhœa preparations, ipecac., small doses of arsenic and opium, and other things for external application, or are they to be allowed to die of fever, general debility, dysentery, diarrhœa, &c., &c., so that we can cling to our old adage that "A little knowledge is a dangerous thing." If the missionary is aware that his knowledge is only small, the little is surely well worth having.

If the writer of your article was not a skilled physician, and was suffering from, say, dysentery, in an out of the way station in Africa or elsewhere, I venture to think he would not speak quite so scornfully of the missionary with the medicine chest, if he found that ipecac., opium, some bismuth and a tonic were available to restore him to health, even if given somewhat according to rule of thumb, and not in a strictly scientific method.

I have never heard of any harm having been done, and I am sure many lives have been saved as the result of the possession of a carefully fitted up medicine chest in the hands of a man who knew how far he could safely go.

I should like to add that I think THE JOURNAL OF TROPICAL MEDICINE is supplying a most definite need in medical literature.

I am, yours faithfully,
M.D.

Woking,
December 26, 1898.

Communications, Letters, &c., have been received from:—

B.—Major E. H. Brown, I.M.S. (Purnea); Dr. J. A. Blayney (British New Guinea).

C.—Dr. D. Kerr Cross (Blantyre); Dr. John Cross (Amoy).

H.—Dr. D. T. Hoskyn (Yokohama); Dr. D. Hardie (Brisbane).

M.—Dr. G. E. Murray (Johannesburg).

P.—Dr. Wordsworth Poole (E. Africa).

S.—Dr. H. J. Strachan (Lagos); Dr. J. Sugrue, R.N. (Gibraltar).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Pacific Medical Journal.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical Brief.
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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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DYSENTERY AS A FACTOR IN LIVER ABSCESS —THE OTHER SIDE OF THE QUESTION.

By Surgeon-Captain W. J. Buchanan, B.A., M.B., I.M.S.
Superintendent, Central Jail, Dacca, Bengal.

In the whole range of the ætiology of tropical disease there is perhaps no one factor which has been more readily assumed as true than the connection between dysentery and abscess of the liver.[1] It is therefore somewhat presumptuous in one to attempt to throw doubt upon a connection which has assumed in many minds the position of a medical axiom.

Dr. A. Davidson (*apud* "C. Allbutt's System") says, "the doctrine of simple coincidence is inadmissible." But what I venture to put forward is, that the question has been too much studied from the liver abscess point of view and too little from the dysentery side, and that, if one can say so, the coincidence is all on the one side.

There are a few points which, it seems to me, have been too little attended to. (1) The question of race, European or native, (2) the commonness of dysentery and comparative rarity of liver abscess, (3) the meaning of the expression "associated with dysentery." I am well aware that the two former points have not escaped the attention of recent writers such as Manson and Davidson. I, however, propose to approach the question on the dysentery side, and to show that in large bodies of men the carefully-recorded statistics of many years show how very common a disease dysentery is, and how comparatively rare liver abscess[2] is. I may premise that I am aware that what we call "dysentery" is not a single disease, nor caused apparently by the action of a single parasite, but by the combined action probably of several varieties, and most decidedly I must protest against the assumption so often made by French and American writers that "tropical dysentery" is synonymous with "amœbic dysentery," meaning by "amœbic dysentery" the form as described by Lafleur in "C. Allbutt's System."

This statement is as far from the actual facts as that of Chauffart which Davidson has already protested against, viz.: "the more frequent, grave and persistent dysentery is, the more frequent, grave and persistent is liver abscess." This is far too sweeping a statement, and is directly opposed to the facts. (*Vide* below.)

Let us first establish how common a disease dysentery is in tropical countries. Although all the readers of this Journal are aware of the fact, yet we may emphasise their belief by some statistics. Open, for example, Table LIII. in any recent Report of the Sanitary Commissioner to the Government of India. I take that for 1896 being latest to hand. A glance at the columns shows at once the high place taken by dysentery, and the low position of affections of the liver in comparison. In the European Army in this year we find only 101 cases of liver abscess compared with 1,841 cases of dysentery; and the contrast between the two diseases is enormously greater when their relative prevalence in the native army and among prisoners is considered.

As may be seen from these Reports, the death-rate in the European army of India from liver abscess varies, being from twelve to seventy times greater than that of the native army from this disease. Over and over again in the Sanitary Commissioners' Reports (India) it has been pointed out how very much more liable European soldiers are to liver abscess than are either native troops or native convicts. In the 1893 Report it is written: "At present it would appear that the two armies, European and native, suffer about equally from dysentery, but there is a great difference in their liability to liver abscess."

Again, the hepatic curve and the dysentery curve are not parallel, even among Europeans. The very decided rise in the dysentery curve for the troops during the Afghan War was not accompanied nor followed by any rise in the hepatic curve, and the same want of parallelism is apparent if the incidence of the two diseases is studied either by geographical groups or by stations. The following table shows the relative liability:—

Most Liable to Dysentery.	Most Liable to Liver Abscess.
(1) Prisoners (native).	(1) European troops.
(2) Native troops.	(2) Native troops.
(3) European troops.	(3) Prisoners (native).

That is, *those which suffer most from dysentery suffer least from liver abscess and vice versa.*

[1] This article does not deal with the whole question of the causation of liver abscess, but only with dysentery as an alleged factor.

[2] The figures, moreover, include every kind of liver abscess, "tropical," pyæmic, enteric, and possibly even hydatid, &c.

The following table,[1] quoted in the Sanitary Commissioner's Report for 1894, gives the percentage of liver abscesses found at autopsies, *i.e.*, in fatal cases of dysentery :—

Country.	Dysentery Autopsies.	Abscess of liver found.	Percentage.
India	1684 ...	364 ...	22
Cochin China ...	160 ...	21 ...	13
Algeria	1001 ...	180 ...	18
West Indies and Senegambia ...	745 ...	183 ..	25
Egypt	11 ...	4 ...	36
Chili	70 ...	21 ...	30
United States ..	9 ...	6 ...	67

These figures are in some instances too small to be of any value. The mixing up of Senegambia with the West Indies is unfortunate in view of the reputed rarity of liver abscess in the latter islands. It is to be noted that this only refers to fatal cases of dysentery, the countless non-fatal cases which presumably were not accompanied with liver abscess are entirely overlooked. Besides, even if 25 or 30 per cent. of liver abscess cases have a "history of dysentery," then some 70 per cent. have no such history. The following figures, compiled from Sanitary Commissioners' Reports for five years (1892-6), show the enormous number of cases of dysentery compared with the few cases of liver abscess, and will give an idea of the strength of the negative evidence in this matter.

In four years (1893-6) there were 7,972 cases of dysentery in the European army in India and 441 cases of liver abscess; that is one case of liver abscess admitted to hospital for every 18 cases of dysentery. The figures for natives show an immensely greater difference. In 5 years (1892-6) in the native army of India there were 30,020 cases of dysentery, and only 54 cases of liver abscess; among the prisoners in all the gaols of India there were, during the same period, 49,723 cases of dysentery,[2] but only 73 cases of liver abscess, or to combine the figures for natives, there were 79,743 cases of dysentery admitted and only 127 cases of liver abscess; that is, *only one case of liver abscess among natives for every* 628 *cases of dysentery—i.e.*, how common one alleged cause (dysentery), how comparatively rare the alleged result (abscess!)—To parody Falstaff's bill, "only a ha'porth of liver abscess for an intolerable deal of dysentery." This does not look as if dysentery was an important factor in the liver abscesses of natives. One could easily show a much larger number of cases of, say, tubercle of the lungs ("associated with dysentery") in 628 cases of dysentery! Hence it will be clear that any theory of liver abscess founded on the co-existence or a history of dysentery is to be received with caution.

It may, however, be argued that it is only the amœbic form of dysentery that is followed by liver abscess. This is a more difficult position to attack, because so little, after all, is known of the relative prevalence of this form of dysentery—if it really is a form by itself and not a clinical picture built up upon the history of a few cases where liver abscess and dysentery ran concurrently in the same patients, for it seems to me that (*apart* from the existence of liver abscess and its special symptoms) the clinical history of "amœbic dysentery" as recently described by Lafleur (*apud* "C. Allbutt's System") is very similar, indeed, almost identical with a very familiar form of chronic relapsing dysentery well known in every Bengal jail and dispensary.

Moreover, the tendency of the day seems to run against the amœba, and evidence is daily accumulating which shows that other and more redoubtable microbes are responsible for dysentery. Again, no one can say that in all the cases of liver abscess where there has been a "history of dysentery" this has been of the "amœbic" form; indeed, recorded histories often point to very mild attacks of catarrhal dysentery only. These have, nevertheless, been accepted as evidence in favour of the dysentery theory as having equal value with the severer ulcerative forms of that Protean disease. If, therefore, "amœbic" dysentery is invoked, the statistics on which supporters of this hypothesis have hitherto relied must be re-calculated, and the onus of proving a case of dysentery connected with liver abscess to be or to have been "amœbic" must rest on the supporters of that theory.[1]

Allusion has just been incidentally made to the value of the evidence (in any given case of liver abscess) of a previous dysentery. What does the expression "associated with dysentery" mean? It appears to mean anything from a severe attack of dysentery immediately preceding the formation of a liver abscess to a vague history of what the patient remembers as having been called dysentery, or it may mean signs found *post mortem* of past or present dysentery, or indeed, possibly signs found *post mortem* of any other ulceration of the great intestine. It is obvious that these histories have very different values. A clear and distinct history of a recent attack of dysentery (as recorded, for example, on a soldier's medical history sheet) is evidence of value, but can anyone say that even good proof of an attack of dysentery *several years previously* is of equal importance?[2] And even granting a clear history of dysentery, we seldom have evidence of the nature of that attack; it may have been purely catarrhal and cured by a dose of castor oil, or it may have been a very severe acute

[1] *Zeitschrift für Hygiene*, 1894, vol. xvi., p. 122. Kruse and Pasquale.

[2] The case-death-rate from dysentery in these 49,723 was only 6 per cent. This may be taken as a fair average mortality for dysentery promptly treated, but it varies greatly according to type, *e.g.*, in the jails of the Central Provinces, India, in the famine year of 1897 the case-death-rate was as high as 29 per cent., or more than four times the average rate. The case-death-rate for five years from dysentery among European troops is only 2·4 per cent., or less than half as much as the native rate. Certainly the European rate is reduced by invaliding, but granting this, and even doubling the percentage for this reason, we get a rate under 5 per cent., or *lower than the native rate*. This shows that no argument can be drawn to show a severer type of dysentery in Europeans; on the contrary, the type in natives appears to be the more severe (yet it is very rarely connected with liver abscess).

[1] Moreover, the very low case-death-rate from dysentery among Europeans in India proves the mildness of the type of dysentery among them (*v.* note *supra*), therefore not amœbic.

[2] This assumes an equally long and for some time apparently harmless latency of the dysentery microbe—a point that has never been explained.—W. J. B.

attack with subsequent relapses. Dr. Manson, indeed, admits this ("Tropical Diseases," p. 365). I cannot believe even if the patient "forgets to mention" (Manson, p. 345) a previous dysentery, that any surgeon, with present views on the ætiology of liver abscess in his mind, will ever "forget" to ask him that question.

It will now be agreed that the evidence connecting liver abscess with previous or present dysentery (upon which the theory of a dysenteric origin is to a great extent based) is of very unequal value, yet it is upon statistics of this kind that the connection between the two diseases has been assumed.

We need not delay over the cases in which a dysenteric attack has taken place during the course of a liver abscess illness, or those cases where the dysentery is "latent" or almost microscopic. Such cases suggest a wish to bend circumstances to theory, or so much the worse for the circumstances!

Having now shown the immensely greater prevalence of dysentery over liver abscess in India,[1] it is not necessary for readers of this Journal to do more than mention that the same facts apply to other tropical countries, nor need we delay over the fact that liver abscess is very rare as a result of the dysentery of temperate climates. Neither in the dysentery of the Irish famine nor in the several recorded outbreaks in asylums and prisons in England was liver abscess at all a frequent complication.

In all the dysentery of the Crimean War there was only one case of liver abscess. Nor need we do more than refer to the fact that in several tropical countries (*v.* Davidson's article) where dysentery is very common liver abscess is almost unknown.[2] The case of the West Indies is the most important. I can find little or no account of this alleged rarity beyond a repetition of the alleged fact. I hope some of the colonial readers of this Journal will be able to verify or refute this alleged rarity of liver abscess in the West Indies.

If, however, such factors as tropical heat, dysentery, want of exercise, alcohol, &c., are factors in India, they should produce the same effect in the West Indies. Again, if we believe the "hepatic insufficiency" theory so ably put forth by Davidson, it should be as active in the West as in the East Indies, and should attack new arrivals chiefly; but though it is said by Manson (*op. cit.*, p. 349) that 40 per cent. of cases of liver abscess occur in the first three years of residence, this does not accord with my own experience, nor with the statements of the Sanitary Commissioner, India, who shows (Report for 1894, p. 38) that liability to liver abscess increases with age, and that, as Bryden long ago pointed out, it is "a disease of degeneration." "As the number of men in our short service army in India under twenty-five years of age has increased, the ratio of hepatitis has decreased." This statement seems to clash with the "hepatic insufficiency" theory, but it *may* be said that the decrease of liver abscess is to some extent due to the spread of temperance[1] in the army. It has become apparent, therefore, that in spite of the thousands of pages written on the origin of liver abscess, we are yet far from a complete explanation. The theory of a dysenteric origin appears so plausible (when the lesser liability of native races is not sufficiently insisted upon) that it is possible that it has turned attention in a somewhat wrong direction. It is obvious that it is only in a very small minority of cases of dysentery that we find hepatic abscess a complication or a sequel. Thousands of dysentery cases recover or die without a single symptom pointing to the liver. It is in the hopes of getting the question looked at from this point of view that I have collected the negative evidence detailed above. I have no alternative theory to offer. Looking at the commonness of dysentery in natives and the rarity of liver abscess, it seems as if something else (*nescio quid*) was wanting which causes liver abscess in Europeans in India, and much more rarely in the West Indies, and this something unknown appears to me to be of far greater importance than a mere history of a previous attack of a common disease like dysentery.

[1] Also in India, European women and children suffer as much from dysentery as European males, but in a far less degree from hepatic abscess.

[2] *E.g.*, in the Spanish troops in the Philippines (*Lancet*, July, 1897, p. 48.)

[1] Temperance in all things should *à priori* diminish the chances of anyone getting liver abscess, but it would have no influence on his chance of escaping dysentery. "Sober and temperate Europeans in India are by no means exempt from liver abscess." (S. C. "Report" for 1896, p. 5).

ULCERATING GRANULOMA OF THE PUDENDA.

By K. MacLeod, M.D., LL.D.

Colonel, Indian Medical Service (Retired), Professor of Clinical and Military Medicine, Netley.

The descriptions which have been published from time to time of a lupoid disease affecting the male and female genitals under the designation of "ulcerating granuloma of the pudenda," have recalled to my recollection several examples of the same or a similar condition which came under my observation in Calcutta. I find that in the year 1881 I made short notes of two well-marked cases, and published them in the *Indian Medical Gazette* for 1882 (p. 121.) The following is the summary of these two cases, which I prepared from the more detailed descriptions contained in the hospital case book. They were included in a list of scrotal tumour operations performed in the Medical College Hospital during the year 1881.

(1) "Hindu male, aged 30. Penis, scrotum and pubis covered with a large serpiginous ulcer of six years' duration, which commenced with a soft chancre. Scrotum enlarged (elephantoid). Ulcer dissected off and thickened scrotum removed; penis dissected out of cicatricial mass; flaps taken from thigh and stitched over testes, which were also stitched together and to surface of perinæum. Operation performed with antiseptic precautions; flaps united, and wound, which remained aseptic, healed kindly. Cicatrisation of penis very tedious. Discharged in one hundred and twenty-three days.

(2) "Hindu male, aged 25. A large serpiginous ulcer

on scrotum, pubis and groins following chancre and bubo contracted four years ago; penis completely embedded in cicatricial mass; scrotum much thickened; edges of ulcer papillomatous. Ulcer dissected off, including the tuberculated edge, except from right groin; penis freed and thickened scrotum removed; flap taken from left thigh; testes stitched as usual. Repair of the large wound very tedious. After a stay in hospital of two hundred and twenty-five days he left with a sound cicatrix. Mass removed weighed 11½ oz."

In some remarks on the scrotal tumour operations of the year I made the following comment:—

"These cases supply an illustration of a disease which is occasionally met with in India, namely, serpiginous or lupoid ulceration of the genitals. The process commences with a venereal sore (chancroid); from this as a centre or starting point, or from a bubo resulting from it, ulceration gradually and very slowly spreads. The skin becomes tuberculated, the neoplasm breaks down and a circle of ulcer results, which is succeeded by an imperfect cicatrix. The scrotal and penile skin is destroyed, the genital organs get involved in a mass of cicatricial material, and the ulceration spreads by circles and bays into the groins, thighs and buttocks. Excision is the most easy and effective treatment, but scraping, and the thorough use of strong caustics are also sufficient to extirpate the disease. In the cases on record the penis and testes were dissected out of the cicatricial mass, and treated on the same principles as after amputation of a scrotal tumour."

I recollect seeing and treating several other cases of this disease, and, if I mistake not, my colleague, Dr. D. O'C. Raye, treated some in his wards; but regarding these I can supply no information.

I have no doubt, now that attention has been drawn to the subject, that numerous instances will be forthcoming from East and West Indies, and probably other tropical localities, and that light will be thrown upon the questions of causation and pathology, which are at present very dark. It appears to me that the condition which I described as serpiginous ulceration of the genitals in 1882 is the same as that which has been recorded by the observers referred to in the note written by Dr. Patrick Manson in your issue for January. The situation of the lesion is the same, and so are its characters—its long duration and slow progress; the peripheral ulceration and central cicatrisation; the serpiginous nature of the former and instability of the latter; the infiltrated or granulomatous ring outside of the ulcerating margin; the absence of signs of constitutional infection; the progressive invasion of sound tissue by a process of infection by continuity; the occasional appearance of fresh foci by contact infection or lymphatic conveyance; and the incurability of the circumferential infiltrative and destructive disease otherwise than by extirpation. These peculiarities establish a close alliance between this disease and tubercular lupus and Delhi boil (so-called oriental sore or lupus endemicus) and indicate a microbic pathogeny. It is from this point of view that future investigation will no doubt be pursued. As regards the supposed venereal origin of the disease several questions arise. Is it a tertiary syphilitic lesion? Is it a consequence of chancroidal infection, the more destructive early character of the process being mitigated by gradual attenuation of the virus or enhanced resistance of the tissues? Is it a different infection from either syphilitic or chancroid which has obtained entrance through a venereal sore or through some abrasion or breach of surface in coitus? The location of the lesion points to communication by venereal agency, but the incurability of the disease by medicines or medicinal applications and absence of signs of constitutional infection negative the syphilitic theory, and the chancroidal lesion as it is usually seen is different in respect of nature and termination from this. I am inclined to think that an affirmation to the third question is the probable solution of the problem of causation and pathology. In the absence of specific information, however, speculation is unprofitable, and it must be remembered that the statements of patients regarding the origin of the thing must be accepted with considerable reservation.

On the subject of treatment the position is firmer. Extirpation of the circumferential thickening seems to be the only means of cure. I have seen extensive and fatal mutilation caused by this disease. I have tried cautery and caustics, but they are unsatisfactory because they destroy more depth of tissue than is desirable, the lesion being cutaneous and skin more resistant; they are apt to do too much or too little; they leave the central unstable cicatrix uncured, and the distortions and disabilities of the organs implicated by the disease unremedied. On the other hand, excision by knife can be conducted thoroughly and precisely; cicatricial thickening can be dissected off as well as actively morbid tissue; the penis and testes can be liberated from the distortions caused by cicatricial contraction and binding, and plastic measures can be resorted to to cover the denuded surfaces by flaps transplanted from the neighbourhood, and so hasten repair and restore function. The cases which I have cited illustrate these points satisfactorily.

THE FILARIÆ OF THE EYE.

By M. T. Yarr, Major R.A.M.C.

Fellow Medical Society of London; Member Ophthalmological Society; Chief Assistant Royal Ophthalmic Hospital, Moorfields.

Filaria Loa.

Of all the parasites which have been found in the human eye and its *adnexa*, undoubtedly the most interesting to students of tropical medicine is the filaria loa, owing to its being peculiar to a tropical country and to the interesting problems in connection with its life history which still await solution.

Filaria loa owes its name to M. Guyot, a French surgeon, who (in 1778) saw several cases amongst the natives of Angola, in West Africa, and utilised the native word "loa" (worm) in its nomenclature.[1] Prior to this, cases had been described by Mougin, who called it a "blood-worm,"[2] and Bajour, who believed it to be a dracunculus.[3] The first record of

the worm, however, is a pictorial one and shews that it was known at least 300 years ago: Dr. Manson has in his possession a woodcut, reproduced from a quaint old book published in 1598,[4] which portrays in realistic fashion a man drawing a guinea-worm from his leg and a woman undergoing a rude operation for the removal of a filaria loa from her eye. This picture is of such interest that I hope the editors of the JOURNAL may be induced to reproduce it in a future number, as it seems quite worthy of a place in their gallery of illustrations of tropical diseases.

Since Guyot's paper appeared not more than a dozen cases have been recorded. The literature of the subject is unaccountably scanty when we consider the length of time that has elapsed since the filaria was discovered and the fact that it is so common on parts of the West Coast of Africa that the natives are able to diagnose its presence in the eye.

The endemic area of loa filariasis appears to be that part of the West Coast of Africa extending from about 5° above the equator to 15° below it: it is exceedingly common in Old Calabar, Gaboon and Angola and affects white residents as well as natives. How far inland the disease extends is unknown: in the most recent case recorded[5] the patient, a white man, became infected at Talagonga on the Goe, 120 miles from the sea. Miss Kingsley, the well-known lady explorer, states that nearly every one on the Ogowe River near Gaboon suffers from these worms.[6]

Loa filariasis can only be contracted in this epidemic area: cases recognised elsewhere have been in natives of this part of the West Coast or whites who had resided there for variable periods. That the parasitism is of long duration is shewn by the fact that it was not uncommon amongst American negroes before the abolition of slavery; the slaves were brought to America in sailing vessels which took weeks and even months to make the voyage. Roulin saw a negress suffering from it who had been six years in America,[7] and cases have been recorded of individuals who had been ten years away from the endemic area still harbouring the worm.

"The male worm measures about 25 to 30 mm. in length by 0·3 mm. in breadth, the female 30 to 40 mm. in length by 0·5 mm. in breadth. Both sexes are filiform, cylindrical, colourless, like fine fishing gut, tapering slightly at the head, more decidedly at the tail. The body is dotted over with minute chitinous bosses. The anus is subterminal. The tail of the male is markedly incurvated and provided with five large papillæ on each side of the anus, and two rather short, unequal spicules. The mouth is simple, punctiform, and without armature" (Manson, "Manual of Tropical Diseases.") M. Guyon[8] described a filaria loa 15 cm. long, extracted by a French naval surgeon from a Gaboon negro, but this was probably a "filaria inermis." Only two cases are on record (by Argyll Robertson and Bernard) of the removal of an intact male filarias loa from the eye.[9]

The parasite is generally found in the bulbar subconjunctival tissue or beneath the skin of the eye-lids, but has also been seen and felt in the subcutaneous tissue of other parts of the body, arms, hands, scalp, thorax, &c. It wriggles about with surprising swiftness, wandering from the eye-lid to the eye-ball and thence disappearing into the orbit within the space of a few minutes. Beyond a very annoying tickling and slight swelling, it seems to produce no ill effects *per se*; the lachrymation and inflammatory symptoms seen are due to the scratching provoked by the violent itching. The natives of the West Coast say that it manifests its presence for two or three days consecutively, and then disappears, to re-appear again in a fortnight; after ten to twenty years it disappears altogether. In this country it only appears in hot weather or in very warm rooms; in Dr. Argyll Robertson's case, the patient, a lady, invalided home from Old Calabar with dysentery and fever, was confined to her room for a lengthened period, and during this time the worm was almost constantly moving about in the left eye; when she became well enough to go out of doors the filaria ceased its visits and only re-appeared at long intervals when she sat near a fire. In one case, at least, the filaria has been seen passing from one eye to the other by wriggling beneath the skin of the nose.

In all probability one individual harbours several worms, and sometimes one, sometimes another, comes to the surface.

It is not so easy to fix and extract the worm as one might fancy; it moves about so quickly that, as a rule, many attempts have to be made before success is attained, and even then it is generally crushed or broken in withdrawal. In Argyll Robertson's patient it was seized on the sclerotic by fixation forceps, and withdrawn through a conjunctival incision; in Bernard's, after several unsuccessful efforts, it was removed from under the skin of the upper eye-lid in a similar manner.

Nothing is known for certain as to the life history of the parasite; its means of ingress and egress, and intermediate host (if any), are still matters of pure conjecture. Manson thinks it possible that filaria diurna is the embryonic form of filaria loa, as the parasites coexisted in one of his cases. He has, however, lately examined the blood in another case of loa filariasis without finding any hæmatozoal embryos, and examination of the blood in Argyll Robertson's case gave a similar negative result. In Bernard's case, unfortunately, the blood was not examined. Undoubtedly the embryos of the female filaria loa closely resemble filaria diurna, and analogy suggests that they circulate in the blood and are taken from it by some suctorial insect acting as intermediate host. It is to be hoped that our *confrères* in West Africa may take up the investigation, and bring to light the method of development and propagation of this parasite.

Filaria Oculi Humani.

Filaria oculi humani is rare, or at all events, only a very few cases have been recorded. It is by no means certain that the filariæ which have been found in the aqueous, lens, and vitreous belong to the same species, but, in the present state of our knowledge, it is convenient for descriptive purposes to assume that they do, and to call this species by the old name, *filaria oculi humani*. To those interested in the filariæ of the blood this parasite is of special con-

cern, in view of its possible relationship to filaria Demarquaii. Dr. Manson, in the course of a recent conversation on the tropical entozoa of the eye, told me he thought it quite possible that the filaria oculi humani seen by West Indian observers (and by Barkan, of San Francisco) is the parental form of filaria Demarquaii, the blood parasite recently described by him as common in the West Indian islands, British Guiana, and New Guinea. This is an expression of opinion on which I am not qualified to pronounce, but I think it right to reproduce it here, as Dr. Manson's conjectures of to-day, founded as they are on an intimate acquaintance with medical zoology and prolonged experience of tropical practice, have a happy knack of becoming the proved facts of to-morrow.

Filaria oculi humani has been seen in the aqueous, lens, vitreous, and beneath the retina. I have never seen a case myself, and can only give brief abstracts of the few recorded cases.

(*a*) *In the Aqueous Humour.*—Only two cases of filaria in the aqueous humour of man are known, though in certain animals, notably the ox and horse, it is by no means uncommon (f. Abildgaard—f. papilloso Rudolphi). The first case was described by Dr. A. Barkan, of San Francisco.[10] The patient was a native of Adelaide, Australia, whence he had recently come to San Francisco. He had suffered from severe inflammation of the eyes, probably granular ophthalmia, ten years previously, when living amongst the Australian aborigines. In the anterior chamber of the left eye a whitish, thread-like immobile foreign body was seen adherent to the lower part of the iris. Barkan performed a downward iridectomy, and removed the foreign body together with the portion of iris to which it was adherent. Professor Knapp subsequently made a microscopical examination of the worm, and pronounced it a filaria medinensis (?).

The second case occurred in Havana. Dr. E. Lopez[11] saw a parasite in the anterior chamber of the eye of a seamstress, a white woman aged 61, which had been there, judging from the narrative, from August, 1890, to the following February, without producing any more serious lesion than slight cloudiness of the cornea and mild iritis. The parasite, apparently a nematode, is described as having been white, 0·025 m. in length, slender as a fine thread, slightly thicker at one end than at the other, and very active. At a subsequent examination it was found to have slipped into the posterior chamber, and only part of it could be seen through the pupil. Instillation of atropine proved fatal to the parasite, and as the patient refused operation nothing more could be ascertained about its structure and zoological characters.

(*b*) *In the Lens.*—Filariæ of the lens have been not infrequently found in the frog and the perch (Von Nordmann), but are very rare in man. Professor Blanchard, in his learned and exhaustive work on Medical Zoology, gives details of the few recorded cases.

In 1831, Von Gräfe sent to Von Nordmann two cataractous lenses removed from an old man: one showed nothing out of the common; in the superficial layers of the other were two fine delicate rings which microscopical examination proved to be dead filariæ, each nearly 2 mm. long, filiform, equally thick everywhere; a digestive tube, mouth and anus could be made out. Again in 1832, Von Nordmann, in examining two cataractous lenses removed from an old woman by Jüngken, found a living filaria, 12 mm. long, in one.[12] In the meantime Von Ammon, of Dresden, sent to Gescheidt for examination another cataract taken from a man of 61. On the inner face of this lens Gescheidt found three filariæ, two 4·5 mm., one 1·5 mm., long; he was unable to make out genitals, but thought the small one a male; one still undulated slightly. These animals were very slender and of the same thickness throughout, save at the head and tail; the former was pointed, the latter slightly dilated and furnished with a short, fine hook; the mouth was small, nearly round, without papillæ; the digestive tube extended without inflection to the tail; beside it was a very fine spiral cylinder. Gescheidt believed these worms to be the same as those described by Von Nordmann.[13]

Since these old observations the only case has been that of Schöler, who, in 1875, showed to the Medical Society of Berlin a woman with a living worm in the lens. Virchow examined the patient carefully and recognised a living nematode, 12 to 15 mm. long, spirally rolled, and in continuous movement.[14]

(*c*) *In the Vitreous.*—In 1858 Quadri[15] was consulted by a woman who complained that she saw a thread constantly moving in front of the right eye. With the ophthalmoscope he saw a living entozoon, 22 mm. long, 0·22 wide, thicker at one extremity than the other, twisting about freely in the vitreous. Delle Chiaji, the helminthologist, confirmed the diagnosis.

Fano[16] in 1868 described a moving body in the vitreous of a child of 12, but it seems probable now that the case was one of persistent hyaloid artery; the same comment applies to the cases published by Mauthner and Ewersbusch.

J. Santos Fernandez[17] has twice met with nematodes in the human vitreous in Cuba; in each case a little animal could be seen to undulate through a central haze; in one case the parasite disappeared and vision became normal.

Filariæ have been found in the vitreous of the dog, hawk, and frog.

(*d*) *Sub-retinal.*—Kahut's case of sub-retinal filaria is, I believe, unique.[18] A man, aged 31, nationality not mentioned, presented himself complaining of defect of vision in the right eye of two months' duration. On examining the fundus a whitish swelling like a pin's head could be seen in the macular region; the rest of the eye was normal. Two months later the swelling had increased to the size of the optic disc, and had a greyish spot in the centre from which a mobile thread-like body protruded into the vitreous. Still later—nearly five months after patient was first seen—pain and photopsies were complained of, and on examination movements in the tumour itself could be detected. Kahut concluded he had to deal with a sub-retinal entozoon making its way into the vitreous, and decided to attempt extraction. He divided the external rectus, rotated the eye forcibly inwards and incised the eye-ball at a point corresponding to the site of the tumour; a gush of vitreous

TINEA IMBRICATA.

The illustration of the epilphytic skin disease, *Tinea Imbricata* (Manson), issued with the JOURNAL OF TROPICAL MEDICINE, February, 1899, will serve to convey a more accurate idea of the disease than can be gained by descriptive writing. The disease is of limited distribution, so local, in fact, that it is, or was, spoken of under the name of Solomon Island, or Tokelau ringworm. The Solomon and Tokelau groups of Pacific Islands are, however, 1,500 miles apart, and although Tinea Imbricata has been traced no further eastward than Tokelau in longitude 170 W., it is met with westward across the Indian Archipelago, as far as Sumatra and Rangoon. In many of these regions the disease appears to be endemic, but it is capable of spreading in the tropical regions north and south of the equator. Turner studied the disease in Samoa where, although not endemic, it spread when introduced with great rapidity, and now is endemic there. Throughout Malaya, in fact, the disease is wide spread, and in the Melanesian Islands of the tropical Pacific it is prevalent. Along the shores of the China Sea also it is not unknown, and cases have been seen along the southern shores of China proper; and in many of the islands from 10 to 75 per cent. of the population is affected. Like many other diseases, as communication is increased, so is the contagion spread and practitioners, in the eastern tropics at all events, have to bear in mind the features of the disease and the possibility of its occurrence in their practice. Given a damp climate and a temperature with a minimum of 70° F., it only requires the introduction of the special fungus to start the disease amongst either Europeans or natives.

The signs and symptoms of the disease are quite characteristic. The concentric pattern of the rings, the thin and flaky scales, the large area of the skin attacked, and the preference for the less hairy parts of the body, serve to distinguish it from common ringworm, psoriasis or ichthyosis. Ring succeeds ring in the development of the disease, each presenting a parallel line to its fellow, until a cluster of scaly rings are formed round one focus of infection. When rings from distinct foci meet, their edges present a scalloped edge at their points of contact. The scales vary in size from minute scaly flakes to ribbons measuring ½ inch by 1 inch or more in length; they are firmly attached at the peripheral or advancing edge but jagged at their free or internal margin. Around the attached edges the skin is of a darker colour, owing to the aggregation of the fungus beneath the epithelium. Any part of the skin, with the exception of that of the scalp and the palms of the hands and the soles of the feet, is liable to be involved in the disease. There is but little itching, nor is there any dermatitis apparent.

The *fungus* resembles the trichophyton—the parasite of common ringworm—but as compared to this well-known fungus it is met with in much greater profusion on the under surface of the epidermal scales, and is further distinguished "by the presence in the interior of the conidia and mycelium, of many brown particles." (Manson in Quain's "Dict. of Med.")

The *treatment* of the disease is fortunately simple; painting with iodine liniment, or on a tender skin with equal parts of the tincture and liniment of iodine, is speedily effective. Chrysophanic acid ointment (20 grs. to the ounce) is usually effective, and may be used when the disease involves only limited areas. If the disease is very widespread over the body, the affected parts must be treated *seriatim* until the whole has been dealt with. As re-infection is possible by means of the clothing, it is necessary that all wearing apparel be boiled.

TINEA IMBRICATA.

STRAITS SETTLEMENTS. From a Photograph by Dr. T. S. KERR, Colonial Medical Service.

Bale & Danielsson, Ltd., London.

followed, carrying with it a little grey lump which on hasty examination with a lens proved to be a rolled-up worm. The wound was sutured, the rectus re-united, and the patient did well, recovering a certain amount of peripheral vision. The drawing of the parasite in Kuhut's paper shows a nematode worm, pointed at one end, blunt at the other, measuring 10 mm. by 0·3 mm. Rudolph Lenkart, who examined it, describes it as "a larval filaria or strongylus, or possibly a scolex embryo."

Filaria Inermis.

This filaria has been found in the horse and ass as well as in man. Only three cases in the human eye are known, although it is possible that the worm described by Guyon as a filaria loa (*vide supra*) belonged to this species; one 115 mm. long, removed from the conjunctiva, is to be seen in the Museum of the Ospedale Maggiore of Milan; another was extracted from under the bulbar conjunctiva of a woman of 60, by Valadà (Blanchard). Angelo Pace, of Palermo, removed one measuring 100 mm. in length by 1·5 mm. in breadth from the eye-lid.[19] In the horse and ass it is frequently confounded with filaria papillosa.

Of the other entozoa of the eye, such as monostoma and distoma lentis, cysticercus, echinococcus, trichina, I do not propose to speak here, as they cannot be regarded as more prevalent in tropical than in temperate climates.

In conclusion, I venture to express the hope that this brief sketch may prove as interesting to others as its subject is to me, and that it may be the means of drawing the attention of our *confrères* abroad to a field of research which could be made as enthralling as any "hobby." I make no pretence of originality, but have confined myself to bringing together a few facts which could only be found hitherto in a scattered —to many inaccessible—form.

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NOTES ON A FEW CASES OF HÆMOGLOBINURIA IN INDIA.

By CHAS. E. BALDWIN SEAL, M.R.C.S., L.R.C.P.

SINCE I came to India in 1891 I have met with six cases of hæmoglobinuria, all of which occurred in patients of European extraction, and in low-lying malarious districts. Five of the cases occurred in a corner of the Darjeeling Terai, and one case in S. Sylbet.

Case I.—The first case occurred in the decline of the rains of 1892, in a patient who had lived for several years in malarious districts, and who had at various times suffered more or less severely from malaria, mostly of an ill-defined, intermittent type; he was a tea planter, aged about 34. The hæmoglobinuria came on suddenly, and not during the course of an attack of malaria, nor had the patient been largely dosed with quinine at any time immediately preceding the attack. The first sign of the attack was a copious discharge of porter-coloured urine; the quantity during the attack was largely increased, but was unfortunately not measured; there was a deposit of a dark grumous material at the bottom of the vessel; the first urine passed was as dark as any, and during the three days he was under my care the colour gradually got lighter and the quantity less. An icteric tint developed within two hours of the onset, and rapidly deepened until patient was intensely jaundiced before the end of the first twenty-four hours. His spleen was slightly enlarged; the liver not at all. The temperature was never above 102° F. He was very restless and would not keep in bed. He was removed to the hills and made a rapid and perfect recovery.

Case II. occurred a few months later in a neighbouring garden in a weakly young man, and was on the whole a milder case. It occurred suddenly. The urine was as high coloured as in Case I., but not nearly so copious; the icterus was not so marked. The spleen and liver were not enlarged. The temperature never ran above 102·5°; the urine cleared very rapidly, the urine was not examined at the time, but about a month after was quite normal.

Case III. was brother to Case II.; he was living on a garden close to the other two cases, and was a slightly built man, about 23 years of age. It was a more severe case than either of the others, lasting longer and producing more prostration. He suffered from a very offensive green diarrhœa—six or seven stools the first day—and the urine retained its dark colour for nearly a week, and cleared gradually. The temperature was never more than 102° F.

The first of these cases was started on small doses of quinine, but as this seemed to increase the restlessness without having any effect on the disorder, it was stopped after the third dose of 5 grains. Gallic acid was then given with seemingly beneficial effects. Cases II. and III. were in the habit of taking small doses of quinine pretty regularly; the first patient was not.

Case IV. was a second attack occurring in Case I. It was characterised by exactly the same phenomena as in that attack, but there was diarrhœa similar to that in Case III. He made a rapid recovery. The

treatment, as also in Cases II. and III., was gallic acid. Cases II. and III. were not removed to the hills; the patients in Cases I. and III. were removed as soon as possible.

Case V. was a recurrence, about nine months later, in the patient Case II. I had seen the patient on the Saturday and examined him carefully, and had found him in excellent health, with his organs, liver and spleen quite normal. He had been taking Fowler's solution for the previous fortnight, and this was stopped. On the Monday evening I was sent for, as he was said to be dying. When I saw him I found he had passed a small quantity of porter-coloured urine (about a pint) in the morning, and since then *nil*. The next day I had him removed up the hill with great difficulty, as he was very restless and troublesome, and kept throwing himself out of the stretcher, and once he succeeded in undressing himself almost completely. Towards evening he passed a small quantity of apparently clear water into the bed, and expressed himself as feeling better, and slept. He suddenly woke up in the night, sat up in bed, and fell back dead. He had had no quinine for a fortnight, nor any attack of fever, and the temperature was below 98° (97·2, 97·6) all through his illness of about sixty hours. There was very slight jaundice; no coma or convulsions.

Case VI. occurred in my own person in the March of 1895, in South Sylbet, after a week of great fatigue and anxiety, when I had had to sit up five nights out of six, and could get no rest during the day. I woke one morning to find some urine which I had passed in the night was deep port-wine coloured, and that I was jaundiced, and feeling wretchedly ill. I passed about six pints of similar urine during the day, and next day it gradually cleared, and the quantity lessened; the icteric tint, however, deepened, and there was constipation. The most distressing symptom was the restlessness, which was intense. My temperature was absolutely normal all through, and I had had no fever nor taken any quinine since the middle of January. The urine was quite normal on the third day, and the jaundice, which was not very marked, disappeared a couple of days later. Treatment, a saline purge.

I was unable to make any chemical examination of the urine or microscopical examination of the blood in any of these cases.

There are several points in common in all these cases:—

(1) They all occurred in low-lying malarious districts.

(2) All the patients had been exposed to malarial poisoning for several years.

(3) The onset was sudden, without any premonitory symptoms.

(4) It did not occur in any of the cases during an attack of malarial fever, either remittent or intermittent, neither did it take the place of a paroxysm.

(5) The temperature was never very high, and in two cases never above normal.

(6) There had been no extensive dosing with quinine before the attack, although all had at various times taken quinine freely.

(7) Five of the cases (three patients) occurred within a few miles of each other, and in two of these patients there was a second attack within a year.

(8) Quinine seemed to increase the severity of the attack.

Cases I. and VI. were moderate drinkers, Cases II. and III. strict teetotallers.

The predisposing causes seem to be residence in a particular part of a malarious district, with, in my own case, intense fatigue superadded. Quinine in these cases could be hardly looked on as an important factor in the causation.

There is a considerable difference, clinically, between my cases and Dr. Powell's; in all of his cases there seems to have been rigors and high temperatures followed by sweating, which phenomena occurred in none of mine. All of his cases, too, seem to have been immediately preceded by very definite malarial phenomena.

Jaundice occurred in all my cases but was very irregular in its intensity. Case I. was very deeply jaundiced in both his attacks, the others less so. The condition of the bowels was variable.

Treatment seems not to count for much, quinine seemingly being contra-indicated. The only medicines I gave to any of my cases was gallic acid, with a saline purge where constipation existed.

COMPOUND TINCTURE OF BENZOINE, AND HORSE-HAIR SUTURES IN MINOR SURGERY.

By R. C. Bennett, M.B., C.M.
District Medical Officer, Trinidad, W.I.

The use of compound tincture of benzoine, or Friars' balsam, in certain cases of injury caused by crushing and laceration of soft parts is, I think, worthy of a few notes.

The class of cases in which I have used this exceptionally efficient aseptic for some years are just those cases in minor surgery which, though apparently trivial, cause trouble and sometimes anxiety—tetanus not infrequently resulting.

I refer chiefly to injuries of the fingers, toes, &c., causing tearing and laceration of soft parts, with or without fractures of small bones and injuries to joints, &c. In some of these cases immediate amputation might be justifiable, but I venture to think that many fingers could be saved by treatment with Friars' balsam. The mode of application I pursue is as follows:—Thoroughly examine the wound and make certain that no foreign matter, such as spiculæ of bone, gravel or other *débris* lie impacted, as the non-removal of one or more of these might be fatal to this form of treatment. Clip away loose shreds of muscle, skin or other soft tissue, wash thoroughly in 1 in 40 solution of carbolic acid, and arrest all bleeding or oozing. *These precautions are necessary.* This being done, bind the wounded parts with strips of lint 4 to 6 inches long *saturated* in compound tincture of benzoine; around this a pad of antiseptic wool is applied which is kept in position by gauze bandages carefully applied. The objections to the Friars' balsam are: (*a*) some smarting on its application; (*b*) the difficulty, if it can be called such, of removing the dressing owing to hardening.

The advantages are: (1) a thoroughly efficient aseptic dressing which does not require to be removed or in any way interfered with, and not necessitating the daily attendance of the patient, and application of frequent and fresh dressings. This is the chief advantage of this form of dressing when applied in suitable cases, and in large outlying tropical districts it is of importance. In most cases, if symptoms do not contra-indicate it, no second dressings are required, and I have not known of a single case, even when urged by an anxious patient to remove the dressings, where the wound was not healing and *perfectly sweet*. (2) Wounds that generally, if not always, suppurate, seem to heal up readily. (3) In compound fractures of fingers not only is an excellent dressing obtained, but the hardening which occurs forms a fair support and acts as a splint. (4) The saving of many fingers that one might be tempted to amputate, and the saving of time, trouble and pain to the patient. (5) The thoroughly reliable antiseptic character of the dressing.

The following are four cases quite recently treated with Friars' balsam :—

Case A.—October 8. Index finger severed, hanging by shred of skin. With sharp scissors skin cut through, and part detached. Dressed with Friars' balsam. October 17. Dressing removed for the first time, and reluctantly, as the girl requested it owing to pain, which was doubtful. Wound clean, healthy and *sweet;* healing well. Friars' balsam again applied. Did well with two dressings.

Case B.—Child, 7 years old, lacerated wound of forehead. Horse-hair suture and Friars' balsam applied, October 11. Dressing removed for first time, October 17. Wound quite healed. Suture removed. Discharged well. No other dressing applied.

Case C.—Lacerated wound of hand. Horse-hair suture and Friars' balsam, October 11. Patient went away into the woods and did not return until 24th. Dressing removed for first time. Wound perfectly sweet and healing.

Horse-Hair Sutures.

Horse-hair sutures are *par excellence* nature's sutures. When compound tincture of benzoine dressings are to be used as described, and sutures are indicated, *no other kind* should be used. These sutures have natural advantages that can hardly be equalled. They are readily obtained and easily made aseptic, are smooth, and run through tissues readily, are pliable and strong, and if not strong enough can be doubled and redoubled. They *never* cause any irritation, and can be left in a wound long after it has healed. An important advantage it has over most, if not all sutures, is that it *never* causes or leaves marks, and this in wounds or delicate operations about the face is a most important factor.

Plague Cultures.—The *Bombay Gazette* expresses the hope that the carrying of plague cultures from an infected area will shortly be made a criminal offence. The possibilities of a little phial of microbes are incalculable. They might decimate the population of a great city.

THE EXAMINATION OF BLOOD FILMS WITH SPECIAL REFERENCE TO WORK ON MALARIA.

By Robert Howard, M.A., B.M., B.Ch.Oxon.

Medical Officer, Universities Mission to Central Africa (Nyassa Diocese),

and

Walter C. C. Pakes, D.P.H.Camb.

Professor of Hygiene in the Bedford College for Women; Demonstrator of Sanitary Science and Bacteriology at Guy's Hospital.

(From the Bacteriological Laboratory, Guy's Hospital.)

The accounts given in various text books and monographs of the preparation of blood films for microscopic examination are by no means satisfactory. All err in one important respect, viz., they lack detail. Many writers after going so far as to recommend a particular method add a note to the effect that the results obtained are not constant.

Some time ago we had convinced ourselves of the fact that, however excellent the results might have been in the hands of the authors of the various methods, we were certainly unable to obtain satisfactory and constant results, and this in spite of our endeavours to carry out the procedure exactly in accordance with the directions. We therefore set to work to try all the various methods, and also modifications of them, in order to see whether by paying particular attention to detail we could obtain constant results. This we claim to have done, and after giving an account of our work we shall summarise our results and point out the details which are necessary to ensure success.

The blood may be examined either in the fresh state or in stained preparations. The former is undoubtedly the most satisfactory method for diagnosing malaria, the latter for examining in such conditions as leucocythæmia.

The blood is generally collected from the lobe of the ear or from the back of the distal phalanx of a finger. Each of these places has its advantages. The lobe of the ear is certainly the most convenient when several large drops are required, as for Widal's reaction, and for the bacteriological examination of the blood, because the ear is more easily rendered aseptic than the finger. It is also an advantage when nervous people are being dealt with, as they cannot see what is being done. The painlessness of the prick is a great advantage when children are the subjects. The finger, on the other hand, is more convenient for the operator, and if a really sharp instrument is used the pain is very slight.

The skin should be cleaned with soap-and-water or alcohol, and then rubbed dry with a cloth. For bacteriological examination where strict asepsis is necessary it should be washed with lysol (2 per cent.) or carbolic acid (2½ per cent.), and then with alcohol and ether, equal parts, which is allowed to dry off, or else is wiped off with a pad of sterilised cotton wool.

The ease with which the blood is collected depends to a large extent upon the suitability of the instrument used to prick the skin. An ordinary sewing needle is practically useless. Surgical needles are

fairly good provided they are sharp, but it is difficult to obtain many drops of blood from one prick. A lancet is the best instrument, because it is sharp and makes a minute linear incision. It has, however, two disadvantages: firstly, it is not easy or suitable for sterilising; and secondly, many patients, and especially neurotic ones, will raise objections to its use, since they imagine that it cannot be used without cutting. In order to obviate this difficulty one of us (Pakes) has contrived an instrument which has the advantages of the lancet without its disadvantages. A full-sized hare-lip pin is cut off at the broadest part of the lance head, and the end is then sharpened from the point to the shoulder, as in the accompanying diagram.

Fig. 1.

This, then, becomes practically the end of a lancet mounted on a pin. It can be easily sterilised by passing through the flame or boiling, and it does not differ much in appearance from an ordinary long needle.

The coverslips and slides which are to be used must be scrupulously clean. As they are bought, they are by no means clean as the word should be understood when working with blood. They are covered with organic matter, and often with grease from the packer's hands, &c. There are two or three ways of cleaning them. Cabot[1] suggests that both slides and coverslips should be washed well in soap-and-water, then in water, and subsequently dried and polished with a clean handkerchief. This is a very satisfactory method for the slides, but the percentage of breakages in the case of coverslips is apt to be great, especially as the thinnest, *i.e.*, No. 1, have to be used, in order to admit of subsequent examination with an oil immersion lens. The method we adopt for the coverslips, is to drop them one by one into a 10 per cent. solution of chromic acid, contained in an enamelled iron dish, and boil them for twenty minutes. They are then tipped altogether into a shallow basin and washed with ordinary tap-water until all trace of the yellow colour of chromic acid has disappeared. The water is next poured off and the slips are covered with rectified spirit. After this they are washed in absolute alcohol, and transferred with clean forceps to a glass-stoppered, wide-mouthed, shallow bottle containing absolute alcohol. One to two or more ounces may be cleaned at the same time. As they are required for use they are picked out of the bottle singly with forceps; the alcohol is allowed to drain off for a second or two, and the remainder is burnt off by just passing the slip through the flame of a spirit lamp or Bunsen's burner.

We were struck with the fact that when we were preparing fresh specimens of the blood we could not obtain such good preparations with the slips cleaned as above as with those cleaned by Cabot's method.

After trying a few experiments we satisfied ourselves that the mechanical polishing was the great factor in success, and we found that coverslips cleaned as above with chromic acid, and then polished by rubbing with a clean handkerchief for a few seconds, after the alcohol had been burnt off, gave as good results as when cleaned with soap-and-water. As in the method we employ, a large number of coverslips can be cleaned once for all and then kept in absolute alcohol till required, it will be seen that it is less laborious than cleaning them individually each time.

In preparing fresh specimens the following points must be observed. It is best to employ $\frac{3}{4}$ inch square coverslips and the usual slides 3 in. by 1 in. The drop of blood should be of medium size; if it is too large, too much will be taken up by the coverslip; if it is too small the coverslip is likely to touch the skin and pick up dirt and grease. Hence if blood flows freely from the prick the first few large drops must be wiped away, while if the drop is too minute gentle pressure should be used to increase its size. Hold the coverslip (cleaned and polished as above) by two of the diagonally opposite corners between the thumb and middle finger and place the index finger on the anterior corner; it is thus fixed by three points of the square and can be firmly held. Lower the coverslip till its centre just touches the extreme top of the drop of blood and then drop it, blood surface downwards, on to the slide, taking care to keep the surfaces of slip and slide parallel.

Another and perhaps easier method is as follows. Holding the coverslip in forceps, lower it till it touches the drop of blood as above, but then place it on the table, blood surface upwards, and lower the slide on to it. The blood at once begins to spread out and the slip adheres to the slide, thus allowing the latter to be rapidly turned over.

If slide and coverslip are clean no pressure is required to obtain a thin film, and pressure should be avoided because it leads to vacuolation of the red discs and also to the escape of intra-corpuscular parasites.

As soon as the film has spread out it should be ringed round with vaseline or cedar oil to prevent evaporation. For a description of the naked eye and microscopic appearances of fresh blood films we cannot do better than refer the reader to the excellent account of Manson.[2]

When films are to be prepared for staining it is no less necessary that the surface upon which the blood is to be spread must be scrupulously clean, but coverslips which have been cleaned with chromic acid may be taken out of the absolute alcohol and used as soon as the latter has been burnt off; no polishing is necessary in this case. The surface must not be touched with the fingers, as where there is any suspicion of grease the blood may miss the greasy portion and be collected in masses at the edge, or while apparently evenly distributed, on microscopic examination the discs may be found to be collected in little groups interspersed with clear areas.

The method of preparing blood films usually de-

[1] A Guide to the Clinical Examination of the Blood for Diagnostic Purposes, R. C. Cabot, M.D.; Second Edition, Longmans, Green & Co., 1897, p. 7.

Note Fig. 1.—The needle is more pointed than the diagram appears.

[2] "Tropical Diseases," P. Manson, M.D., First Edition, p. 22, &c.

scribed in the text books is known as the "two coverslip" method. A drop of blood is collected on one slip and a second slip is gently dropped upon it so that the two form a star with eight points. When the blood has spread out between these they are separated by drawing one over the other. The chief advantages claimed for this method are its extreme simplicity and that it requires no apparatus. The disadvantages are unfortunately very soon discovered. If too little blood has been collected it is extremely difficult to separate the slips without breaking one or other of them. If too much, the film is too thick and the corpuscles are crenated; even if the right quantity has been collected the majority of the blood is apt to collect at the edges or in ridges. The explanation of this last phenomenon is, we think, that it is very difficult to keep the coverslips parallel when drawing them apart. Fairly good films are only made by good manipulators and then only after long practice. Cabot[3] admits that even in his hands the uppermost of the two films is never so good as the lower one.

Manson[4] has recommended that the films should be made by collecting the blood on the edge of a piece of thin gutta-percha tissue or tissue-paper, and drawing it along the surface of a slide. We have tried this repeatedly, and are convinced that after a very little practice a tyro can obtain very fair specimens, certainly much better than he could hope to obtain with the two coverslip method without endless practice.

There are three objections to using slides. Firstly, they are much more likely to collect dirt and grease. Secondly, they are very much less handy than coverslips during the process of staining. The third and perhaps greatest objection is that being so bulky they are very much more inconvenient to pack, carry about, or transmit through the post. This is a great consideration for any one who is studying, say in Africa or India, and is some distance from the laboratory.

Fair films may be made by applying Manson's method to ordinary-sized coverslips, but it is difficult to gauge the amount of blood requisite to cover so small an area. We therefore tried arranging three or four coverslips in line, and fixing them by means of a specially contrived clip,[5] and then drawing the paper over all four slips at once. We found, however, that the blood tended to run down into the cracks between the slips, and further that the first slip was too thickly and the last too thinly spread. We therefore tried specially long coverslips 1½ in. by ¾ in. On these we found that we could make excellent films, quite as good as on a slide. In fact, as it is only the middle half of a slide that is used, whereas the whole surface of a coverslip can be covered, the size of the films is practically the same. The clip was adapted so as to hold four of the long coverslips securely fixed by their extreme ends, and we have found it a great convenience. If it is not used the end of each slip must be held down with the finger or forceps while the film is being spread.

If a large number of specimens is wanted the coverslips may be scratched with a diamond and broken across, thus giving two ¾ in. square. For diagnostic purposes, however, especially in cases of malaria, the long film is an advantage, as a considerable quantity of blood can be examined in one specimen and it is easy to arrange that the upper end shall be rather thickly spread and the lower quite thinly. By this method we claim to have combined all the advantages of Manson's slide-method with the use of the coverslip.

As regards the tissue used, we have not been successful with gutta-percha tissue and so have discarded it. We have tried various kinds of tissue paper and have finally accepted cigarette paper as the most convenient. There are two small points to be noticed. Firstly, the edge used must be the original machine-cut edge; and secondly, it must be that which is parallel to the ribs. The best papers are the Tarlene, or the Zigzag, not the familiar A. G. papers. Strips are cut across the ribs so that each is about half an inch wide and as long as the original cigarette paper is wide. Should cigarette papers not be at hand, ordinary note paper may be used, but it is not nearly so good.

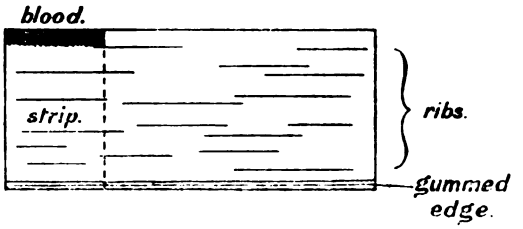

FIG. 2.

In making the film the strip is held between the thumb and first finger and is lowered till under surface adjacent to the machine-cut edge just touches the drop of blood as it rests on the skin, and then by a slight lateral movement the drop is converted into a streak, as in the diagram. This end of the strip is at once laid blood downwards on one end of the coverslip, the other end being still held, and the blood having been allowed to spread out between the paper and the coverslip, the paper is slowly drawn along the slip towards the other end. A fresh strip of paper should be used for each film.

The size of the drop of blood is of importance. With a normal patient a few films are sufficient to enable the observer to gauge the correct amount. When the patient has great diminution in red corpuscles and the blood is correspondingly watery, a larger drop must be taken as if a much thicker film were intended, the increase in size being proportional to the degree of the anæmia. If this point is not attended to the discs will be found to be somewhat widely scattered, and hence the films will be much more tedious to examine.

After the films are made they are allowed to dry in the air. This only takes a few seconds, if they are thin and even. They may then be packed together by placing coverslips and small pieces of clean paper

[3] *Op. cit.*, p. 32. [4] *Op. cit.*, p. 30.

[5] This clip, devised by one of us (W.C.C.P.), consists of a light block of wood on which the slips are held by the pressure of a brass spring. It was exhibited at a Meeting of the Pathological Society on December 20, 1898. It can be obtained from Messrs. Watson & Son, 313, High Holborn. Price 3s.

alternately. During this process the coverslips should be held in forceps, not with the fingers, for until the film is fixed it must not be brought into contact with any moist surface, or the red discs will be destroyed and the blood laked. If kept dry, the films will keep any length of time, but in very damp climates, as in Africa, it might be advisable to fix them before sending them home.

Arrived at the laboratory, the next procedure is to fix the films. The fixing agents we have tried are heat, alcohol, alcohol and ether, corrosive sublimate, osmic acid vapour and formalin.

Heat is perhaps the best fixing agent, as it can be used for every purpose. The optimum conditions are a temperature of 115° C. for two hours. The special advantage is that the granules are preserved better than by any other method. The disadvantages are that it necessitates a regulated hot-air oven, and is not so quick as some other methods. The oven method is no disadvantage in a well equipped laboratory, but few medical men have room or sufficient use for one.

Cabot[6] suggests the following as a substitute for an oven. A strip of copper 3 × 12 in. supported over a flame very soon gets a constant temperature at any given distance from the flame. On this, find the boiling point of water by dropping small drops of water on it, and put the coverglasses at this point face downwards.

A mixture of equal parts of absolute alcohol and ether for half an hour or longer also fixes the films well. Next to heat it is the best for preserving the granules, and it gives the corpuscles a clear, distinct outline.

Immersion in absolute alcohol for from ten minutes to half an hour gives very good fixation as far as red discs and intra-corpuscular parasites are concerned, but it is less good for the granules in the leucocytes. For malaria however, with certain stains, it is excellent.

For work in tropical countries, the two last, being volatile, are somewhat difficult to deal with. The method we have adopted will get over the difficulty to some extent. The alcohol or alcohol-ether is placed in a Petri dish (*i.e.*, a shallow glass dish, with overlapping glass cover, usually termed in the bacteriological laboratory a "plate") about 2½ in. in diameter and covered over with its proper lid. The evaporation and hydration which take place are very small, and we have found that we can effectually fix films in fluids which have been in the Petri dish for two or three days in a warm laboratory. In the tropics it would be better after use to pour the alcohol back into a bottle kept for the purpose.

We thought that it might be possible to replace these volatile fixing re-agents by a saturated aqueous solution of corrosive sublimate. This only requires five miuutes for thorough fixation of the film, but it has the disadvantage that it destroys the granules and limits the number of stains that can be used. Before staining a film fixed in this manner, it must be well washed in water, treated for a few seconds with Gram's iodine solution (*i.e.*, liq. iodi. one part, water fifteen parts), then with alcohol (methylated spirit will do) for two or three minutes; otherwise crystals of corrosive sublimate are left on the film. Should the supply of absolute alcohol run short, corrosive sublimate could always be obtained, and this method may then be used with advantage.

The vapour of osmic acid fixes the films as well as any of the above reagents, but it restricts the stains within very narrow limits.

It may be mentioned by way of caution that in all blood films, but especially after fixation with corrosive sublimate or osmic acid, little groups of blood platelets which take a basic stain may be found. These have often been called sporulating parasites; but if the observer is alive to the danger, a very little practice will prevent any mistake as to their identity.

After the film has been fixed it becomes necessary to stain it. The best known blood stain is the Ehrlich-Biondi triple stain. Many modifications of this have been made, and many are the receipts given in the text books for making them up from the constituent elements. It is simpler and more satisfactory to use the Ehrlich-Biondi-Heidenhain powder which is supplied by Grübler. We have employed this made up according to Cabot's directions,[7] *i.e.*, Ehrlich-Biondi-Heidenhain powder gr. xv. (*i.e.*, one gram approximately), absolute alcohol 1 cc., water 6 cc., and also as recommended on the label by Grübler. Of the two we prefer Cabot's mixture, which, although a very strong solution, is easy and convenient to work. The stain is dropped on to the coverslip with a glass rod or pipette and after five minutes washed off with water. This is generally admitted to be the most satisfactory distinctive stain for the leucocytes and their granules, but it entirely fails to stain the malaria parasite.

Another well-known combination and one which is specially recommended for malaria is that of eosin with methylene blue. It is in the details of this method, however, that the text books are most meagre. One is recommended to make up solutions of certain strengths of "eosin" and methylene blue. Grübler[8] (whose laboratory is the recognised source of pure histological stains) supplies no less than four different eosins. These are (1) aqueous eosin, "yellowish"; (2) aqueous eosin, "bluish"; (3) blood eosin; and (4) alcoholic eosin. Of these, the first three are readily soluble in water or alcohol, the fourth is practically insoluble in water, but fairly soluble in from 60 per cent. to absolute alcohol. All these readily stain the red discs, but they differ in that the first three—whether alcohol or water is used as the solvent—are much more easily removed by subsequent manipulation than the alcoholic stain, which is most persistently retained. There is less difficulty about the methylene blue, since Grübler only supplied one variety of this stain.

Besides the above there is also a number of commercial varieties of both stains, which, however, are unsuitable for histological purposes.

[6] *Op. cit.*, p. 33.

[7] *Op. cit.*, p. 33.

[8] Grübler's stains may be obtained from Messrs. Baird & Tatlock, Cross Street, Hatton Garden, E.C., or from C. Baker, 244, High Holborn, W.C.

Of these two stains eosin is strongly "acid," having a special affinity for hæmoglobin and eosinophile granules; methylene blue is "basic" and stains the nuclei. They are, moreover, direct antagonists, for if either stain is in excess in a mixture, or, when used as a consecutive stain, is allowed to act for a sufficiently long time, it will completely turn out the other. It must be stated, however, that eosin turns out methylene blue much more readily than methylene blue turns out eosin. Recognising this principle of antagonism, various attempts have been made to produce mixtures in which the two stains shall be exactly balanced, with the idea that each will then stain its appropriate element. Among the commonly described mixtures are Plehn's, Romanovsky's, Thayer's modification of Romanovsky's, and Geppener's.[9] Each of these is recommended by its respective author as specially applicable to work on malaria.

However ideal such an arrangement may be, and however satisfactory in skilled hands, there are factors in the manipulation which are not easy to determine and which render these mixtures—as their various authors allow—uncertain. We are quite prepared to admit that when plenty of material is available, and when one is dealing with undoubtedly malarial blood, these mixtures may be useful for studying the various phases of the different parasites, but their uncertainty renders them unsatisfactory, perhaps almost dangerous, for diagnostic purposes.

Our non-success with these mixtures cannot be attributed to the fact that we have used the wrong eosin, for we have tried to make up the mixtures with each of the four kinds. For instance, in endeavouring to make up Plehn's solution, we found that if alcoholic eosin were used it completely precipitated on the addition of the water and the films stained with methylene blue: if we used aqueous eosin our films usually stained only with eosin. That we were alive to the fallacies of the eosin is shown by the fact that we did not abandon this stain until we had made eight different attempts to prepare it. It is possible that this mixture may work well if it is kept for a long time before use. No such suggestion, however, could be found by us in any description.

Similar unsatisfactory results were obtained with the other mixtures, though with these it was sometimes the eosin and at other times the methylene blue which predominated.

Quite apart from the staining powers of these mixtures there remains the objection that, as most of them formed copious precipitates owing to the interaction of the stains, and as this precipitate, according to the instructions usually given, must not be filtered off, the films are apt to be obscured by precipitated stain which cannot be washed off.

For work in the tropics there is the further, and we consider greater objection, that the mixtures will not keep, but must be made up practically every time that they have to be used, and the excess stain is wasted, whereas if single stains are employed they can be poured back and used again. This is no slight consideration when the supply of reagents is limited.

[9] For references see "Lectures on the Malarial Fevers." By W. S. Thayer, M.D. Henry Kimpton. 1898. Pp. 40 *et seq.*

For work with non-malarial blood where the staining of the leucocytes rather than of the red discs is of importance, the mixtures are not attended with such drawbacks, and they undoubtedly may stain the granules very clearly. There is one mixture we have got to give fairly satisfactory results, and this is:—equal parts of one per cent. aqueous solution of aqueous "yellowish" eosin, and saturated aqueous solution of methylene blue, diluted with five volumes of water and filtered. This is a modification of the one suggested by Ramsden (*Guy's Hospital Gazette*, vol. x., p. 149), and is the same as that which Thayer recommends, but diluted and filtered.

Having discarded the mixtures we next endeavoured to find whether any single stains would give satisfactory results. We began with methylene blue, which we knew to be a good basic stain.[10] This stain can be used after any method of fixing, but its effect varies greatly with the solvent. When dissolved in distilled water, or in ordinary tap water, unless the latter is very distinctly alkaline, it acts as a pure basic stain, *i.e.*, the nuclei of the leucocytes and malaria parasites are stained blue, whilst the red discs show distinctly of their natural pale yellow colour. The pale discs, when properly fixed, are quite distinct, and at the same time extremely transparent, so that the intra-corpuscular parasites stand out well. This transparency of the discs may be taken advantage of when the films, either from accident or design, are thick. When pigmented parasites or crescents are being dealt with, and they are few in number, they can be more easily found in thick films stained in this method than in thin double-stained films. This stain should be used in saturated solution. A certain quantity ($2\frac{1}{2}$ grams to each 100 cc. of water, is ample) of the dry stain should be placed in a bottle to be used as the stock bottle, and the bottle filled with water. This should be left for at least a week, and should be shaken up from time to time. After the lapse of this time the supernatant fluid should be filtered into a smaller bottle, while so long as undissolved stain is left at the bottom fresh water may be added to the stock bottle. In order to prevent the growth of moulds, &c., in the solution, 1 cc. of a 10 per cent. solution of formalin in water may be added to each 100 cc. of stain. We have found that this does not in any way affect the staining power. It is impossible to over-stain with this stain, but it is not necessary to stain for more than five minutes.

Several other solutions of methylene blue are commonly prepared with the object of obtaining a more rapid and deeper stain. Among these may be mentioned carbolic methylene blue, Löffler's methylene blue and borax methylene blue.

The two latter stain very rapidly (half to one minute) but the discs are coloured an opaque blue and hence the contrast between discs on the one hand, and leucocytes and parasites on the other is less marked. Carbolic methylene blue causes the discs to become

[10] The method of staining and mounting which we have adopted throughout is as follows:—A little stain is poured into a Petri dish, and the coverslip floated on it film downwards; it is then removed with forceps, washed in tap water, dried between layers of clean filter paper, and then over a flame, and finally mounted in Canada balsam.

granular and vacuolated. For these reasons we cannot recommend them.

Ehrlich's acid hæmatoxylin is another widely used basic stain. If employed for blood films the best methods of fixing are heat or corrosive sublimate. It is of little use in malaria work, because, firstly, it does not stain the parasites at all well; and secondly, it is very apt to precipitate and must be filtered each time it is used. Moreover, the film must be extremely well fixed, otherwise being strongly acid it has a tendency to dissolve the hæmoglobin out of the discs.

At the suggestion of Dr. Alfred McConkey we have tried carbol thionin blue[11] as a malarial stain. He had previously found that it was an excellent contrast stain for blood, staining the discs a pale grey and the nuclei of the leucocytes red. We have found that it will stain after every method of fixation, but after corrosive sublimate the discs are very dark and hence the contrast is less good. It stains the parasites of malaria well, and this is true even of the young forms. To obtain the maximum contrast the films should be left in the stain for three minutes. Its only disadvantage is that it does not stain the granules in the leucocytes, and this cannot be remedied by combining it with eosin. It has a tendency to form a crystalline precipitate and it may be necessary, therefore, to filter the stain from time to time. It may be remarked in passing that it is an excellent stain for showing blood discs in sections of inflamed tissues. In aqueous solution thionin blue only stains the nuclei, leaving the discs quite unstained.

Our next endeavour was to try various consecutive stains and study the conditions necessary for success with them.

As we have before stated Ehrlich's hæmatoxylin does not stain the hæmatozoon of malaria at all well and therefore this, followed by eosin, is only to be recommended for non-malarial blood. If this sequence is used the procedure is as follows:—Fix by heat or corrosive sublimate; stain three minutes in hæmatoxylin; wash well in tap water; and then stain for half a minute with a 1 per cent. solution of any of the aqueous eosins. These two stains have not the antagonistic properties possessed by methylene blue and eosin, and hence fair results are easily obtained.

As above mentioned, eosin turns out methylene blue far more rapidly than methylene blue decolourises eosin. Hence when these two stains are employed in a consecutive method it is always necessary to stain first with eosin and then with methylene blue. In order to obtain uniformly satisfactory results with the methods here described the times laid down must be carefully adhered to.

The various eosins resolve themselves into two groups, of which the three aqueous varieties form one and the alcoholic another. The members of the aqueous group are fairly readily decolourised by an aqueous solution of methylene blue, whereas the alcoholic variety, being almost insoluble in water, resists its action for a long time. Hence the necessary procedure varies considerably according to which kind is used.

In like manner the kind of methylene blue solution is of importance. In fact, saturated aqueous methylene blue is the only one which can be used as a consecutive stain to eosin, since Löffler's and borax methylene blue both tinge the red discs, thus rendering them an opaque purple, which effectually obscures the parasites.

When aqueous eosin is to be used the films may be fixed with heat, alcohol-ether, or alcohol. The two former are best when it is desired to retain and examine the granules in the leucocytes. For malarial parasites alcohol is fully as good and of course simpler. With alcoholic eosin alcohol is rather less satisfactory than heat or alcohol-ether because the ground work has a tendency to take the eosin stain. The alcoholic eosin is the only form of eosin which can be satisfactorily employed after fixing with corrosive sublimate, as the aqueous varieties do not give the discs a sufficiently intense colour. After fixation by osmic acid vapour the red discs cannot be stained with any kind of eosin.

When staining with aqueous eosin we employ a 1 per cent. aqueous solution, to which 10 per cent. of alcohol may be added to make it keep better. The films should be left in this solution for thirty seconds; they should then be washed in water and left in the aqueous methylene blue for not less than three minutes and not more than five minutes. The resulting preparation under the microscope shows the nuclei of the cells and the parasites coloured blue and the discs a pinkish yellow. There is no great difference between the three watery eosins, but we prefer the "yellowish" or "bluish" to the "blood" because they give a more intense colour to the discs. If the films be left much more than five minutes in the methylene blue it will be found that the red discs have been decolourised, and the specimens are hardly to be distinguished from those stained simply with methylene blue.

For alcoholic eosin the procedure is as follows:—Stain thirty seconds in a ·5 per cent. solution in 60 per cent. alcohol (this is practically a saturated solution), wash in water and stain with methylene blue for about thirty minutes. The long time which the methylene blue requires to act depends upon the difficulty with which it replaces the alcoholic eosin in the nuclei of the cells and the parasites. The difference between these preparations and those stained with aqueous eosin lies chiefly in the fact that the discs are distinctly red and more opaque, so that the contrast with the blue is more marked.

A new consecutive method, which combines fixing and staining, has recently been described by Wermel.[12] The film without previous fixing is placed for two minutes in a 5 per cent. solution of aqueous eosin in 50 per cent. alcohol, containing 2 per cent. of formalin. It is then washed and placed for a similar time in aqueous methylene blue also containing 2 per cent. of formalin. We find that it fixes and stains the leucocytes excellently, but the result of the formalin

[11] In Muir & Ritchie's "Bacteriology" the receipt is given as follows:—Thionin blue, 1 gram; 1 in 40 aqueous solution of carbolic acid, 100 cc. It is recommended for staining bacteria (Thionin blue is obtainable from Grübler or his agents.)

[12] *Medizinskoje Obosrenje* (Russ.), May, 1897.

is to make the discs granular and it is therefore totally unsuited for work on malaria.

We have tried several stains in the place of eosin, such as vesuvin, aurantia, saffranin, &c., but we could not find that they possessed any special advantages, whereas they all had the great disadvantage that they would not stain the granules in the leucocytes. Acid fuchsin, though it stained the granules, had the great disadvantages that the depth of colour imparted to the discs varies greatly with the time during which it is allowed to act, and that it was readily turned out by methylene blue.

In drawing our conclusions we do so from the examination of upwards of a thousand stained films, of which more than two hundred were of malarial blood. What we have said about the malarial parasites refers chiefly to the tertian and quartan varieties, for unfortunately we have not been able to obtain many specimens of young malignant parasites. We conclude, however, that since we have succeeded in obtaining good and constant results with quite young tertian and quartan hæmatozoa, we could have obtained equally good results with the malignant ones, and the few specimens which we have examined bears out this conclusion. We regret also that we have been unable up to the present to work out the staining reactions of the flagellate bodies.

We are convinced that by working on properly cleaned coverslips instead of on slides, the resulting specimens are freer from dust and precipitated stains, and thus freer from many of the fallacies against which warning is often given in the text books.

If both fixing reagents and staining fluids are kept as recommended above in Petri dishes, contamination of the films may be almost entirely avoided.

We have felt that the blood platelets which are often seen, especially after fixing with corrosive sublimate, might easily lead to error, and we find that Thayer says that this has actually happened. The advisability of carefully examining normal blood both in fresh films and stained by the various methods advised, cannot be too strongly urged upon all who are not experts in blood work.

The following is a brief summary of the procedure which we recommend :—

(*a*) When blood does not contain malaria parasites;
  (1) Fixing by heat and staining with (*a*) Ehrlich-Biondi or (*b*) aqueous eosin followed by aqueous methylene blue, or (*c*) alcoholic eosin also followed by aqueous methylene blue; or
  (2) If heat be unavailable fixing with alcohol-ether and staining as above.

(*b*) When malaria parasites are to be sought for;
  (*α*) Single stains.
    (1) Aqueous methylene blue after any fixing agent.
    (2) Carbol thionin blue after any fixing agent except corrosive sublimate.
  (*β*) Consecutive stains.
    (1) Fixing by heat or alcohol and staining with aqueous eosin, followed by aqueous methylene blue.
    (2) Fixing by heat or alcohol-ether and staining with alcoholic eosin, followed by aqueous methylene blue.

At the suggestion of one of us who is shortly to proceed to Central Africa, Mr. C. Baker, of High Holborn, has supplied a Ross diagnostic microscope, with the case adapted so as to hold, in addition to the microscope and two objectives, all the above mentioned necessaries for blood examination, viz., spirit lamp, glass bottle to hold coverslips kept in absolute alcohol, glass tube to hold three of Pakes' special pins kept in alcohol to prevent rusting, coverslip clip, tin box in which to pack coverslips after the films have been made, forceps, slides, vaseline and cedar oil. The whole outfit weighs only 4 lbs. and measures 11 in. by 3½ in. by 3 in.; it contains everything requisite for the immediate examination of fresh films and for obtaining dry films for subsequent investigation.

If the method of fixing and staining recommended in this paper be adopted, the following reagents and stains will be required; absolute alcohol, xylol, Canada balsam, immersion oil, formalin, chromic acid, half dozen Petri dishes about 2½ in. in diameter, spare forceps, filter paper,[18] cigarette paper, slides and coverslips (¾ in. squares and 1½ in. by ¾ in.). It would be best to take out the latter ready cleaned, packed in stoppered bottles in absolute alcohol. Corrosive sublimate, carbolic acid, vaseline, liq. iodi. and methylated spirit would be found in the ordinary stores of any medical man.

In enumerating the stains approximate quantities are given in order to indicate the rate at which they are likely to be used; the amounts here stated should suffice for one or two years' work. Methylene blue 20 grams, thionin blue, 5 grams, aqueous eosin "yellowish" or "bluish" 10 grams, alcoholic eosin, 5 grams and (if work on non-malarial blood is intended) Ehrlich-Biondi 10 grams.

A considerable number of the malarial blood films examined for the purposes of this paper were obtained by one of us at the Branch Seaman's Hospital, Royal Albert Docks. We wish to offer our best thanks to Dr. Manson for the opportunity thus afforded us and to testify to the unfailing courtesy and consideration which we have experienced at the hands of the resident staff of the hospital.

Paper read at the Annual Meeting of the British Medical Association.

AN ACCOUNT OF THE MEASURES TAKEN TO CONTROL THE EPIDEMIC OF PLAGUE IN THE CITY OF BOMBAY DURING THE YEARS 1897-98.

By Surg.-Major H. P. Dimmock, M.R.C.S., L.R.C.P.Lond.
Indian Medical Service.

The title of this paper indicates the object with which it is written, viz., to place before the British Medical public the details of a most extensive and widely organised system of dealing with the plague, that most dreaded and insidious epidemic disease, which in ancient times was completely uncontrollable, and in modern times baffles all efforts to stay its progress, for the sufficient reason that the sanitary conditions of the communities attacked by it have usually

[18] We have found that the best filter paper for this purpose is that supplied by Baird & Tatlock as "German lined."

not advanced beyond a primitive indifference to hygienic principles.

In order that the mind may grasp the nature of the preventive and controlling measures most likely to be efficacious in such a fatal and terrifying disease, whether encountered in the precincts of a great city or in a desert village, a scientific and reasonable basis for such efforts must first be thoroughly worked out, and upon this basis the various lines of offence and defence against the insidious and invisible foe must be developed with the greatest vigilance, foresight and determination. Even after all this had been done and the disease has apparently been mastered, there must be no relaxing of the measures adopted for a considerable time to come on account of what may be termed the *latent vitality* of the disease, which is intensely liable to recrudesce or burst forth in a renewed epidemic.

In formulating such efforts, wherever plague appears the conditions of the population attacked by it must be carefully reviewed, preparatory to applying the results of modern knowledge of the disease and its processes, such as have been derived from the labours of Kitasato, Yersin, Haffkine, Hankin, Cantlie, Lowson (of Hong-Kong) and Childe (of Bombay) in the first instance, and later of Bitter (Cairo), Koch, Gaffky, and their colleagues (Germany), Hermann, Franz Muller and his colleagues (Austrian), and the Russian investigators, Wysokawiez, Jassenki and others.

In reviewing the sanitary aspect of the disease as it appeared in Bombay, the conditions of the people will be found to be similar to those of any other crowded Oriental city where the first principles of wholesome housing have either been entirely neglected, or where, as in the city of Bombay, the immense difficulties of sanitation and house accommodation have increased beyond control by a rapid and excessive immigration and increase of population due to the attractions of a growing commercial prosperity.

In plague epidemics, experience has conclusively shown that the main factor of its diffusion is to be found in over-crowding. Its occurrence may be either an accident of importation or a blazing up of its endemic form. Arguments have been adduced to establish the theory of its endemic origin in Bombay, but they do not stand the test of verification and merely prove what is now an acknowledged fact, that the epidemic was preceded by either scattered cases or by the unrecognised existence of a mild epidemic for some considerable time. All factors of diffusion of plague are intimately associated with over-crowding, such as want of ventilation, insufficient sunlight, lack of personal cleanliness and hygiene, and all the unhealthy surroundings of the poor in a large city, and particularly in an oriental one.

Other weak points in the system of sanitation, such as deficient drainage and conservancy, aggravate the above conditions, and though they do not act directly upon the spread of the disease, the effect that such deficiency must have upon the tone of the general health and the resulting incapacity to resist morbific processes, must be acknowledged. At the same time it is curious to note that plague, like some other infective and contagious diseases, is as rampant in a small village, where drainage is not of such vast importance, as it is in a large city, and here again the virulence is entirely due to the overcrowding of families in small dark rooms (from choice as well as from necessity) associated with the other conditions.

It cannot be denied that sanitary difficulties of great magnitude exist in Bombay, due to the physical conformation of the island, to the very nature of the soil which is largely one of reclamation, and to the habits of the people which are the heritage of generations of blind indifference and fatuity in those matters that are essential to the well-being of all communities and cities.

In spite of this, and the fact that the city was in a state that exposed it to the introduction and spread of plague, Bombay compares favourably in its general sanitary condition with most oriental cities, and even with some continental ones. Dr. Simpson, the late distinguished Health Officer of Calcutta, and his successor now in office, Mr. Neilde Cooke, when shown round the worst localities where plague had committed its ravages in Bombay, pronounced favourably both on the state of the houses and their sanitation compared with similar localities in Calcutta. The successive municipal commissioners of the city and many of the enlightened citizens have made strenuous efforts to rectify the errors of past ignorance, and to carry out the projects of modern sanitary science. The splendid water supply, the general drainage scheme and the careful conservancy of the city are noble evidences of progress.

At the time of the first appearance of the epidemic, and even now during its lingering presence with the possibility of recrudescence, the poor reside in houses known as *chawls*, that is, large lodging-houses of many rooms, capable of accommodating as many as a thousand souls, each room being inhabited by a family of several individuals.

These rooms are in many cases very small (12 by 12 by 10 feet being an average size for a family of five) and with no outlet except on to a long dark corridor, so that a lamp is necessary to view them even at mid-day; in other cases the windows are entirely closed in accordance with the peculiar ideas of the people.

Although there are minute religious instructions concerning cleanliness in the preparation and eating of food, and in washing before meals, there is but a very perfunctory observance of them or of the hygiene of the person, clothing, bedding and general conditions of the room; all the individuals sleep on the floor, generally with a closed door, and the confined space reeks with the organic impurities of exhalation and secretion. This state of affairs has probably a great effect in a city and upon each epidemic of plague, for the disease has been most prevalent in Bombay during the cold-weather months when the people crowd into their rooms for warmth and shelter, while its virulence has waned and finally decreased in the hot weather, when the people leave their rooms and sleep on the roofs or in the street. Beyond these evidences no other relation of a decided kind can be attributed to the influences of meteorological conditions.

In the chart delineation, comparing plague and

general mortality, there is a high rise of both, coincident with the usual depression of temperature in the cold season; with the rise of temperature there is a coinciding fall in the mortality records, both plague and general.

Later, with the rise of humidity during the monsoon, there is a slight rise of mortality, plague and general, and this is less likely to recede as the cold weather approaches and the most favourable conditions for the spread of the disease again predominate.

(The occurrence of plague in other parts of India at other seasons of the year and under entirely different meteorological conditions is beyond explanation at present, and is one of the many apparent contradictions with which investigators of this disease are constantly met, but when all the literature of plague has been compiled, the meaning of these vagaries will perhaps be made clear.)

Such were the conditions which invited the spread of this contagious and infective disease in Bombay. That it *is* infective in a certain degree must be conceded from the distinct evidence afforded by the cases of pneumonic type which could only be developed by the inhalation of infective particles.

(2) The nature of the disease must next engage attention, and the basis of any such enquiry is the "*fons et origo*" of the malady—the plague bacillus. This bacillus has been fully described by many writers, and its virulence and power of multiplication are really terrific, even when observed under the artificial conditions of scientific investigation.

It is certain that the plague bacillus develops under the conditions of darkness, moisture, warmth and organic albuminous nutrient media, and all these conditions are found in the small crowded rooms that have been described.

Any scientific investigation, be it ever so careful and thorough, must obviously be wanting in the natural requirements of the bacillus, and thus it is that artificial experiment has failed to demonstrate some of the most striking characteristics of the microbe, especially its power of lurking continuance in the region of its original habitat, and its varying behaviour under transfer either by artificial or natural means. An instance of the former is to be found in the persistence of contagion in an infected room for days, weeks and even months after it has been evacuated, provided none of the other conditions have changed, and laboratory experiments have given numerous instances of other striking characteristics, but have failed to explain how it is the microbe thrives under certain natural conditions but diminishes in virulence under like conditions artifically arranged.

Mr. Hankin's numerous experiments with woollen materials is a particular example, for though careful cultivation of the bacillus was made in them, its virulence could only be maintained for a definite period, while soiled bedding if undisturbed will retain its virulence for a long period; disturbance and exposure to light and air must necessarily accompany artificial experiment, and these are unfavourable to the growth of the microbe.

(3) Further studies of the physical, clinical and pathological nature of plague lead to a chain of more or less definite conclusions as to the primary occurrence and its spread among the population, the means of infection, the period of incubation, the method of its invasion of the human host, the special characters of the disease and its ultimate effects, all of which knowledge is invaluable in the selection of extensive measures for the control of an epidemic.

The first recorded appearance of the disease in Bombay in recent years is now fixed early in August, 1896, when the disease was observed and afterwards verified by Dr. Ismail Jan Mahomed, Dr. Viegas, Dr. F. N. Surveyor and other distinguished native practitioners. In August and the succeeding month of September, 1896, so many cases were observed by other leading medical men that a feeling of alarm arose and the public attention was called to the danger that threatened the city.

It is impossible, in the entire absence of information about the very early cases of plague, to say how it found an entry into the city. The disease had been raging in Hong-Kong two years previously, and it is endemic in certain areas of the Persian Gulf territories, all of which places are in frequent sea communication with Bombay.

There had been no records of similar epidemics in Bombay for fifty years, but between 1830 and 1836, plague had certainly committed ravages in the presidency.

The epidemic of 1896 began in the region of the shipping, and the stalwart coolies working in the docks were the principal sufferers. Coincidently with these occurrences, and previous to an increase in the severity of the epidemic, rats died in extraordinary numbers in the same locality. These circumstances were followed by an increase and extension of the disease into the city accompanied by the same peculiar relation between the rat mortality and the appearance of plague cases among the people.

From the numerous observations evidence has been gained that the main cause of the spread of the disease is by human contact; further, that the first infection of human beings is often derived from plague-infected rats, and, *vice versa*, that rats of a certain vicinity may be infected by a migrating infected human being and become thereby a fresh source of danger. Professor Koch has emphasised the important influence that rats have upon the spread of plague as the result of his observations on plague in Bombay, and the fact may be accepted in its full significance when we consider that rats live in sewers and holes, and, when stricken by plague, come out into open day, evidently suffering from great thirst, for they are found in numbers dying or dead near the washing places where there is water for them to drink. Rat bite is a common complaint in Bombay, and it is well known that the animals move freely and unmolested in large numbers about the houses, and run over the sleeping inmates at night. Contact with plague-stricken rats in this way is probably the cause of many cases of plague.

Important as is this question of infection of human beings by rats, the capacity man has of conveying it to man is still more important. From the very earliest stages of the epidemic it was continually seen that an infected human being who was allowed to stay in his own room invariably infected those of

his friends and relations who remained with him, and even persons within the same building, so that a number of cases would occur in it in quick succession, and by the migration of some of the sufferers the same sequel of events would take place in other houses. Hence a plague epidemic starts from a centre of infection and spreads widely around it, at the same time starting other centres of infection which in turn widen and meet until the whole city becomes infected.

The period of incubation of the disease marks the rate of progress, and by experience, confirmed by the Venice Convention, is fixed at *ten days*. Reference to the progress maps shows that the intervals at which the different wards became infected, and the rate of progress of the epidemic in each ward would be explained by an average period of incubation as stated. It was impossible to verify many cases on this important point, as the people, from panic, became deceptive and evasive of any inquiry; nevertheless, there were many facts which helped to confirm the statement. Probably eight days is nearer the exact period, for when people were removed from infected houses to a health camp, hardly any cases occurred after detention for a week, although a fair number occurred in the first few days.

The method of invasion of the human host by plague is to be studied in the clinical features of the disease, and these are comprised in three groups:—(1) Fever with enlarged glands—the commonest type of the disease; (2) Fever without enlarged glands, but associated with a rapidly fatal blood toxæmia; (3) Primary plague pneumonia.

It is not the intention of this paper to describe the types of the disease or its clinical aspects, but to deduce from these three main groups the manner in which the poison enters the human body.

THE FIRST GROUP, WITH ENLARGED GLANDS, the predominance of which is so great as to have given rise to the generic term bubonic plague; the lymphatic glands of an extremity or other region become swollen, painful, and enlarged, and in a great number of instances will suppurate and slough away if an incision to release the pus is not made. In the greater proportion of these cases the glands of the lower extremities, femoral and inguinal, are affected; next come the glands of the axilla and then the cervical glands. It is a common-sense deduction that if the lymphatic glands in a single region become enlarged and inflamed in this way, the cause of the condition is to be sought in the introduction of some poisonous material into the distal lymphatics. I do not see how any other explanation is possible.

When such local symptoms are accompanied with specific general symptoms such as we have in plague and all of which have been produced times out of number in laboratory experiments, conclusions only point one way. The same remarks apply to cases in which the axillary and cervical glands are enlarged. That this deduction is true has been shown in many cases, as, for instance, infection occurring in persons suffering from an ulcer on the leg, showing distinct local inoculation, and one notable case, that of Miss Macdonald, of St. Bartholomew's Hospital Nursing School, a most distinguished and devoted nurse, who developed the cervical form of plague by infection from the saliva of a patient, who in his delirium spat in her face, the *materies morbi* entering her eye. The lymphatics of the left side of the face and neck became acutely inflamed, the general symptoms of plague appeared and she died in four days. Objections have been raised to this explanation, because in a great number of cases no open sore is present and no point of inoculation can be detected.

Circumstances indicate that the microbe has acute powers of penetration, and if it should form a nidus in the flexures and clefts and folds of the toes and fingers, it might easily find a nutrient medium for its development where this property of acute penetration could be exercised. Another possibility is inoculation by the bites of insects, such as flies, mosquitoes, fleas, bugs, &c., which abound in all the native houses.

Mr. Hankin made some very interesting investigations and showed that ants could certainly carry the plague bacillus. The life history of the microbe has perhaps not been altogether discovered, and there may be spore forms of the minutest size which are the true carriers of the disease. Scabies and skin disease are exceedingly common as well.

A brief review of the habits of the native of India will help to show the grounds of the above conclusions. They all of them move about their houses with bare feet, which are often fissured or excoriated; the house latrines and the bathing places are used by the sick and healthy alike and are generally saturated with organic matter; they sleep with their heads covered and their legs exposed, on the bare ground or on quilted cotton mats which are never washed and are indescribably dirty, and the women clean their household utensils with mud from the kennel of the street, which is an open drain.

(*To be continued.*)

THE *Pekin and Tientsin Times* of September 10, 1898, announces the appearance of a new Journal on medical science in the West. The Journal is to be published in Hong-Kong and edited by Dr. Wan-Man-Kai, formerly a student in the Tientsin Medical College, and at present House-surgeon in the Alice Memorial Hospital, Hong-Kong. The periodical appears monthly and consists of six parts: (1) Articles from celebrated works; (2) Cases of new and difficult diseases; (3) New theories and methods; (4) Elementary treatment; (5) Hygiene; (6) Various. It will also be illustrated.

THE Hong-Kong Government, from information received, being suspicious that plague existed in the city of Canton and neighbourhood, caused diligent enquiries to be made, with the result that as far as Canton itself is concerned "there has been no plague or sporadic cases in Canton since the last epidemic in the spring of 1898." An epidemic of influenza and a severe outbreak of malarial fever no doubt caused the report that plague was virulent. In Fatshan, some few miles up the river from Canton, sporadic cases of plague are occurring.

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MEDICAL REGISTRATION IN INDIA.

IT is now some eighteen years since the medical profession in Bombay first drew attention to the anomalous position it occupied with regard to quacks, owing to the fact that there was no Medical Registration Act for the city or island. A representation on the subject was made to the Government of Bombay setting forth the manner in which non-registration affected the public and the profession, and proposing the enactment of a law of registration for the Island of Bombay. The proposal was submitted by the Local Government to the Imperial and Provincial Governments of India for their opinion, and as a result of this consultation a decision was arrived at, that the introduction of a system of medical registration into any part of India was, at that time, premature. It was unlikely that a decision of this kind would prove satisfactory to the petitioners or to the large number of Indian medical men who were interested in the movement, and who were hoping that if the medical men in Bombay were successful, a similar enactment would be extended to the other large towns. Medical graduates of the Universities and licentiates of the Colleges, who had settled in the large towns, had only too much reason for complaint. They were surrounded by barbers, carpenters, washermen, milksellers, cooks, painters, masons, &c., who, having failed at their calling, had betaken themselves to the practice of medicine, and were deceiving the public by their high-sounding titles, and by the proclamation of wonderful cures; many were practising under the names of Vaids, Hakims and Kobirajes, but it was only the few who were conversant with the native system of medicine which the names implied, and not one of them had any knowledge of the structure of the human body.

With this chaotic condition of affairs there was plenty of material to keep the agitation begun by the profession in Bombay smouldering, notwithstanding the unfavourable attitude of the Governments, and it was only a few years later, in 1887, that the next manifestation of discontent showed itself, but this time in Calcutta. Then Dr. Birch brought the subject before the Calcutta Medical Society, and after a debate in which many of the leading medical men in Calcutta, native and European, took part, it was unanimously resolved: "That the Medical Society of Calcutta are of opinion that there is need for a Registration Act in Calcutta." The resolution did not go further, for it was considered to be desirable to advance on the lines of least resistance, and not to jeopardise the strong case made out for Calcutta by making the resolution too wide and applicable to other parts of India. The paucity of qualified medical men in India had been one of the reasons assigned for the rejection of the representations of the Bombay profession. The objection could not apply to Calcutta in 1887, where an increasing number of qualified medical men were every year beginning practice.

In order to assist the Government by every possible means in an admittedly difficult task, the Society drafted a scheme such as was considered would meet the case, adapting the provisions of the English Medical Registration Act to the circumstances of India. Dr. McLeod, the Secretary of the Society, in forwarding the resolution and scheme to the Bengal Government, explained

that the main objects of the Society's suggestions were to enable the public to recognise genuine and safe medical practitioners from dangerous and ignorant pretenders, and to prevent the latter from assuming and parading titles and appellations which imply a training, knowledge and skill which they do not possess.

While the Calcutta Medical Society was thus active, the Medical Union in Bombay also bestirred itself and drafted a similar scheme for Bombay. The best laid schemes, however, at times come to naught, and this was the destiny both of the Bombay and Calcutta schemes. The representations failed to accomplish the object which the promoters had in view. Again the matter came up for consideration at the first Indian Medical Congress in 1894, and a resolution was unanimously carried as to the necessity of medical registration in India. This resolution shared the same fate as its predecessors, and it appears that the medical profession in India is no nearer to the attainment of the desired object. We are glad to see that in spite of the many discouragements met with, there is still the same determination to have this grievance removed, and it is with pleasure we see that the Indian Medical Association has revived the subject. In this connection the recently published Indian Medical Directory of Dr. Wallace's will prove most valuable, for it shows that India possesses a very formidable list of qualified medical men, who very reasonably consider themselves ill-treated in that they enjoy no legal recognition and status.

We trust this will be the last occasion that the Government of India needs to be approached on a subject which intimately affects the health of the public and the interests and prestige of the medical profession in India.

PROFESSOR VIRCHOW is the fifth scientist on whom the Physico-Mathematical class of the Royal Academy of Berlin has conferred the gold Helmholtz Medal. Its weight is 620 grammes, and it was struck in memory of Helmholtz's 70th birthday, in 1892. Herr Virchow's predecessors were Helmholtz himself, Dubois-Reymond, Weierstrass, and Lord Kelvin.

Article for Discussion.

ACCLIMATISATION.

DURING the late war in Cuba a great deal has been said and written concerning the acclimatisation of the combatants, and the advantages the Spaniard possessed over the American from the fact, that the former was "resident" at the seat of war. By many it was stamped as an inexpedient, unwise, cruel, or some such, action to send American troops to a tropical climate at the most unhealthy season of the year; not so much because it was unhealthy, as because the soldiers were being sent to be opposed to troops which were thoroughly acclimatised. The logic of this belief is not quite scientifically clear. The Americans did not hesitate to attack, during the unhealthy season in Cuba, so much because it *was* the unhealthy season, as because their enemies were "seasoned," "acclimatised," or "immunised," and supposed to be fit therefore to better endure a campaign when exposed to the exigencies of climate. A belief which well-nigh deferred warlike operations, which meant the maintenance of a large army and navy on the war footing for six months, and the possibility of allowing the enemy a period in which to prepare for defence, would have been truly an expensive one, had it been acted upon.

What was the practical issue? an attack during the unhealthy season by the unseasoned troops, a rush of men fresh to the tropics and full of go and vigour; on the other hand, a backboneless defence and the complete collapse of the "acclimatised" troops. It was the very rapidity of the onset and the alacrity of the attack that saved the Americans from succumbing to the climatic diseases. Had they met with a reverse, had they waited until thoroughly equipped for the field from a military point of view; had the campaign lasted months instead of weeks, the climate would have begun to tell its tale and the medical records would have been very different. But even then the question is would they have been in a worse plight than their enemies who were resident in the country?

The term "acclimatisation" signifies the gradual adaptation of animals or plants to altered conditions of nutrition and of climate, and to the power of resisting diseases differing from those met with in their original habitat. It will be observed that the process is claimed to be a gradual one, and it is found to be so in the case of both plants and animals. In the three previous issues of this JOURNAL the question of Europeans becoming naturalised in the tropics, was discussed. There it was shown that the Dutch in South Africa, the Red man in America, the negro, &c., had each and all changed from their original habitat and flourished in conditions remote from their ancestors. But in each of these examples the process was gradual. The Red man travelled south, not by steamboats or railways, but by a gradual colonisation. From North to South America he crossed the isthmus, and encamping on fresh territory, settled there, whilst his progeny sought fresh hunting grounds and added a few miles further south to his patrimony. The extension from the north to the extreme south of America may have been, and probably was, the work of centuries. With the Dutch it was the same; 200 years elapsed between the time they landed in latitudes but tolerably different from their home, until they became fitted to live in regions nearer the equator. The acclimatisation of a race is a gradual process; it is not an individual but a race possibility; and to speak of the young Spanish soldiers, mostly country lads drawn from agricultural pursuits, as being acclimatised, is a misinterpretation of the term. The Americans had not to meet acclimatised troops; the Cubans no doubt came under this category, but the Spanish soldiers were being decimated by disease at the time the war broke out, and it only required the fatigue and exposure incident to a military campaign to develop the latent phase of malaria into active illness, or the environment of camp life to generate into activity the ailments which perpetually threaten the European in the tropics. Gauged from the point of acclimatisation, therefore, the Americans had the best of the situation. Residence in an unhealthy district no doubt weeds out those susceptible to disease, leaving the survivors, as a body, more capable of resisting the deleterious effects of climate, and in this sense alone can an army composed of youths from Europe be deemed acclimatised.

One must guard against mistaking "domestication" for acclimatisation. A man may be resident in the tropics for many decades and may have his family home there, but, although he is domiciled there, he is not necessarily acclimatised. The canary is bred and reared in northern Europe in captivity, but the canary is wholly unfit for independent life in the woods and coverts of these northern lands. It is in no sense acclimatised or naturalised and never will be; at the same time it is domesticated and can continue its species under the altered conditions. True acclimatisation can only come by heredity and that, too, with variation in the progeny. It is a gradual process and the time involved is to be counted by generations not by years only. The progeny of the Anglo-Saxon in America and Australia is a distinct variation from the parent type, and as time advances the range of variation will no doubt increase. In these countries the immigrants have become acclimatised, or in other words naturalised; and in the case of the United States, if acclimatisation is to count for anything, the troops engaged in the recent war should have been better fit to endure the fatigues of war in a country adjacent to the land of their acclimatisation than young lads from Spain, whose only claim to acclimatisation was, that they had not died or been invalided like so many of their comrades, during the year or two of their stay in the malarial-stricken districts of Cuba. The result justifies this conclusion, for there is no doubt that the Americans, by their prowess, their staying power, and their freedom from epidemic disease, proved they were better fitted to endure the hardships of the campaign than the so-called acclimatised, but really the disease-spared soldier from Spain.

J. C.

UPWARDS of 2,600 people died of plague in the city Bangalore.

THE Plague in Mysore has now extended to the Gold Mines.

Correspondence on Articles for Discussion.

THE SIESTA. BATHS AND BATHING.

MANY natives and customs exist outside the British colonies. Maybe these notes from a Spanish-American country will be of interest. 3·30° N. sounds tropical enough, and that is the latitude of Palmira, the agricultural centre of the Caves Valley. Who are the natives? The most beautiful permutations and combinations exist. The aboriginal Indians—descendants of the Incas and a host of tribes—scarce; descendants of the Spaniards and of the free negroes. Then an occasional Chinaman has dropped in somewhere. A few hundred Britishers settled in Colombia at the time of the revolution and they have contributed to the mixture.

As for the Siesta it is a case of snakes in Ireland. I believe it is long out of use in many other Spanish-speaking lands. Here the first meal of the day (excluding the 6 o'clock coffee) is between 9 and 10. Work is resumed and goes on till 4. The women work like the men, generally a little more so. Not that I pretend that the day's accomplishment is a large affair.

Bathing is universal; between 10 and 11, like Adam and Eve, they all go to the river to bathe. One Palmira stream has some 2½ feet of water on occasions. But no one washes; a few minutes' splashing, some douches over the head, and into the old rags, without any rubbing or drying. (Of course we have our special skin diseases not worth attempting to cure under these conditions.) Soap for washing purposes is a rarity and towels don't exist. On a journey the hands must not even be rinsed. A journey of ten days (horseback) is no uncommon affair, but you mustn't wash the whole time nor bathe for several days afterwards. Nor is any washing permitted in times of sickness. A mulatto consulted me today on account of a stricture, who assured me (unnecessarily) that he had been unable to bathe for a year—he had been so ill. Don't be deceived into thinking the natives understand the conditions of the country better than foreigners. You must have a dietary composed of salted meat, garlic, fat, and plantains.

All the foreigners I know here have had malarial fever at some time or other. But none on that account gives up the morning cold tub. Like myself, they find it comes like the Pickwick, the Owl and the Waverly pen.

M. D. EDER.

Palmira, Republic of Colombia,
December 27, 1898.

Recent Literature on Tropical Medicine.

OPHTHALMOLOGY IN ITS TROPICAL BEARINGS.

THREE papers of special interest to students of tropical ophthalmology have recently appeared in the *Wiestnik Ophtalmologii* (Moscow).

TRACHOMA AND MUCO-PURULENT CATARRH IN SYRIA AND PALESTINE.—Dr. Th. Herman was sent to Syria and Palestine to study the types of trachoma and conjunctival catarrhs prevalent in these countries. He found trachoma and conjunctival affections in general much more widespread in Palestine than in Syria, and enumerates the influences which he believes produce the greater prevalence in the former country. Of these the chief are: the prolonged rainless season (May to October) during which dense clouds of dust are being constantly blown about, and there is practically no shelter from the pitiless rays of the sun; the incredible filth of the inhabitants owing to scarcity of water; the enormous number of flies; and the insufficient protection afforded by the national head-dress. Once the rainy season begins ocular troubles lessen.

The trachoma prevalent is of a sub-acute or chronic type, with enormous granulations; corneal complications occur in 12 per cent. of the cases in Syria, in 24 per cent. in Palestine, and are as a rule not of a grave character. Next in importance to trachoma the author notes a mild variety of muco-purulent catarrh mainly affecting children and often appearing in an epidemic form. I have seen a similar form of muco-purulent ophthalmia in China and Japan; at first the disease closely resembles purulent ophthalmia, but the cornea is but rarely affected, and the vast majority of cases terminate after a few days in apparently complete recovery.

EYE DISEASES IN TURKISTAN.—A lady doctor, Mme. Dikanskoga, contributes an interesting article on the eye diseases of the recently annexed tribes in the Turkistan country north of Afghanistan and Persia. Trachoma of a severe type, leading early to cicatricial contractions, is very rife. The authoress has seen numerous cases of entropion in children under ten. The natives practise a rude operation for the cure of entropion; an extensive fold of the skin of the eye-lid is pinched up between two pieces of stick, the ends of the sticks tied tightly together, and the apparatus left *in situ* till it drops off with the necrosed fold of skin. The immediate results are stated to be satisfactory, but relapse occurs sooner or later. The Afghan "doctors" who practise in the country improve on this procedure by excising a portion of skin and bringing the edges of the wound together with thread; these professors of the healing art also couch for cataract, with what result is not stated. Blindness is exceedingly common, the main causes being, in order of frequency, trachoma, glaucoma, small-pox; glaucoma being responsible for no less than 23 per cent. This last observation is of much interest in view of the

wide-spread belief amongst practitioners in the tropics that glaucoma is more prevalent there than in temperate climates.

Larvæ in Trachomatous Eyes.—Ivanoff, of Kazan, describes three cases of trachoma in which the living larvæ of a fly, the "sarcophyl Volpharthii" (*sic*) were found in the conjunctival sac at the external angle of the eye. In one case the number of larvæ reached the enormous total of 125. Beyond excoriating the conjunctiva they seem to have done little harm. The same larvæ have been found in the nostrils and external auditory meatus.

Malarial Eye Affections. — Under the heading "Affections Oculaires d'Origine Malarienne" the *Recueil d'Ophtalmologie* publishes a lengthy review of my paper on "Malarial Eye Affections" which appeared in the October number of the Journal. It is to be hoped that this notice of a very humble contribution to the study of an interesting class of diseases, appearing in a periodical so widely read by ophthalmic surgeons all over the world, may induce other and better qualified workers to take up this important subject. Professor Guarnieris's recent researches on the microscopical anatomy of the retina in malaria are of great value and open up a fruitful field of investigation; and there are many other points on which trustworthy evidence from those dealing with the necessary clinical and pathological material is required. There are grounds for believing in the existence of a special malarial irido-cyclitis, characterised by periodicity and tendency to relapse. Cases of keratitis profunda and keratitis dendritica presenting peculiar features have also been attributed to malaria. So far however, the evidence has hardly been sufficient to warrant a special nomenclature, and the tendency in the past to label "malarial" any disease attacking the victims of paludism has been productive of much error and confusion.

M. T. Yarr,
Major, R.A.M.C.

TURKEY.

Beri-Beri on the Arabian Coast.

Dr. Stékoulis, of Constantinople, in *Janus* for January-February, 1899, reports an outbreak of Beri-beri on board a ship in the Red Sea under peculiar conditions.

The s.s. *Nour-el-Bahr*, with a crew of thirty-three persons, twenty-eight of whom were Indians from Bombay, sailed from Genoa, where she had just been built, to her station at Camaran on the Red Sea, on September 21, 1898. On November 17, twelve of the crew, all Indians, were seized with Beri-beri, and three of them died in the course of a few days. The ship was new, and consequently clean; the food and water were wholesome as far as is known, the rice used by the Indians being obtained in Italy and Egypt; and it was seven months since the Indians had left Bombay. The mystery is how did the disease arise? So far as can be gathered it was not due to contagion, and the outbreak seems to have been "spontaneous," that is, originated on board the vessel itself. Seeing that seven months had intervened between the time the Indian crew had left Bombay and the date of the outbreak it can hardly be conceived that the disease could have been latent for so long a period. Such a conclusion might have been discussed had only one or two cases occurred, or if the twelve cases that developed Beri-beri had done so at intervals, but the sudden appearance of so large a number points to a common source of infection. What that source was, however, we are not, in the present state of our knowledge, able to decide.

UNITED STATES OF AMERICA.

Yellow Fever.—The Archinard-Woodson Bacilli.

In the *New York Medical Journal*, of January 28, 1899, the Doctors Archinard (two) and Capt. Woodson, M.D., U.S. Army, published the result of their bacteriological researches in yellow fever during the outbreak in New Orleans in 1897. They describe two bacilli, in fact, which however, they regarded as interchangeable and accounted for by cultural variation.

The bacillus they describe has the following characteristics: a short thick rod, 2 to 4 mm. in length and 1 to 2 mm. in breadth. In different media pleomorphism is apparent. It is actively motile, aerobic, growing best in the presence of hydrogen. The bacillus stains well in all watery solutions of basic aniline dyes, and unstains by Gram's method. It grows best in neutral and weakly alkaline media, both solid and fluid, and at a temperature of 37°C.; on potatoes it produces a whitish yellow transparent growth; it does not liquefy gelatine.

The bacillus agrees in well nigh every point with the Bacillus icteroides of Sanarelli. The bacillus has some points in common with the coli communis, but has characteristically specific differences therefrom. The observers obtained their information (1) from 39 yellow-fever autopsies in 32 of which the bacillus was found; (2) in blood taken from the veins of patients suffering from yellow fever the bacillus was found in 4 cases out of 5 examined; (3) in the exhaled breath the bacillus was found only twice in 12 cases; (4) in 12 cases in which the scrapings of the skin were examined the bacillus was found twice; (5) 5 to 10 cc. of the bacillus culture injected into the vein of a rabbit and subcutaneously in the guinea pig were always fatal.

Blood Examinations at Camp Wikoff, U.S.A.

In August and September, 1898, Dr. James Ewing was detailed for duty in the camp at which the troops returning from Santiago were located. The special work entrusted to Dr. Ewing was to render what assistance blood examinations might give in the diagnosis of tropical and other fevers. Of 800 examinations, 605 proved the diseases to be of malarial origin. Of these 605 cases, plasmodia were found in the blood in 335 cases, while in 270 the diagnosis was based upon the clinical history and the discovery in the blood of malarial infection. Of the 335 cases in which organisms were seen, the signet-ring form only of the æstivo-autumnal parasite was found in 88 cases; crescentic bodies only in 134 cases; and both rings and crescents in 27 cases. The æstivo-autumnal parasite was found in 261 cases; the tertian in 74; and the two were found associated in 12 cases.

It appears, therefore, that about 80 per cent. of the Cuban malarial fever are of the æstivo-autumnal variety, 20 per cent. of the tertian type, and 4 per cent. show a double infection. Quartian fever would seem to be extremely rare.

Of the 605 cases of malaria, 39 died (6½ per cent.). Of these 39 cases, 25 showed æstivo-autumnal infection, 2 tertian, 2 double infection, and in 10 no distinct parasites were found. In the æstivo-autumnal cases, rings were found in 12, crescents in 9, and in 4 both rings and crescents. Dysentery, not always amœbic, obtained in 6 of æstivo-autumnal fatal cases; 4 had severe diarrhœa and colitis; 8 were jaundiced, one died of pneumonia, and one of nephritis.

Methods of Blood Examinations.

(1) Success in the examination of blood is the result of experience.

(2) The best stain for blood parasites, as in malarial disease, is the double eosin and methylene blue.

(3) For staining granules in white cells Ehrlich's triacid stain is to be employed.

(4) The hematocrite is more convenient than the leucocytometer, and gives as good results; it deserves a separate place in the clinical examination of the blood.

(5) The type of malarial fever endemic in New York and suburbs is nearly always the tertian. The quartan type and the crescent form occur at a great distance from the city.

(6) The microscope enables us to distinguish between the various types of the blood diseases.—Dr. H. Heiman *in Post Graduate.*

The Transmission of Disease by Certain Insects, Ticks, Bed-bugs, Ants, &c.

In this paper, which appears in the *New York Medical Journal* for October 22, 1898, Dr. Charles F. Craig continues his discussion on the subject of the agency of insects in transmitting disease. There can be no doubt that the necessity of research in this direction is very real, and that such investigations are likely to be brilliantly successful is well brought out in the present paper. The disease of cattle to which the term "Texas fever" has been applied has now been clearly demonstrated to be closely related to the life-history of the cattle-tick (*Boophilus bovis*). This parasite is invariably found adhering to the body of cattle the victims of Texas fever, but it is only quite recently that the laborious researches of Theobald Smith and F. L. Kilborne have shown that the parasite is in reality an accessory before the fact in determining the disease. The symptoms of Texas fever are those usually associated with febrile attacks, together with hæmoglobinuria, which last symptom has given rise to the term "red-water fever." Careful and repeated examination of the blood has revealed the presence of a parasite in the corpuscles, to which the name *Pyrosoma bigeminus* has been given by Smith and Kilborne, and which belongs to the protozoa. It is considered that this organism (the life-history of which considerations of space do not allow us to enlarge upon) is the cause of the disintegration of the red corpuscles, with the resulting hæmoglobinuria. Now comes the curious connection between this minute protozoon and the cattle-tick. It has been conclusively proved that animals free from infection become tainted when they feed in a field on which diseased cattle have grazed, and, indeed, that unless the tick or its eggs be present infection does not occur. At the same time it has been equally clearly shown that the ingestion of ticks with the food does not cause Texas fever. Clearly, therefore, the disease is transmitted through infection of the blood of the beast by the tick-bites. And the inoculation of the blood of a diseased upon a healthy animal is always followed by the appearance of the symptoms of Texas fever. At present the actual presence of the micro-organism has not been clearly demonstrated in the bodies or eggs of the tick, but there can be little doubt that before long such evidence will be forthcoming. This investigation is still more interesting when regarded in connection with Dr. Manson's views on the relationship between malarial fever and mosquito bites, and it is another proof of the great importance of the study of animal diseases, and of the claims of veterinary medicine to be included in the purview of human pathology.

Dr. Craig passes on to the transmission of disease by bed-bugs. Some facts relating to the history of this disgusting insect are interesting. It appears that to the Romans it was by no means unknown, and it is referred to in Pliny's "Natural History." It appears to have been recognised in this country in 1503, but it seems scarcely possible that, as the author thinks, it was but little known in the days of Queen Elizabeth. The universal custom in those days of using wooden bedsteads, and of keeping bed-chambers close and stuffy, together with the carelessness of personal cleanliness and hygiene, make it hardly possible that, where the conditions were so favourable, the filthy insect did not thrive.

A case is brought forward in which there is a certain amount of evidence that phthisis was communicated by bugs, through the occupation of a bed by the brother after another brother had died therein of that disease. Disinfection is said to have been practised, but considering that after the proceeding the bed was swarming with bugs, it cannot be said to have been effectual. The second occupant of the bed died of the same disease, and his body was covered with bites. Sixty per cent of the insects were tuberculous, and inoculations from them produced tuberculosis in guinea-pigs. Further, bugs have been placed in contact with tuberculous sputum, and after several weeks virulent cultures were obtainable from the insects. Very conclusive evidence of the active part taken by these insects in propagating relapsing fever has been adduced by Titkin, of Odessa. After being fed on the blood of a patient suffering from the disease, their bodies were carefully examined microscopically. Large numbers of spirochetæ were visible, and these were mobile. Their vitality persisted no less than eighteen hours within the insects. This evidence is extremely strong, and it can hardly now be doubted that these loathsome creatures do take a most active part in the propagation of disease. Thus the instinctive horror of these abominable pests is more than sentimental; it is a provision of nature to guard us against the access of many deadly diseases. Dr. Craig alludes to some very remarkable experiments on the transmission of cancer to white mice by means of bed-bugs from the cages of cancerous mice, in which perfectly healthy animals became in the course of some months cancerous. Further experiments of this description would be extremely interesting, not only from the nature of the apparent transmission, but also from the point of view of the contagiousness of the disease.

The paper concludes with some remarks on the relation of ants to the transmission of plague. The bodies of rats dead of plague were full of ants, and Hankin found on examining these ants that they contained the plague bacillus in virulent form and in large numbers. Here is clear evidence of another mode of transmission of this terrible disease—one, too, more difficult to prevent and less tangible than that by the agency of rats. The whole of Dr. Craig's paper is of the highest interest and importance, and we trust that the line of investigation treated of in this paper will be followed up in all directions. It is quite certain that if this is done results of the greatest importance both as regards prevention and treatment must result.—*Treatment.*

Malarial Nephritis.

In the *American Journal of Medical Science* for December, 1898, Professor Thayer, of the Johns Hopkins University, gives a categorical statement of nephritis in malarial fever estimated on observations made of 758 cases. Albuminuria, according to Professor Thayer, occurs in 46·4 per cent. of malarial cases, and casts in 17·5 per cent. In the malignant forms of the disease evidence of renal disease was much more frequent than in the benign. Although the proportion of cases of acute nephritis (4·7 per cent.) prevailed to a less extent in malarial affections than in many of the more common zymotic diseases, it is of sufficiently frequent occurrence to call for close clinical observation. It is a well-recognised fact that chronic Bright's disease is a frequent sequela of long-standing malarial infection.

FRANCE.

Blue Nasal Secretion.

At the annual congress of the French Society of Otology and Laryngology, M. Molinié, of Marseilles, related the case of a young woman, aged 25, in whom, after a severe attack of grippe, there occurred a discharge of blue secretion from the nose. In the beginning the discharge was generally viscous and colourless. Several times during the day, however, the mucus was streaked by lines of blue as deep as methylene blue. Examination of the nasal fosse demonstrated that the source of the secretion was the right middle meatus.

A short, squat bacillus with rounded extremities, coloured by methylene violet and gentian violet and retaining its colour under the Gram reagent, was found. Although cultures did not yield the characteristic blue colour, it is very probable that this case of blue chromorhinorrhœa was due to the development of a pyocyanic colony in the frontal sinus of the right side.—*The Medical and Surgical Review of Reviews*, December.

Importance of Rest in the Treatment of Anæmia.

W. Edgecombe (*Bull. Med. Jour.*, June 25, 1898) deduces from the observations of Oliver on the loss of hæmoglobin during the day and the increase at night in health—changes which are intensified by exercise, but not affected by massage—the importance of rest during the treatment of anæmia, so as to economise the diurnal destruction, thereby increasing the amount of hæmogoblin.

GERMANY.

Ankylostomum Duodenale.

W. Zinn and Martin Jacobs published a short account of their investigations regarding the pathology of ankylostomiasis, which is reviewed by Scheube in *Janus*, January and February, 1899. The authors do not regard the ankylostomum as a toxic-producing agent, but merely as a direct cause of anæmia owing to the abstraction of blood from the system, and therefore when present in small numbers cause no illness. So commonly is this parasite met with that, in all cases of tropical anæmia, the possibility of ankylostomiasis ought to be entertained and dealt with; old tropical residents returning to Europe suffering from anæmia require watching for a long time after reaching home. The authors contribute two charts showing the geographical distribution of the parasite.

In the *Wiener Klinische Rundschau*, 1898, No. 23-27, Leichtenstern states his opinion concerning the part played by the ankylostomum in producing disease. He devotes the first part of the work to show that Looss in his researches has found nothing new, and declares that all that *Looss* proved was that he had been able to generate the larvæ of ankylostomum in the intestines of puppies, and studied the various stages of the larvæ therefrom. Leichtenstern believes in the toxic power of the parasite, but does not exclude other means of poisoning. The author is of the opinion that the quantity of blood withdrawn by the ankylostomum, even when present even in great numbers, is insignificant. In many cases occurring in Leichtenstern's practice fifty to one hundred worms have been brought away by medicine, yet the patient has not been anæmic. The author differentiates three clinic stages of ankylostomiasis; (1) the stage of incubation, lasting four to five weeks, unattended by symptoms; (2) the stage of acute anæmia, caused in part by the abstraction of blood by the developing parasite, but mostly in those cases where a huge number of these larvæ are matured at the same time. Clinically this stage is characterised by colic, intestinal flux with blood and acute anæmia; *post-mortem* signs are a chocolate brown and blood colour contents of intestine and petechiæ in the mucous membrane of the intestine; (3) the stage of chronic anæmia caused by loss of blood, and the toxic influences due to the presence of the parasite in the intestine.

The Serumtherapy of Relapsing Fever.

Loeventhal (*Deut. Med. Woch.*, October 27 and November 3, 1898) immunised three horses by injecting them with blood containing spirilla of relapsing fever. 283 cases of this disease, treated in the Moscow clinic, are divided into two groups. The first, 152 in number, were treated in the ordinary way, and the remaining 131 were injected with serum obtained from the immunised horses. Of the 131 cases 84 were injected during the first apyrexia, and a few during the last days of the first paroxysm; 15 cases were insufficiently treated. Lastly, 16 were injected in the second paroxysm, 8 during the second apyrexia, 4 in the third paroxysm, 2 in the third apyrexia, and 2 in the fourth paroxysm. Of the first group of uninjected cases, 10 died and 2 were reinfected, so that 150 were left. Of these 18 had one paroxysm, 46 two, 65 three, 10 four, and 1 five. Six cases were treated with antistreptococcus serum without result. Of the 131 injected cases, 1 died, leaving 130. Of the 88 sufficiently treated, 89 had no further paroxysm. These 89 patients received 1,661 c.cm. of serum, or, on an average, 18·45 c.cm. per injection; 31 of the 88 had two paroxysms, 11 three, 1 four, and 1 five. Compared with the first group, these cases showed a more prolonged apyrexia after the first paroxysm, and a shorter second paroxysm. The single fatal case occurred at the end of the second paroxysm. In the whole 131 cases, 328 injections were given, amounting to 6,251 c.cm., or on the average 19·05 c.cm. per injection; and 47·06 c.cm. for each person. Thus the dose, compared with that of other curative sera, was not large. The pain caused by the injections was greater than that observed with antidiphtheritic serum. There was only one abscess among the 328 injections. Respiratory complications were equally present in both groups. There was a general exanthem, with joint swelling and albuminuria, and casts in 2 of the injected cases. One of these died, and at the necropsy there was found cardiac disease, phthisis, acute nephritis, &c. The author thinks that the renal complications were caused by the serum. Skin eruptions were noted in nearly 20 per cent., including the so-called early rash. Skin affections are often seen in relapsing fever, but the author would put them down here to the serum. The first injection should be given on the third day of the first apyrexia, and the second on the fifth day. Injections given on the fifth day of the first paroxysm are useless, and so also the commencing the serum treatment in the second paroxysm. If the treatment has been begun in the first paroxysm it should be extended during the following apyrexia. According to statistics, relapsing fever ends with one paroxysm in not more than 25 per cent. at most, whereas in the injected cases the percentage stood much higher. The average stay in hospital has been calculated at thirty days in the injected cases, whereas, according to statistics, it is 36·8 days. Loeventhal thinks that these good results can only be attributed to the serum treatment. —*British Medical Journal.*

THE CLIMATE OF THE EGYPTIAN SOUDAN.

Dr. Felkin read a paper on the climate of the Egyptian Soudan at a meeting of the British Balneological and Climatological Society. The paper is given in the January number of the Society's Journal, from which the following extracts are given. Dr. Felkin remarks that "owing to the immediate political outlook, it may be useful to briefly consider the influence of the climate of the Egyptian Soudan upon the Anglo-Saxon race, or the question of its suitability for their residence.

"In view of the present opening up of that country, the question has already been put to me, will colonisation be possible there? and individuals have asked me what chance they would have of living comfortably—that is to say, without their health breaking down—as emissaries of civilisation in the Egyptian Soudan.

"It is needful to consider, in the first place, the extent of the country. The Egyptian Soudan extends over 950,000 square miles, as compared with 121,000 square miles which make up the United Kingdom; it is, therefore, about eight times as large as the British Isles. It stretches north and south across nearly 24 degrees of latitude, from Egypt to the south end of the Albert Lake—some 1600 miles; and east and west it is from 1200 to 1400 miles broad.

"The population of this country has been roughly estimated at about ten millions, of which about three-quarters are negroes, but probably the slave trade, war, and famine, have reduced this figure considerably since the Mahdi's revolt.

"It will be readily understood that a land of such extent, and stretching as it does through so many degrees of latitude, must present very varied physical features as well as different climates. The conflicting reports as to what the Soudan is are due to various travellers describing what they have

seen; few have had the good fortune to travel throughout its whole extent.

"We are somewhat aided in our study of this subject by the fact that, again roughly speaking, the country may be conveniently divided into two parts by latitude 9°30 N. To the north of this line we find deserts, rocks, slight rainfall, great heat, an Arab population, and only three rivers—the Blue and White Niles and the Atbara. To the south of the line—a land most fertile, extremely well watered, with an abundant rainfall, a high mean annual temperature, and inhabited by negroes.

"Keeping this broad division in view, we can proceed to examine the climate rather more in detail, premising that in respect of altitude, in the south and west the mean altitude is about 4,000 feet, and it gradually sinks to 1260 feet at Khartoum.

"The actual mean annual temperature of the year in the northern part of the Egyptian Soudan is over 80° F., and the mean annual range of temperature, as far south as El Obeid, is about 30° F. I mean by this the difference between the coldest and the warmest month. The country south of Obeid to 5° 30′ N. latitude has a mean annual temperature of about 75° F., with a mean annual range of temperature of only 5 or 10 degrees, and the relative humidity of the year exceeds 70 per cent.

The mean temperature in the coolest season at Khartoum is 23·2° C.; in the hottest months, 36·8° C.; mean of year, 30° C.

"With regard to annual rainfall, it varies considerably. In the north, from the frontier of Egypt proper to Dongola, it is under 5 inches a year; from Dongola to Khartoum under 10 inches; from Khartoum to lat. 10° N. it varies from 10 to 25 inches. From lat. 10° N. to 4° N. it is about 60 inches. In the southern part of the Egyptian Soudan, the rainfall is pretty evenly distributed throughout the whole year, but from 5° to 10° N. there are two rainy seasons.

"The following observations taken at El Obeid may also be given as representative of the climate of Kordofan:—

June, 1875	... 28·1157	... 88° F.	mean.
July ,,	... 28·1054	... 83°	,,
Aug. ,,	... 28·0950	... 80°	,,
Sept. ,,	... 28·0976	... 80°	,,
Oct. ,,	... 28·0928	... 84°	,,
Nov. ,,	... 28·0917	... 78°	,,

"As we are dealing with a country that includes within itself a large slice of Central Africa, let us consider for a moment what Central Africa is. It presents striking peculiarities. It forms, as it were, an isolated entity, which has until very recently been cut off from communication with the rest of the world by its curious physical conformation. We may quite well describe Central Africa—as I think was first done by the late Colonel Grant—by comparing it with the centre of a soup plate, turned upside down. The rim of the plate represents the low-lying coast region; we then come to the slopes leading up to the plateau, and the bottom of the soup plate is the Central African plateau. We have then only to imagine upon this plateau three or four mountainous masses and three great rivers taking their rise in several great lakes on the plateau. They cut their way through its rock-bound borders, through gorges and over rocky beds, to find their way to the ocean surrounding the continent.

"It is to these facts, thus briefly stated, that the entity of Central Africa has been maintained for centuries. Low-lying coasts with pestiferous marshes and wide tracts of malaria have prevented much intercourse between the highlands and the coast, and the fact that the navigation of the Nile, the Congo, and the Zambesi is impeded by cataracts of no slight magnitude has prevented more highly civilised nations over-running the country, as they have done North America and elsewhere. For there has existed also a barrier on the north of the plateau in the shape of the great Sahara desert, and on the south in the form of the tsetze fly and the malarious regions south of the Zambesi.

"Now the Egyptian Soudan is about a quarter of the imaginary inverted soup-plate, and this implies that we have there a sample of all the different kinds of African climates, the meteorological conditions of which are distinguished by the regularity of the phenomena of weather, a regularity due to the massive form of the continent and its equatorial position.

"The only difference which we find in the Egyptian Soudan is this—that there is a very gradual rise up the Nile Valley to the plateau, and not the sharp rise which obtains both on the east and west coasts of the continent. So that in travelling up the river from the deserts between Dongola and Khartoum to the Albert Lake we gradually pass from a dry, hot climate, to one having a somewhat lower temperature, the air being humid and less tolerable than the drier atmosphere to the north.

"One is obliged to leave the river in order to gain those higher and comparatively healthy parts of the plateau which lie to the east and west of the Nile Valley.

"I must not omit to mention the Nile marshes, which extend from Bor to the Sobat. The grass which floats on the top of the water is so thick that it is quite possible to walk upon it, and it acts as a kind of force pump to force the water down to Egypt at the fall of the Nile. The surface of the water here is covered by a dense tangled mass of papyrus, ambatch, and other water plants, which grow to a height of from ten to thirty feet. There are millions of mosquitoes and other flies to torment the traveller; sixteen out of every twenty-four hours have to be spent within mosquito curtains, and the water one gets to drink is of the worst. The dreary stillness, and the hot, sultry, humid atmosphere, with no sound but the unceasing buzz of countless insects, is almost intolerable.

"This part of the Nile corresponds to the malarial belt upon the coast, and forms the most unhealthy part of the journey up the Nile Valley to the central plateau. But as soon as steam navigation becomes a regular thing, the water channel will begin to be kept free and the marshes will become less unhealthy, the blocks probably being prevented altogether.

"I have often ventured to give the opinion that acclimatisation in the tropics can only be expected if migration occurs step by step, and that in estimating the possibilities of acclimatisation we must count by generations rather than by years. At the same time—for you will say that this is not very practical in the consideration of present emergencies—there are areas in the Egyptian Soudan where I believe the British can do well at once. Unfortunately it has been up to the present time impossible to reach these places quickly enough to prevent loss of health before getting there. Now, however, the railways are beginning to be built, and steam communication on the Nile is being improved; so that we may hope that it will soon be possible for individuals proceeding to the regions I am about to mention to arrive there without having their constitutions undermined.

"In the northern districts of the Egyptian Soudan there seems to be no doubt that, in the dry, although hot, climate, Britons can live—if they know how—with a prospect of good health throughout nearly the whole of its extent. Practically the only unhealthy spot is Khartoum, because at the rise of the Nile the present site of the town is flooded, and at its fall the place is surrounded by an unhealthy swamp, which gives rise to fevers and dysentery. Fashoda, too, is rather unhealthy, but that is due to the southerly wind blowing across the marshes near the Sobat, and to the insanitary condition of the town itself.

"In the south there are two large areas where I feel convinced that, with reasonable precautions, we could thrive—in the Latuka and Shuli countries to the east of the Nile, and in the southern part of the Bahr-el-Ghazal district, in Makraka and the Niam-niam countries. In both these districts the altitude is considerable, severe malaria is unknown, and I have no hesitation in saying that we could exist there under conditions of comfort and prosperity. In

the course of time this region will be best reached from Mombasa, on the east coast, by the railway which is now being built from there to the Victoria Nyanza.

Great care should be taken to prevent persons with a tendency to gout or rheumatism, diabetes or albuminuria, those with a nervous or alcoholic family history, or those suffering either from acquired or hereditary syphilis, from going to tropical Africa; and more attention should, I feel sure, be given than is usually done, to the character and temperament of those who go out. There are some men about whom one can almost see at a glance that they are unsuited for tropical life, and more especially for a life under the conditions which for many years will obtain in the southern parts of the Egyptian Soudan. Men of courage, of well-balanced natures, over 25, and able to bear the necessary discomforts and worries incidental to the life with equanimity, are those who should be sought to carry our flag, our commerce and our civilisation, to the fertile regions now opened up to us.

New Drugs, Instruments and Surgical Appliances.

UNDER the name of "Frame Food Diet," the Frame Food Company, Limited, has supplied us with a food which is rendered specially nourishing by the addition to it of the soluble phosphates and albuminoids extracted from wheat bran. During the preparation of the cereal the amylaceous ingredients are converted into diastase, rendering them thereby readily assimilable by infants and invalids at home and abroad. For residents and travellers in the tropics "Frame Food" is especially suited. The great difficulty in medical practice in warm climates is to secure a readily digestible material of such a nature that a "tropical liver" can tolerate or digest it. Cod-liver oil and highly saccharated preparations are not suited for invalids during hot weather in the tropics; but in "Frame Food" we possess a cereal preparation of high digestive properties, eminently suited for children and for invalids suffering from the anæmic and digestive ailments consequent on tropical life.

A specially prepared "Jelly" has been recently issued by the Company, which by its pleasant taste and digestive properties is sure to speedily become a favourite. The "Frame Food" Jelly, like "Frame Food" Diet, contains the organic phosphates and albuminoids (extracted from wheat bran) which are most needful for those whose powers are so frequently sapped by the trying conditions of a hot climate. This delicious preparation possesses the nutritive and digestive properties of malt extract, costs less, and is more pleasant to the palate. Children eat it with avidity on bread-and-butter or in puddings. By invalids and convalescents it is readily assimilated, and supplies nourishment in a pleasant and efficient form.

HARTMANN'S Wood Wool Waddings and Tissues are so well known and appreciated that it seems unnecessary to specially mention their virtue. We would remind tropical practitioners that Hartmann's Wood Wadding or Tissue is especially useful in their practice. Coolness is an essential element in surgical dressings in the tropics, and wood wool pre-eminently fulfils that necessity. Its absorbent and antiseptic powers, its coolness, the perfect drainage it provides and the great economy it secures are factors in its favour which not only recommend themselves, but establish the pre-eminence of Hartmann's wood wool dressings as an especially suitable application in tropical surgery.

Review.

THE MEDICAL AND SURGICAL "REVIEW OF REVIEWS." An Indexed Monthly Summary of the Best in the Medical and Surgical Periodical Literature of the World. Edited by Nathan E. Boyd, M.D. Price 20s. annually. Publishing offices: Connaught Mansions, Victoria Street, London, S.W.

We have before us the fourth number of the second volume of this Journal. In appearance and style the Journal is all that could be desired, and the matter contained in the present issue is of a kind to attract medical readers. The necessity for a Journal with an aim such as is expressed by the legend "Review of Reviews" is well nigh imperative, and success is sure to follow so excellent an attempt to condense the knowledge contained in the widely scattered medical literature of the present day.

The difficulty is to narrow the work of such a periodical, and the real danger is that the "Review of Reviews" becomes overloaded with material to such an extent as to defeat its ends. Important facts are shortly told, requiring neither the prolixity of the clinical lecture nor the padding of the professional journalist. Year by year time becomes a more important factor, and the call for condensation in literature is imperative. We heartily welcome every attempt in this direction, and in the interest of the readers and of the Journal itself we would urge the necessity of keeping the record of fact, and not the discussing of opinions, as the primary object of the *Review of Reviews*.

News and Notes.

THE PROPOSED INDIAN AND COLONIAL ADDENDUM TO THE BRITISH PHARMACOPŒIA. — The General Medical Council has under consideration a report on the proposed *addendum* to the *Pharmacopœia* of a list of drugs in use in certain British Colonies. Every practitioner in the Tropics is more or less familiar with local remedies of a potency preferable to the marketable commodity of the same drug imported from Europe or America. Moreover, many native or locally employed drugs of a species not mentioned in the European *Pharmacopœia* frequently recommend themselves to European practitioners and are extensively used by them. Hitherto it has been impossible to interest home authorities in such remedies; but, considering the interest which is now being evinced in "things tropical," practitioners in warm climates may hope to obtain a hearing; thereby contributing to the advancement of science and our knowledge of drugs. The Colonial Office has, with the alacrity and energy characteristic of the present *régime*, already collected evidence from many and various sources in this direction.

SANITARY ORGANIZATION IN INDIA.—According to Dr. MacRury, late Sanitary Commissioner, Bombay,

"the sanitary engineering should be done by a *specialist*, not a man who has been digging canals or dumping metal on roads the most of his service, and then sets up as an amateur sanitary engineer. An ordinary civil engineer is no more qualified for the duties of sanitary engineering than a general medical practitioner for that of health officer, without special study and training." "The real want of India is a trained and scientific corps of officers, invested with sufficient power to regulate and amend the willing efforts of judiciously constituted district authorities, and controlled by a board or council at Simla of administrative capacity and skill, and fit representatives of modern sanitary science and engineering."

THE final arrangements for the establishment of the Liverpool School for the Study of Tropical Diseases have now been made. The scheme has met with substantial local support. Mr. Jones' annual grant of £350 has been followed by promises from commercial circles connected with the port of about £900 per annum. The school will be connected with the Royal Southern Hospital and University College. A large ward has been set aside temporarily for the reception of patients suffering from tropical diseases. Connected with the ward is a good laboratory and convenient offices. The hospital annually receives a large number of patients from the docks suffering from the ailments of tropical climates.

INDIAN AND COLONIAL STUDENTS IN EDINBURGH.—At a meeting recently held in Edinburgh, Sir William Muir in the chair, it was decided to recommend a scheme brought forward whereby special residences should be provided for the use of students attending the University from the Colonies and India. The meeting also expressed its sympathy with the work done in this direction in London, and a committee was appointed to act in conjunction with the London Committee in furthering the project. This is as it should be; and we congratulate the Edinburgh authorities upon their forethought in this direction.

Communications, Letters, &c., have been received from:—

A.—Dr. Robert Ashton (Mirzapore).
B.—Dr. F. Burge (Shanghai); Staff-Surg. Ernest E. Bray, Mediterranean Squadron; Dr. John Brock (Mombasa).
C.—Dr. W. H. Crosse (London).
L.—Dr. Ovideo Lemos (Brazil); Dr. A. Lutz (Brazil).
M.—Surg. William E. Marshall, R.N., Mediterranean Station.
N.—Dr. P. Nightingale (Kensington).
P.—Mr. G. Paterson (Edinburgh).
S.—Dr. W. Loudon Shain, (Sao Paulo); Dr. W. H. Stalkartt., R.N. (Portsmouth); Mr. H. J. Spon (London).
W.—Mr. W. White (Lydd).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Pacific Medical Journal.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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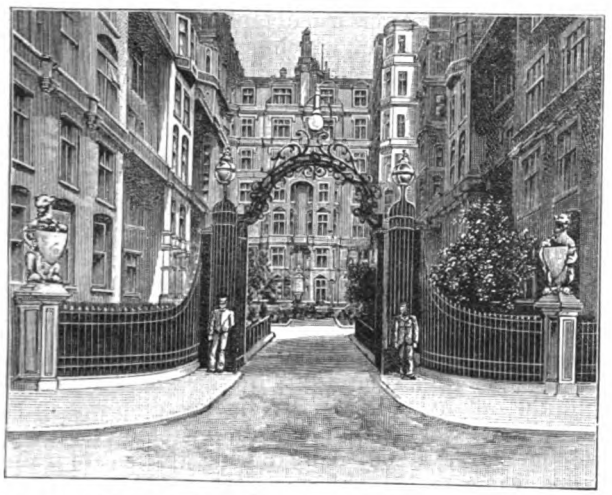

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THE VALUE OF PROPHYLACTIC ISSUE OF CINCHONA PREPARATIONS. AN EXPERIMENT IN INDIAN JAILS.

By Capt. W. J. Buchanan, B.A., M.B., Dip. State Med.
Superintendent Central Jail, Midnapore, Bengal.

There is probably no question in the whole range of tropical pathology of more practical importance than the value of the preparations of quinine in the prevention and treatment of malarial fevers. The following paper is an attempt to collect the opinions of medical officers in civil employ in various parts of India on this subject, especially of those civil surgeons in charge of jails.

For the past three or four years it has been the practice to administer daily, or less frequently, a dose of cinchonidine or quinine to every prisoner in the jails of Bengal and Punjab during the whole of the rainy season, *i.e.*, from, say, June to November. A gigantic experiment of this nature on 18,000 Bengal and 12,000 Punjab prisoners under the special conditions of jail life, seems as if likely to finally solve the question; but for various reasons it has been found practically impossible to obtain the precise experimental proof one desires. In the first place, all prisoners were ordered by Government to receive the prophylactic, this prevented control experiments on any large scale. Again, it so happened that the first two years of the experiment *coincided with* two extremely healthy years (and in spite of cholera and plague, a healthy year in India means a non-malarious one, and *vice-versâ*); the third year of the experiment (1897) was also unfavourable for precise observation, as owing to famine in many districts the health of the prisoners admitted to jails was inferior, and owing to the demands of the military department, a large number of the medical officers of jails (officers of I.M.S. in civil employ) were recalled for service on the frontier, with the result that their jails were left without their superintendence at the time most important for the interests of this experiment.

In spite, however, of these drawbacks, I have received 51 replies to my series of questions, details of which will now be given.

The following were the questions asked, and I have, after each, synopsised the replies received. In some cases all questions were not directly answered, hence the total of replies to individual questions do not correspond to the 51 papers replied to.

(1) What preparation of cinchona used?

To this 40 reply that they have used cinchonidine sulphate and 9 quinine sulphate.

Cinchonidine sulphate is the Paris preparation of that name. It has the appearance of quinine but is much cheaper (4 to 1). It is undoubtedly the best preparation of cinchona (except the quinine salts). It is often confused with, but is different from, cinchona febrifuge and cinchonine, and other cheap preparations of cinchona which have deservedly fallen into disrepute in India.

(2) How does cinchonidine sulphate compare with quinine?

A majority (30 to 20) of the replies are in favour of quinine. None go so far as to say cinchonidine sulphate is superior, but 20 replies say it is of equal value. This, considering its cheapness for use on a large scale, is important testimony in its favour.

(3) The dose of the prophylactic used?

Quinine was used in 3-5-gr. doses (generally 5), and cinchonidine in 6-10, or at times, 15-gr. doses. In 48 out of 51 replies it was given every day, in 2 cases on alternate days, and in 1 case only twice a week. Nearly every medical officer who replied preferred to give it before (rather than after) food, generally in the early morning (at the opening of the jail 35, at midday meal 10, others not stated). In the great majority of cases it was given in solution with sulphuric acid (44 out of 51), with lime juice in 4, in powder 2, in pills none. In 40 cases it was administered alone, in 6 cases with iron (ferri. sulph.), and 4 cases with liq. arsenicalis.

Opinions as to its Value.

In 47 out of 51 replies the verdict was favourable to the prophylactic issue of cinchona preparations. In many cases medical officers stated they preferred quinine to the cinchonidine preparations. In the unfavourable replies the medical officers stated that they could see no appreciable difference in the health of their prisoners in years in which cinchona was issued and the years in which it was not. Others were distinctly of opinion that even when the cinchonidine did not materially lessen the numbers admitted, yet it had an undoubtedly good effect in lessening the severity of the cases which did occur.

One medical officer of many years' jail experience

(Surg.-Lieut.-Col French Mullen) writes: "I think it has proved of decided value and acts as a preventive in regard to sickness generally, including bowel complaints and pneumonia." He also states that cases of severe malarial remittents have been much milder since the use of cinchona sulphate as a prophylactic. The same officer, however, notes that, "Sore tongues" were more frequently present during the years he issued cinchonidine than in former years. This is, as Dr. French Mullen says, a disappointment, for that these cases (often wrongly called scurvy) are due to malarial attacks, is a well-founded belief of Bengal medical officers. Surg.-Major Tull Walsh writes: "Theoretically I hold it to be of value, and I think there have been fewer cases of malarial fever, but the difference is not very marked as shown by hospital statistics." In his Jail Report for 1897, Dr. Walsh writes: "The effect (of anti-periodics) is, I think, good and probably the addition of ginger and sulphuric acid may tend to check bowel disease." I am indebted to Surg.-Lieut.-Col. D. I. Macdonald, senior medical officer of the Penal Settlement in the Andaman Islands, for some interesting statistics. There, something of the nature of a control experiment was made; the results, however, are not decisive. In 10 convict stations in the Andamans with an average daily strength of 4,651 life convicts, the admissions from fever were 132 per cent. with 14 deaths, or a percentage of ·30. In 11 other convict stations, with an average daily strength of 2,752, the admissions from fever were 103 per cent., with 11 deaths, or a death percentage of ·39. In the former cinchonidine was given, in the latter none, in the former while the admission percentage was higher, the death rate was somewhat lower; in the latter the percentage of admissions was lower, and the death percentage somewhat higher. So that we must agree with Dr. Macdonald that no definite conclusion can be made in favour of the cinchonidine issue from these figures.

Let us next see if the provincial jail statistics for the whole province of Bengal prove anything. Here, again, we meet with figures which do not prove much. The following tables give the number of admissions and deaths *per* mille for all jails of Bengal Province (*i.e.*, Lower Bengal, Behar and Orissa), for past six years; the figures for "malarial fevers," "diarrhœa and dysentery," and "all causes," are only here quoted.

Year	Malarial Fevers		Diarrhœa and Dysentery		Deaths from all Causes	
	Admission Per	Death mille	Admission Per	Deaths mille		
1896	292	3·6	332	8·3	28·7	Prophylactic used.
1895	423	1·5	364	9·9	27·3	
1894	406	1·6	379	19·	47·1	Prophylactic *not* used.
1893	409	1·4	272	12	32·3	
1892	372	1·6	329	11·	43·7	
1891	447	2·2	292	8·8	30·9	

It will be seen that it is impossible to draw any certain conclusion from these figures. Those for 1895-6 which appear favourable, are after all, little better than the (exceptionally healthy) year 1891, when the prophylactic issue of quinine was not in use. The fact is, the number of deaths from "malarial fevers" is always low, that is, there are hundreds of mild attacks which are recovered from, and when a patient dies his death is usually due either to dysentery or pneumonia, both of which diseases, without being malarial, are very strongly predisposed to and rendered more severe and dangerous to life by previous repeated attacks of malaria.

Failing therefore to get more than a general impression in favour of prophylactic issue of cinchonidine from either the replies of medical officers or from reported statistics, I may be permitted to give some personal experiences.

In the beginning of 1895 I took charge of the large Central Jail at Bhagalpur. The previous year (1894) this jail (as in all other Bengal jails) had attained to the bad eminence of being the most unhealthy on record (viz., 48 per mille deaths). In the years 1895 and 1896 the prophylactic issue of cinchonidine was introduced and most carefully administered to every prisoner (over 1,200) daily during the entire rainy season, June to November, *post hoc*, but not necessarily *propter hoc*, I had a jail death rate of only 9 in 1895, and only 7·2 in 1896, both far and away the best on record for that jail. Again, in spite of a most malarious and famine year, the death rate of Buxar Central Jail, to which I was transferred in 1897, was only 13· per mille, also a best on record. But it would be a mistake to suppose that the marvellous improvement in the health of these jails in these three years was due to the prophylactic *alone*. In the first place the years 1895 and 1896 were dry years and the public health was exceptionally good, though far from proportionately with the jail. Again, numerous sanitary improvements were carried out during those two years, which helped in a like direction, yet nevertheless, *a fall of over* 40 *per mille in the death rate is a remarkable and exceptional fact*, and must in part be attributed to the use of the prophylactic. My experience in Buxar Jail last year (1897) is even more to the point. The full figures for 1897 have not yet been published, but the year was one of heavy rain over all the province of Bengal, which, as is usual, produced in most places, along with good agricultural crops, a plentiful crop of malaria. Buxar town and the railway cantonment suffered especially (the ground water in parts is very high during the rains). From some rough statistics I prepared I was able to show that the unprotected town and railway communities suffered from "fever" more than three times as much as the protected jail prisoners. Many outside people had eight or nine attacks of fever during the four months (August to November), whereas it was the exception for the prisoner (average daily strength 1,180) to have more than one attack each. Nevertheless some 600 cases of fever came to the jail hospital during the season, but in every case the fever was mild, was seldom recurrent and never lasted for more than one or two paroxysms. Moreover, towards the end of the season, I personally made an examination of some 600 prisoners *and found only* 2 *per cent. had spleens*, which could be felt on gentle pressure below the ribs (a slight degree of enlargement which is

generally neglected in India). Again, there were extremely few cases of sore mouth and blue black gums, such as we were accustomed to see in the pre-prophylactic days, and bowel complaints were very largely absent, in fact, conspicuous only by their rarity. In fact, in the opinion of some medical officers this lessening of bowel complaints, which has been most marked in many of our largest Bengal jails, is one of the most satisfactory results of the prophylactic issue of quinine, &c. It cannot, however, be put down to the quinine alone, as in nearly every case quinine was issued along with dilute sulphuric acid, and in several jails along with iron or with ginger powder. The sulphuric acid has another advantage, viz., that it is a well reputed prophylactic against cholera; but we cannot attribute the comparative absence of cholera from our jails to this, as no great epidemic has existed in Bengal for past three years, nor have sporadic cases been very prevalent. It is, however, worth bearing in mind that, by the issue of quinine with sulphuric acid, we, as it were, "kill two birds with one stone," or protect against two diseases. One minor point is also established by the series of replies on which this paper is based, viz., that the prophylactic issue of cinchonidine in no way interferes with the use of quinine in the treatment of cases of actual fever.

Another point on which some difference of opinion exists is, whether the prophylactic should be issued in moderate doses or less frequently in large doses. This is often a matter of convenience only, but in a prison where its administration is easy, I prefer to give it daily. Theoretical considerations point the same way, as quinine is excreted in about thirty-six to forty-eight hours, it is obvious that if given at greater intervals than this there will be times when none is in the blood, hence I prefer the daily moderate dose.

Conclusions.

It will appear from above that the opinion of the fifty-one medical officers in India who replied to my queries is on the whole favourable to the prophylactic issue of quinine or cinchonidine. That the results are not more marked is due to many considerations, viz., a somewhat changing population (in small jails with short term prisoners at least) and local considerations of climate and season. It is also more than probable that the results would have been better marked if quinine had been given in place of cinchonidine. In India this prophylactic has been tried against the æstivo-autumnal fevers, it has been very little tried in cases of spring fevers, indeed in my experience the fevers we get in the plains of Northern India in the dry hot months are not malarial but due to exposure, heat, sun, &c. I have certainly found the hæmatozoa much less frequently in the spring months than in the rainy season, from August to end of October. Personally the result of my three years' experience of this prophylactic on a large scale (not less than 1,200 prisoners daily) is to make me enthusiastically in favour of it and I believe the more thoroughly it is carried out the greater will be the benefit to the persons taking it. In the ensuing season I shall try the effects of still larger doses.

Paper read at the Annual Meeting of the British Medical Association.

AN ACCOUNT OF THE MEASURES TAKEN TO CONTROL THE EPIDEMIC OF PLAGUE IN THE CITY OF BOMBAY DURING THE YEARS 1897-98.

By Major H. P. Dimmock, M.R.C.S., L.R.C.P.Lond.,
Indian Medical Service.

(*Continued from p.* 190.)

The second group of cases with blood toxæmic symptoms are analogous to cases of septicæmia and are due to direct infection of the blood stream. These are the cases in which the bacilli are detected in the blood in the earliest stages of the disease, while in a large number of those of the bubonic type, examination of the blood has given negative results in the earlier stages, because the bacilli are arrested by the lymphatic glands.

In the later stages bacilli can be detected, as they will have developed in such numbers as to pass into the blood; in all persons dead of plague, bacilli are found in the blood. The general symptoms of the two groups are thus somewhat different. In the first group, or bubonic cases, the bacilli are arrested at the successive clusters of glands where they incubate and manufacture the toxins which are gradually poured into the blood stream. These cases pass through the regular sequence of symptoms according to the virulence of the poison absorbed.

In the second group of cases the bacilli pass at once into the blood, in which they develop enormously and manufacture the toxin directly in that fluid; they are consequently the severest type of cases, often proving fatal within a few hours with all the symptoms of acute toxæmia.

I have also seen cases of this kind in a state of collapse resembling that of cholera. These cases usually die before any glands become affected, but if they survive a few days the glands of the whole body may become enlarged, closely resembling cases of acute septicæmia, and this view was communicated by me to the Director General I.M.S. in November, 1896.

The third group of cases, called the pneumonic, from the primary infection by the microbe taking place in the lungs, are also generally fatal. The symptoms are pneumonia of a lobular type with profuse sero-sanguineous expectoration and the general symptoms of plague. The expectoration holds a perfect culture of the bacilli, which can be detected at once and in enormous numbers. These cases are most intensely infectious. The excreta and secretions of all persons and animals suffering from plague are probably acutely infectious shortly before death, and this is a great argument for the early removal of cases to hospital before they can infect the room or house; and even if the case is a hopeless one, it is a grave wrong to the living to allow them to remain in the same dwelling with people who are not suffering from the disease.

All persons suffering from plague should be removed to an isolated plague hospital, and those persons who have attended upon the cases or dwelt in the same room or house with them should be removed to

isolated quarters; the room and house on being evacuated should be given up to a thorough process of cleansing and disinfection. Infected bedding and clothing should be burned.

Simple as these instructions are in the reading, the application of them was attended with great and many difficulties. The wide reaching effect of the plague epidemic in Bombay and its interference with the trade of that great port which has large interests in the commerce of the whole world, raised questions of imperial and international importance. The Government therefore decided to take over the direction of plague operations in Bombay and accordingly appointed a Committee consisting of four members, one of the members, General Gatacre, C.B., D.S.O., being the Chairman, whose genius, energy and many fine qualities are well known to the English people. The other members were Mr. Snow, I.C.S. (the Municipal Commissioner), Mr. James, A.M.I.C.E., (a civil engineer on the Municipal Staff), and myself. The duties of the Chairman were by no means of a medical nature (a misunderstanding on this point has arisen in the minds of many medical men), but required administrative and political abilities of the highest order, to organise the many different measures and to apply them to the large city of Bombay, teeming with a crowded population of many races, castes and creeds, who are imbued with fatalistic and fanatical ideas and easily led by misrepresentation. The other members of the Committee were required to work under the direction of the Chairman and to exercise supervision in financial, engineering and medical arrangements respectively. This supervision required the frequent personal direction of the Committee in every part of the island of Bombay, so that they were continually moving about at all hours of the day and night. In consequence of the resistance of the people to the measures proposed and to the concealment of plague cases, careful search for these had to be made. To supply the necessary staff a certain proportion of the British and native military forces in Bombay, as well as a large police force, were placed at the disposal of the Committee. They were utilised for house visitation parties, as clerks, storekeepers, ward masters, dressers, orderlies, and for ambulance work. A large proportion of the municipal staff was also placed at the disposal of the Committee, and included health inspectors, sub-inspectors, muccadums (head men of gangs), conservancy staff, &c. The medical services provided a number of medical men of higher and lower grades to fill the various posts, and these were supplemented by the engagement of numbers of native practitioners and senior students. The Justices of the Peace and the leaders of communities also were invited to help; they cordially gave a great deal of valuable time and energy to the cause as volunteers. On General Gatacre's departure for England on duty, his office as Chairman was filled by Sir James Campbell, K.C.I.E., Indian Civil Service, who possessed great ability as an administrator and had intimate knowledge of the different castes and people in Bombay.

When the work became very heavy other members were added to the Committee, and the supervision was thus divided up. The Surgeon-General with the Government of Bombay, also took office on the Committee.

FIRST SCHEME OF PLAGUE OPERATIONS, MARCH, 1897.

Chairman of Committee—BRIGADIER-GENERAL GATACRE, C.B., D.S.O.

I.—Formation of a Central Office for the Plague Committee with Secretaries and Clerical Staff, stores and other departments, and having telephonic communication to all parts of the city and with the various district offices of the Committee.

II.—Division of the island into ten districts, according to the number of houses and the density of the population. Each district was placed in charge of a medical officer who was styled "District Medical Officer."

III.—The selection of sites for Government hospitals, fifteen in number, with due regard to districts and population.

IV.—Detailed construction of Government hospitals, fifteen in number, organisation and staff. Standard unit—one section hospital for twenty beds, area 120 by 24, superficial area for each bed, 144 sq. feet. Standard equipment according to calculation. Standard staff for unit—1 hospital assistant, 2 nurses, 1 ayah, 4 ward orderlies, 1 water carrier, 2 sweepers (scavengers), 2 nurse's servants, 1 hospital cook. These hospitals were mostly temporary buildings of bamboo, matting and palm leaves, lime washed frequently.

Three of them, viz., Old Government House Parel, Arthur Road Hospital and Grant Road Hospital were permanent buildings. The Parel Government House Hospital was instituted and organised for Government by General Gatacre and Surgeon-Captain Thompson, before the Plague Committee was appointed. All the additional hospitals were in working order in three weeks' time. Twelve segregation quarters for families of patients were attached to each hospital.

V.—The selection of caste or community hospitals was made to meet the scruples of different sects. They were under the control of the District Medical Officers but the sect supplied all staff, arrangement and equipment. Munificent native gentlemen cordially aided the movement. The hospitals were generally carefully selected houses in the town.

VI.—The organisation of each district. A District Medical officer in charge. Sub-divisional medical officers as required to each sub-division of the district, usually two or three. The following medical officers were obtained to take charge of the districts:—Surgeon-Lieut.-Col. H. Hamilton, I.M.S.; Surgeon-Major H. G. Deane, A.M.S.; Surgeon-Major J. C. Culling, A.M.S.; Surgeon-Captain Jennings, I.M.S.; Surgeon-Captain Brogden, A.M.S.; Surgeon-Lieut. H. A. L. Howell, A.M.S.; Surgeon-Lieut. C. R. Morgan, A.M.S.; Surgeon, R. S. Bernard, R.N.

Each District Officer had a central office, with all requirements and a telephonic communication all over the town. His duties were:—

(1) To superintend and exercise sanitary supervision of the whole district in their charge, reporting to the Plague Committee and Health Officer daily.

(2) To supervise the working of the district hospitals and to attend the same in routine.

(3) To supervise the work of the search parties in conjunction with the Justices of the Peace.

(4) To control the disinfection of the infected areas and buildings.

(5) To inspect all segregation camps in their districts.

(6) To call the attention at once of the officer appointed by Government for the condemnation of insanitary buildings to any such premises in their districts.

(7) To report on registration of burials and cremations in their districts and to supervise sanitary condition of cemeteries.

(8) To send daily reports to the Plague Committee and Health Officer of casualities occurring during twenty-four hours preceding 6 a.m., with details of—

(*a*) What has transpired during the preceding twenty-four hours in their districts.

(*b*) Any suggestions they have to offer.

Staff for Search Parties.

A Justice of the Peace, a Medical Officer or an influential citizen in charge as conductor of search party, a sub-inspector (European usually), 3 military Sepoys, 2 police Sepoys, a locksmith, an ambulance with Sepoys, a lady doctor if required (military cordons and large search parties were organised and resorted to occasionally), disinfecting staff as required to cleanse, disinfect, and whitewash the room or house as soon as the case was removed.

Process of Disinfection.

(1) Immediately a patient is removed from the room, the disinfecting staff should be ready and brought into operation.

(2) All rags, bedding, clothing of the patient and rubbish generally, should be carefully lifted up and removed and burned outside the building. In placing the articles outside, they should be carefully laid down so as not to raise the dust.

(3) No brushing of walls or floor should take place; this is a most dangerous proceeding and is calculated to spread infection.

(4) The first work in all instances is to flood the floor with a solution of perchloride of mercury not weaker than 1 in 1000; the junctions of floor and walls and all corners should then be mopped with the solution as well as the wall, as far as the mop will reach, and above this a small hand pump should be used; the floor, if made of earth, should then be dug up to a depth of 4 in.

(5) All furniture that can be dealt with should be likewise disinfected with perchloride of mercury solution, either with a pump or with a cloth dipped in the solution.

(6) After the above work has been thoroughly done and the solution has dried, quick lime in a hot state, and in as strong a solution as possible, should be laid on all the walls, floor and ceiling.

(7) In event of the whole house requiring disinfection, the privies should be attended to first, not forgetting the shafts, then the staircases and corridors should be operated upon; lastly, the rooms in order, first by washing every part with perchloride of mercury solution, and after twenty-four hours laying on the quicklime as described in paragraphs 4 and 6.

(8) All bathing places and the drain pipes from them should be carefully disinfected by flooding them with perchloride of mercury solution, and, when necessary, they should be altogether removed and replaced with new ones at the cost of the owner of the premises.

VII.—Land traffic inspection, inward and outward. Two railways, G. I. P., and B. B. and C. I.; two roads, Sion Causeway, Bandora Causeway. Staff: one superintendent of railway inspection—Surgeon-Major A. F. Street, D.S.O., I.M.S.; afterwards Surgeon-Captain Jennings, I.M.S. Inspecting staff at different stations as required; two on the railways at junctions about 40 miles away from Bombay.

VIII.—Inward sea traffic: Inspection and Observation Camps.—(Plague had spread to many places along the coast, and the people were returning to Bombay, where the epidemic was gradually diminishing).

Coasting steamers were inspected at three landing places. Native crafts at five barges anchored at different stations in the harbour and at points on the island shore.

The whole staff and arrangements were placed under the control of the health officer of the port, Surgeon-Major MacCartie, C.I.E., I.M.S.

Observation Camps.

Five in number were erected at intervals in the vicinity of the docks. Suspected persons were detained and sent to a camp. 263,289 persons were inspected, 4,835 persons sent to camp, 57 actual cases of plague detected. The arrival of these immigrants was a danger and a great difficulty. The camps had to be arranged to meet the scruples of many different races and castes.

IX.—In addition to these measures the committee organised plague operations at places away from Bombay; the district of Kolaba, the islands on the other side of the harbour, and Cutch Mandvie in Kattywar had trained medical, nursing, hospital and disinfecting staffs sent to them.

Disinfecting staffs were also sent to other parts of the Presidency and ambulances were dispatched up country.

SECOND SCHEME OF PLAGUE OPERATIONS, JULY, 1897, TO APRIL 30, 1898.

Chairman, SIR JAMES MACNABB CAMPBELL, K.C.I.E., I.C.S.

The epidemic had waned, and on the 11th July the returns shewed :—

Plague attacks	...	...	0
Plague deaths	...	...	0

The monsoon set in early in June. Famine having ravaged the country, many of the famine stricken began to come in from the country to the city. Cholera appeared, and in the first week of August 220 deaths from this disease occurred, after which it gradually decreased. Relapsing fever was also epidemic. A large proportion of the medical staff was removed to supply the needs of the Army, and

the committee had to fill their places with men who had not the requisite special training, and a number of medical men and nurses were brought out from England to further strengthen the staff. The basis of the original organisation was maintained and plague operations were conducted as usual. Some of the Justices of the Peace had left the city or ceased working, and their places were supplied by increasing the medical sub-divisional staff. This arrangement was found to work more expeditiously and was continued. At the request of the Municipal Commissioner, the districts were re-arranged to coincide with the municipal division of the island into wards, and the district medical officers were redistributed accordingly. The plague was slightly recrudescent, but its progress was not very marked.

July.	Imported cases	10.	Total 52.
August	,,	,, 9.	,, 65.
September	,,	,, 25.	,, 155.
October	,,	,, 29.	,, 207.

The city was gradually filling with a returning population, and in December had a total population of 800,000. Further measures were adopted to grapple with the danger of another epidemic, which threatened to be most serious with a full population, and the resulting damage to trade.

These were:—

(1) The appointment of civil and military officers of tried ability and experience as district officers. This was to provide for the deficiency of trained district medical officers.

(2) The institution of sea quarantine. Government declared which ports were infected and persons arriving from these ports were placed in quarantine. This necessitated the erection and equipment with staff of large quarantine camps.

(3) The institution of land quarantine for the detention of all persons coming from infected places by road or rail. The word "quarantine" was discontinued and the camps were called *detention camps*.

To provide for the better classes of people, surveillance tickets were issued on good surety to those who agreed to present themselves at the camps for ten days for medical examination.

(4) Contact camps for the segregation of "contacts," *i.e.*, those persons who had been in the same room with or in attendance on plague cases.

(5) The vacation and thorough cleansing of infected houses and quarters of the city. A number of sailors from the R.N. squadron were employed for this work. The houses were at the same time ventilated and improved in every possible way by a company of sappers engaged for the purpose.

(6) The formation of health camps both public and private was executed on the same lines as the public and private hospitals, for the reception of "evicts" or persons from vacated houses.

(7) Extensive measures were taken for the cleanliness of persons and for the disinfection of clothing at all camps. Disinfecting tanks and steam sterilisers were used for these purposes.

(8) At this time Government had instituted detention measures of a similar description at all towns and cities in the Presidency, and had several large detention camps on the lines of rail and road, so that the Bombay population was practically isolated, and as the mortality among them began to rise to an extent apparently, though not proportionately, as great as in the epidemic of 1896-97, a feeling of alarm and unrest pervaded the whole city.

(9) A more accurate system of registration and notification of the cause of death was arranged for in consultation with the heads of the different castes and religions, but it was unpopular with the lower orders, and did not meet with great success at first. It is, I believe, being gradually brought into force.

(10) In consequence of the increased unpopularity of plague measures and subsequent to the riots, the leaders of the different races and castes were called upon to form themselves into local volunteer committees for carrying out the measures of the official staff.

With these extensions and modifications the plague operations were ordered on the same lines as previously.

TWO CASES OF POISONING.

By G. F. Reynalds, M.R.C.S., L.R.C.P.
Taqual, Gold Coast, West Africa.

I. By Calabar Bean.

W. X., a Krooboy, aged 32 (*circ.*), whom I had often seen and knew to be a strong, stoutly-built man, was reported to me to be away from his work, and was said to be ill in his village, and as he had expressed a wish to see me, I had him brought up to my quarters. I saw the patient on December 12, 1897; he had then been ill for nearly a week, and his condition was as follows:—patient looks extremely ill and is very weak; cheeks are sunken and pinched; eyes are sunken in their sockets but of a markedly bright, glassy lustre; pupils are small and fixed, do not react to light or accommodation; distant vision is fairly good, near vision is blurred, and the man gropes his way about with one hand outstretched in front of him, like a blind person; complains of dizziness and great mental confusion, makes motions with his hands indicating that he has a whirling sensation within his head; has vomited several times during the past three days, but not to-day; tongue is broad, flabby and tremulous, with a thick dirty-yellow fur all over the dorsum; appetite very bad; some amount of thirst, but not at all distressing. Temperature in axilla 98°; pulse 90, feeble, small volume, irregular in time and force; heart-sounds are weak but clear, no heart bruit; respirations 22, full and apparently normal; lungs, liver and spleen, normal; no difficulty in talking or swallowing; abdomen slightly concave, not painful or tender to firm even pressure, except for a slight amount of tenderness in the epigastrium; bowels costive; examination of the urine revealed nothing abnormal; limbs look shrunken and are much wasted; great loss of power in the arms and legs; both grasps feeble; knee jerks absent, the gait is shambling and the feet are dragged along the ground; is able to stand with the eyes open, but when told to close the eyes he

begins to sway about at once and would fall; as far as I could ascertain his sensory impressions were only very slightly impaired; there was no deafness or loss of smell. I came to the conclusion that I had to deal with a case of calabar bean poisoning, and subsequent investigation proved this to be so. The man had had some trouble with a woman, and her husband had mixed small quantities of pulverised calabar bean in the patient's rice after it had been prepared for him to eat. On the day I saw the patient I gave him a hypodermic injection of sulphate of atropine gr. $\frac{1}{60}$, this I repeated eight hours later; the same night I gave him 10 grains of calomel, and the bowels were well relieved early next morning; this was the only medicinal treatment employed. I had the man isolated and well guarded. His food at first consisted of arrowroot, and later of soup, fowl and bread twice a day, and at each meal I allowed him three ounces of brandy in a glassful of water; he rapidly improved, and resumed his work in eleven days' time. Calabar beans grow and are very plentiful in this district; one can frequently see pods full of the beans lying about the roads. The poisonous properties of the bean are fully known to the natives, with whom, as a poison, it is a great favourite, being employed for this purpose as often as sasswood, if not more so.

II. By Dynamite.

Y. Z., a Bassa man, aged 30 (*circ.*), had an attack of gonorrhœa for which he went to a native doctor, who gave him medicine sufficient to fill two large gin flasks, each holding about fifty fluid ounces; this medicine was of a clear yellow-colour, looking like tincture of quassia and having no sediment. The patient took one flaskful with him and left it lying about whilst he was at his work; the other flask he locked up in his house. On the second day of taking the medicine he noticed that the colour had changed and that the liquid was thick and turbid; ascribing the change to fetish he went on taking the medicine till he had had four more doses, when he began to feel dizzy and weak, and vomited once or twice. He was then informed that a fellow countryman had put some dynamite into his medicine with the intention of killing him, on account of some family quarrel. I saw the patient on September 30, 1898, the day after the medicine had been tampered with; he came over to see me bringing his two flasks with him and his condition was as follows:—patient is uncertain in his gait, staggers about and is unable to walk by himself, although he can stand alone for a short time; complains of intense headache and much dizziness; is not able to talk much, hesitates and seems confused; temperature in axilla 100·2°; pulse 48, full volume, soft and regular; heart beat at apex forcible and rapid; nothing abnormal heard on auscultation of heart and lungs. Had taken one dose of the altered medicine this morning and three doses yesterday; vomited this morning after drinking the mixture. Has no appetite at all but great thirst; tongue flabby and tremulous, with a thin white fur all over the dorsum; hands tremulous, cannot lift anything to his mouth with certainty, but has to be guided; knee jerks slightly diminished; pupils react to light and accommodation; passes urine in normal quantity, no albumen, blood, sugar or bile present; bowels costive; the gonorrhœa was very slight, probably a simple urethritis only. I gave the patient calomel gr. x., and a mixture containing liquor strychninæ ♏ vij., tr. digitalis ♏ x., infus. gentianæ comp. ad f. ʒ ss., thrice daily. In three days' time he appeared to be quite recovered, and resumed his usual work. On comparing the two flasks of medicine, the one that had been left in the house was clear as described above, the other (containing about twenty ounces) had three-fourths of an inch of dark brown sediment; filtered, the wet sediment was of an indian red colour; washed with water it was seen to consist largely of fine sawdust with many small white specks scattered about; when dried the sediment, unwashed, was of a light pink colour, looking like calamine. From the quantity of the medicine I examined (four ounces) I separated eighteen minims of nitro-glycerine, and probably some more nitro-glycerine was dissolved in the filtered liquid: the patient's ordinary dose of this medicine, as shown to me, was about three and a half fluid ounces. I am informed that the dynamite used here consists of about 25 per cent. of nitro-glycerine, and 75 per cent. of a composition largely made up of fine sawdust with silica or clay. The man who had put the dynamite in the medicine bore a bad reputation and had once killed a man, so he told me, by putting dynamite into some gin, which the victim drank with fatal results.

NOTES FROM LAGOS, WEST AFRICA.

By Henry Strachan, L.R.C.P., M.R.C.S.
Chief Medical Officer.

I.—*Tubercle in Ships.*

The first note is made not in Lagos, but *en route* thither from Liverpool. It is to draw attention to the risk run by passengers to the West African colonies of contracting tubercle on the voyage, on board the ships conveying them. So many poor sufferers from phthisis form the majority of the passengers to Madeira and the Canaries at certain times of the year, that the probabilities are great that most cabins will more or less be infected with tubercle, and it is by no means unlikely that a passenger will find him or herself cabin-mate with some *poitrinaire* whose expectoration is profuse and frequent, and whose cough is, at night, most distressing.

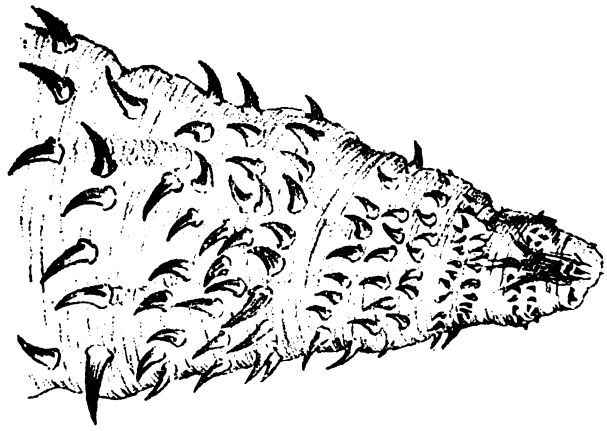
This is not as it should be in these days when all are agitating for the establishment of means for preventing the spread of what is acknowledged to be a communicable disease. Cabins should be reserved for the use of invalids suffering from "chest complaints," or if this be not possible, all cabins should be disinfected at the end of each voyage, and the bedding of the berths, or at least the pillows, renewed. In the meantime, I advise every traveller to take his own pillows with him.

II.—*Larvæ in Human Skin.*

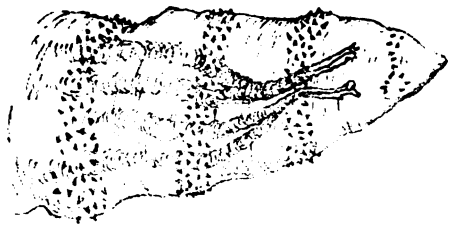
The deposit of ova by insects, generally dipterous, in the human skin is a well-known phenomenon in

tropical countries, and here one often finds that so-called "boils" have been caused in this way. I have not identified all the flies that have this disagreeable trick (for it must be regarded, I think, as a mistaken choice of food supply, not an intentional attack on a human being), and the larvæ vary. The one of which I give drawings causes twinges of acute pain every now and then, explained by the fact that the anterior portion of the body is covered with, on each segment, rows of recurved large chitinous spines. At the tail-

LARVA OF FLY (hatched in human skin, West Africa). (Highly magnified.)



Anterior end, with strong, large spines.



Posterior end, showing orifices of tracheæ and very small spines.

end open the orifices of the four main tracheæ, which are bound into two just before they reach the stigmatic orifices. This tail-end lies at the bottom of a minute hole in the centre of the "boil," and enables the larva to breathe. A drop or two of cocaine and sharp knife constitute the treatment as regards essentials. I append a drawing of the head and tail-ends of such a larva.

III.—*Discovery of Existence of Ankylostomiasis in Lagos.*

There is marked mortality among the natives from "dropsy" and "anæmia." On seeing some of the cases so diagnosed I was struck with the resemblance to the ankylostomiasis of the West Indies, and on investigation the presence of the causative parasite was at once demonstrated.

The usual thymol treatment, which I for many years found efficacious in Jamaica, has proved of equal value here, and the recovery of the cases, especially children, is usually rapid. Of course the re-infection after leaving hospital is as certain, as I found in the above-named Colony. If it is hard to convince the Jamaican of the necessity for boiling his drinking water, it is impossible here. Besides the ankylostomum duodenale, other intestinal parasites, *e.g.*, ascaris lumbricoides, oxyuris vermicularis and trichocephalus dispar infest nearly everyone.

IV.—*Water as a Means of Conveying Causes of Intestinal Diseases in West Africa.*

Every one I was going to write, and do not think I shall be far wrong if I do write it, is more or less affected with some form of intestinal trouble. For even the European residents are not all free, though they do use filters (so-called). I have strenously advocated the boiling of the water in all households, and understand that it is more the practice now than was formerly the case. But by fruit and raw vegetables the ova may be conveyed to the human host, as the water used for washing them is in the vast majority of cases unboiled, even when the drinking water is carefully attended to by prudent persons.

The introduction of the causative poisons of dysentery and other intestinal diseases by this means is not to be forgotten. The well and tank water are both often found to contain fine, almost microscopic, crystals of some micaceous or granitic rock, and the irritation they might cause may be perhaps to some extent responsible for the gastro-intestinal irritability and catarrh so prevalent even in cases where more potent and popular concomitants of liquid refreshment are not indulged in.

The consequence of the unsatisfactory nature of the water supply is that all who can, drink only imported aerated waters. I have no doubt that those who do so habitually and invariably, escape almost entirely much of the intestinal disease which is equally responsible, in my opinion, with malaria, for the terrible amount of illness and death for which this part of the world has, alas, a merited reputation.

V.—*Native Mortality.*

When I mention that the mortality among natives in Lagostown, of about 35,000 inhabitants, sometimes a little more or less, owing to the tidal flow of traders from and to the interior, reaches the awful figure of 72 per thousand per annum, I think it will be seen that the idea that the natives here enjoy good health is baseless.

The chief causes of this death-rate appear to be intestinal diseases, fever (malaria), dropsy and anæmia (? ankylostomiasis), and among the Krüs and others, lung trouble during the rainy season. And no doubt the infant mortality, great as it is, is not all on record. Suckling for three years, bad feeding and superstition, are the main factors bringing about the great loss of infant life.

VI.—*Dr. Koch on Quinine.*

No one, I think, who is engaged in combating malaria in West Africa but must regret that the remarks made by this eminent and greatly respected authority should be discussed in lay papers, or that a matter on which there is still so much room for difference of opinion and farther investigation should appear to be at once settled by an *ex-cathedra* statement. Here already the public is divided into a quinine and anti-quinine section. Each person

decides for himself, though absolutely ignorant of medicine, the very important question as to whether or not he will take quinine "lest he should change his ordinary fever into black-water fever!" Surely the doctor in West Africa has a sufficiently difficult task already without this burden being laid on him!

That quinine is abused as well as wisely used in Africa there can be no doubt. Some persons habitually *drug* themselves with it, others take it whenever they fancy they have cause, and the doses are sometimes appalling in size!

In sixteen years' experience of the treatment of malarial fevers I have not found the need for large doses of quinine except when given *per rectum*. I have hardly ever given a larger dose than 10 grains at a time, and the usual one is 5 to 6 grains repeated every fifth or sixth hour.

A small occasional dose of phenacetine helps the patient by relieving head-ache and general malaise, but I do not believe in large doses of it, except where hyperpyrexia is an immediate danger to life. And I cannot too strongly condemn the way in which antipyrine and phenacetine—drugs which have so marked an effect on the circulatory system and on hæmoglobin—are taken by the public without medical authority. I must acknowledge at the same time that I have come to the belief that phenacetine and antipyrine are too much used as *routine* treatment by ourselves, and too much reliance placed on them, because the subjective condition of the patient is usually so rapidly and so markedly improved; while all the time the actual cause of the illness is "scotched, not killed." Quinine, until a better drug is found, must still be considered our mainstay in the treatment of malarial fevers, whether or not the malaria parasites differ from one another in different parts of the world. The clinical aspect of the disease they cause appears to me to be certainly identical in the West Indies and in West Africa, and so far as reading would teach me, in Italy, America and other regions, quinine is at present *the* drug that destroys them.

VII.—*Black-Water Fever.*

Whether black-water fever is or is not a malarial fever I am not prepared from my own observation to say at present. It occurs in persons who have not taken quinine before the attack most certainly, and equally certainly great numbers of habitual quinine takers have escaped it. In the West Indies I have not seen one case in fifteen years, while thousands of patients have taken quinine, the quantities amounting sometimes to drachms, or even ounces (of course in proper doses spread over lengthened periods of time). I find it therefore very hard to believe that the disease is caused by *quinine*, as is so emphatically asserted by some.

I must also say that some cases have recovered when quinine has formed no part of the drug treatment. But I hope to deal at greater length with this matter in subsequent notes.

SOME CAUSES OF DEATH IN REMITTENT MALARIAL FEVER.

By W. E. DE KORTE, M.B. (Lond)., *Graaff Reinet, Cape Colony.*

THE various causes of death enumerated will be understood to apply to that form of intermittent fever found in the South African Karroo. The plasmodium of this form of malaria closely resembles in appearance that of the so-called æstivo-autumnal fevers.

I have not found the parasite pigmented, but the contained granules have a slightly opalescent, greenish tinge. Laveran's corpuscles can be detected after allowing the blood to incubate in a wet chamber for three quarters of an hour, but as yet flagellated forms have not been met with.

(1) *Perforation of the Gut.*

This may occur at any time after the seventh day from the onset of the fever. The invasion is usually severe in such cases as terminate in perforation of the intestine.

Vomiting of an astonishing amount of bile along with bilious diarrhœa are among the first symptoms. A remission of the temperature in the evening instead of in the morning, as ordinarily happens, may warn one that the case is going to be of exceptional severity. In such cases it is apparent from the first that the chief onslaught of the disease is on the gastro-intestinal tract. The liver and spleen are usually painful on pressure, the abdomen is distended with flatus, and there is pain at the epigastrium. Further developments are ushered in by diarrhœa starting afresh, for after the primary bilious diarrhœa there is usually constipation. The looseness of the bowels gradually increases, and then without further warning the patient becomes suddenly collapsed, and may die at once from syncope due to the shock of the perforation, or, should he rally, succumbs to peritonitis. I have some reason to think that the accumulation of the plasmodia malariæ in the capillaries of the intestinal mucosa may be so intense at one spot as to lead to thrombosis in that area, and so lead to perforation, and falling short of this, would explain the concomitant physical signs of enteritis.

But perforation of the gut may be brought about in another way. In every case of malaria there is a certain amount of gastro-enteritis, and perforation may occur in an otherwise favourable case by injudicious feeding, such as meat given through ignorance.

(2) *Hæmorrhage from the Bowel.*

There can be little doubt that the presence of the malarial parasite profoundly alters the blood. That a destruction of the red-blood corpuscles occurs is shown by the pallor which so soon supervenes in malaria and the melanæmic condition of blood itself. Hæmorrhage from the nose, petechiæ in the skin, and hæmatemesis are by no means rare occurrences in cases of malaria, and we can only assume that such hæmorrhagic tendency is due to the hydræmic condition of the blood. In some cases of hæmorrhage from the gut there is general oozing from the capillaries all along the bowel. The blood vessels do not seem able to contain their contents. In other cases I have suspected that, although warned to the

contrary, some solid food was given. I consider, therefore, that hæmorrhage from the bowel may be brought about by injudicious feeding, and this more probably at a late stage of the disease, when the bowel is rendered less resistant by the accompanying enteritis. In some cases hæmorrhage from the bowel is so fast and furious as to make it certain that bleeding occurs from a vessel of such size as could hardly be occluded by the malarial parasite. In such cases I believe that the blood vessel is opened by an ulcerative process comparable to that which occurs in typhoid fever, and one which is induced by the presence of the plasmodium malariæ in the intestinal mucosa. No opportunity of an autopsy having occurred, positive proof of this theory is wanting. Of the various drugs tried to control intestinal bleeding of this sort, turpentine has proved the most effectual.

(3) *Coma with or without Hyperpyrexia.*

It is in such cases that the great advantage of the hypodermic method of giving quinine is abundantly shown, for the patient is usually unable to swallow, and the administration of quinine by the rectum is but a tardy method when there is no time to be lost. Five grains of the hydrochloro-sulphate of quinine injected hypodermically, and repeated if necessary, will convert an otherwise hopeless case into one of recovery.

In a considerable proportion of cases of any severity, the prognosis can be based on the somnolency of the patient. A drowsy patient will probably make a good recovery, and conversely, restlessness is of bad augury. It is interesting to note that a certain amount of quiet delirium during sleep is generally a favourable symptom. It follows from this that coma looked upon as a degree of unconsciousness is difficult of explanation, if considered as dependent on the toxicity of the blood, for cases in which the blood is evidently so profoundly altered as to lead to local gangrenes, coma is not necessarily present, even though the temperature be high; nor can the hyperpyrexia explain the coma, for we may have coma with a temperature no higher than 103° F. The phenomena of hyperpyrexia and coma are explicable, however, on the assumption that the stress of the disease falls on the capillaries of the brain, and the parasite has been observed in the capillaries of the corpus striatum, and this would explain the high temperatures sometimes observed.

Coma attended with hyperpyrexia is very profound; reducing the temperature with wet packs and stimulating with a strong faradic current does not restore consciousness. Judging from the rarity of motor disturbances, the dilatation of the pupils, and irregularity of the heart, it seems that the structures at the base of the brain, rather than the cortex, are the parts affected by the malarial parasite. Furthermore, the symptoms of coma without excessive temperature resemble those of basal meningitis; thus there may be opisthotonos, slight cephalic cry, constant turning of head from side to side, &c.; optic neuritis is not always present. There is some reason for thinking that a certain degree of coma may be brought about in another way, and that is by the absorption of leucomaines from the bowel. Cases are met with occasionally closely resembling typhoid fever. The bowels become distended with gas, the patient becomes unconscious, and the first impression is that perforation of the gut or intestinal hæmorrhage has occurred. If the bowel can be emptied of its very foul contents the patient regains consciousness, at any rate for a time. Now this effect cannot be obtained in cases of true coma, at any rate not by the same means.

(4) *Necrosis.*

The incidence of local necrosis of the skin is often unexpected, for it may occur in a simple and straightforward case, one in which the patient hardly complains of being ill at all. In such cases the illness is generally of some three weeks' duration, and considerable wasting of the tissues has occurred. The first intimation of necrosis may be the formation of a few small boils on the legs, side of the neck, and often on the nose. The apices of these boils next necrose, or we may have a surface of a square inch of skin becoming black and gangrenous, a slight bruise determining the site. The temperature rises several degrees, the ocular conjunctiva becomes injected, and without further developments the patient sinks rapidly. Recovery sometimes takes place when only a few boils necrose, but when a larger area of skin dies the condition of the blood is evidently such as to be incompatible with the maintenance of life. In the subject of necrosis it seems very difficult to induce quinism.

UNDULANT (MALTA) FEVER.

By Captain M. Louis Hughes, R.A.M.Corps.
Assistant Sanitary Officer, Aldershot.

Synonyms. — Mediterranean, Malta, Neapolitan, Rock (Gibraltar), &c., fever; febris sudoralis, adenotifo, fièvre typhoïde sudorale italienne, &c.

Definition.—An endemic specific fever, occasionally prevailing as an epidemic, having a long and indefinite duration, and an irregular course with an almost invariable tendency to undulatory pyrexial relapses. It is usually characterised by constipation and profuse perspirations, and accompanied by symptoms of a neuralgic character, often accompanied by swelling and effusion of the joints and other rheumatoid symptoms. After death, the spleen is found to be enlarged and often softened, many of the organs congested, but Peyer's glands neither enlarged nor ulcerated, nor is ulceration present in other parts of the small intestine. There is a constant occurrence in certain tissues of a definite species of micro-organism.

Geographical Distribution.—It is widely distributed on the islands and shores of the countries bordering on the Mediterranean, south of latitude 46° N., from Gibraltar to the Red Sea, while somewhat analogous (if not identical) cases met with in India, China, America and elsewhere lead to the belief that the disease may prove to be endemic in those countries also.

Etiology.—The disease is due to the entrance into the body of the minute "micrococcus malitensis" of Bruce, which is also pathogenic to monkeys, and can

be cultivated artficially. It is present in the spleens and livers (and probably other organs) of those suffering from the disease. It does not enter the body by food, water or milk, nor is there evidence in favour of transmission by inoculation. It is not propagated by direct contagion from man. There is strong evidence to connect the occurrence of cases with pollution of dwellings by the urine and fæces of those suffering from the disease. How the poison traverses the few feet which intervene between this fæcal pollution and the patient's body remains to be solved. The writer has been led to believe that it enters the body as an aërial poison (or dust) by the mouth, giving rise to some soreness of the throat and tonsils, with pain and slight swelling of the cervical lymphatic glands. Mosquitoes and sandflies, *per se*, in sanitary houses certainly have no connection with the disease, but their possible relation to it in fæcally polluted places should be investigated. There seems to be no doubt, however, that it is a filth disease, propagated by a micro-organism present in the discharges of those who are suffering from the fever. This organism is moreover presumably able to exist in the soil, but is apparently not present in clean, unpolluted soil.

Those attacked are generally between the ages of six and thirty, though no age is exempt. Residence in an infected area does not confer immunity, but natives suffer less than aliens, as is the case with other endemic diseases. Sex and station in life have little influence. The attack rate in the Mediterranean is lowest during the first quarter of the year, rising rapidly in May to a maximum in July, August and September, after which it falls, reaching the lowest rate in December, January and February (average of 1339 cases). This curve bears a direct ratio to the mean air temperature and an inverse ratio to the amount and continuance of the rainfall. One attack appears to confer some immunity, though it is doubtful whether this is absolute or lasting in its effect.

The incubation appears to vary, but is probably about ten days.

Symptomatology. — The disease has many of the general and local symptoms that would result from a pyæmia, in which the virus was unable to produce actual suppuration. From this point of view we meet with variations in severity extending from a rapidly fatal general infection to a chronic hectic pyrexia with variable local symptoms. As a matter of descriptive convenience only, the writer has divided cases into three types, any one of which may, however, by an increase or decrease in severity, pass into any other type.

(1) Malignant cases occur with somewhat sudden onset, severe headache, pains "all over," nausea, foul tongue and breath, possibly vomiting, and other symptoms usually associated with high pyrexia. Such a case left to run its course will, after a few days of high pyrexia (104° to 105° F.) develop basal pneumonic congestion, and later on lobular consolidation, the stools and breath becoming extremely offensive. Later on the pulse will begin to flag and intermit, the breathing become embarrassed, and obstinate vomiting may set in. Symptoms of the "typhoid state," with delirium, coma and death from hyperpyrexia, heart failure or lobular pneumonic consolidation or a combination of them all, result. At the *post-mortem*, even a few hours after death, such corpses have been described as "horribly offensive and rotten." Happily these malignant cases are rarely met with when treatment is prompt and energetic. Occasionally mild and chronic cases may develope a similar malignancy late in an attack. Other fatalities, at a later stage in the disease, result from syncope, debility, pericardial effusion or concurrent phthisis.

(2) *Undulatory Type.*—These cases are marked by intermittent waves of remittent pyrexia of variable length, marking the pyrexial intensity, separated from one another by periods of temporary abatement or absence of symptoms. These pyrexial waves are the only constant feature of this fever, and for this reason the author has suggested the name *febris undulans*, for want of a better. In typical uncomplicated cases, confined to bed, there is usually a premonitory stage of low spirits, sleeplessness, anorexia with dyspeptic symptoms, and each evening headache and slight pyrexia. The temperature next rises gradually, remitting each morning about half the amount of the previous evening rise. With this are combined slight headache, pains in the back and limbs, moist, furred, swollen tongue, a bad taste in the mouth, epigastric tenderness, and constipation. Most patients, however, do not at first report themselves sick, but, imagining that they have "a bilious attack," or "liver," take aperient pills, and attempt to work the illness off by hard exercise. The result is that all the symptoms become exaggerated, and on admission the temperature is high, and the headache severe. The temperature having reached 103°-105° F. in the evening, accompanied by some bronchial catarrh or hypostatic pneumonic congestion in proportion to the severity of the case, after a variable period gradually falls to normal or thereabouts in the morning; and though it may be slightly higher in the evening, the patient feels better and wants to get up, while the primary wave may be said to be over. After a day or two, however, the temperature again begins to rise, and a relapse ensues, similar to the primary attack, but usually less prolonged and less severe. This subsides, but is followed by other relapses forming the undulatory temperature charts so characteristic of this fever. Such pyrexia is nearly always accompanied by obstinate constipation, though diarrhœa may occur temporarily in very severe cases, more especially during the primary attack. Each daily remission of temperature is accompanied by profuse sweating. Anæmia and muscular wasting are progressive and often extreme. At any stage, but usually late in the attack or during convalescence, symptoms of localised interstitial neuritis may occur, leading to obstinate sciatica, intercostal neuralgia, &c., or to symptoms referable to irritation of the peripheral sensory nerves or of the nerves of special sense. In many cases effusion into one or more joints may suddenly occur, of a transitory and metastatic nature, but causing extreme tension and pain; or acute orchitis may appear. Finally the patient is reduced to an emaciated, anæmic, bedridden condition; subject to attacks of bronchial catarrh, lobular pneumonia, cardiac palpitation, rheumatic or neuralgic complications on the

slightest exposure to chill, change in the weather, or excitement. Disappointed at each relapse, his whole expression is the picture of despondent apathy, his only wish to get away to England. His emaciated appearance, his profuse night sweats, often intermittent pyrexia and cough, remind one forcibly of the later stages of phthisis. Gradually, however, towards the end of the second or third month his temperature becomes normal or subnormal in the morning, and but slightly above normal in the evening; next the evening rise ceases, giving place usually to a day or two of subnormal temperature, after which convalescence is established. The strength now slowly returns, the cheeks fill out, and the patient gets up for an increasing period every day. After a few weeks, if not invalided home, he is sent to a sanitarium, or perhaps to duty, but for months after is liable to attacks of neuralgic pain, to swollen joints or testicles, combined with slight pyrexia. Final recovery cannot be said to take place for many months, and until the anæmia has disappeared and the mucous membranes have regained their complete functions. When once, however, completely free from the disease, it does not recur like paludism.

(3) *Intermittent type.*—In these cases, the temperature intermits daily, and being of a non-paroxysmal nature, they resemble hectic fever cases. If, however, the temperature be accurately taken, so as to register the daily maximum and minimum, these are found to form waves of pyrexial intensity similar to those previously described, the daily remittence being exaggerated into an intermittence. These cases are, as a general rule, shorter than the undulatory ones, complications being milder in character when present. Constipation, night sweats, and progressive anæmia accompany the pyrexia, while arthritic effusion may occur. The patient is usually less despondent, the absence of marked undulations does away with the disappointment felt at each relapse, the patient's daily remark being that he is "just the same." In many cases non-paroxysmal pyrexia and profuse night sweats are the only symptoms present, and the patient, if allowed, wishes to get up and eat solid food each morning, but suffers from slight *malaise* in the afternoon. Such cases go on steadily in spite of all the drugs in the Pharmacopœia, and though an indiscretion may bring on serious symptoms, they seem to cease spontaneously, or at the commencement of some new line of treatment, which, however, fails to stop other and similar cases.

Between the undulatory and intermittent types we meet with every variety of curve that can be said to approximate to the above individual descriptions, or to a mixture of both; but there is always a tendency to the formation of waves of pyrexial intensity if the curve is accurately registered.

Special symptoms.—In severe cases the face may be cyanosed, but in long cases towards the end of an attack the face becomes of a dull clay colour, the skin tightly drawn over the skull, with an expression of listless resignation to an uncertain fate. There is no exanthem, but sudamina are not uncommon during and after the third week, especially when the skin is not properly attended to. Prickly heat (which may become pustular) is an annoying complication of hot weather, while in the spring and autumn boils may occur. Subcutaneous hæmorrhages, combined with scorbutic symptoms, are of rare occurrence. About the fourth week desquamation takes place, being most noticeable on the soles of the feet, where the skin peels off in large flakes. Towards the end of long attacks the hair falls out extensively, but is gradually replaced by new growth during convalescence. As in enteric fever, there is a certain amount of cutaneous bronzing, but nothing approaching the pigmentation of paludism. A distinctive and disagreeable odour is present in nearly all cases, being most especially marked at *post-mortem* examinations. The profuse diaphoresis following the fall of temperature when intermittence is present is most characteristic, and gives rise to the name "febris sudoralis." The sweat rolls off the patient's face in large drops, soaking through the pillow; while at the same time it soaks through his flannel suit, the sheets, and even the blankets, occurring usually about one or two o'clock in the morning; this lasts an hour or more, necessitating two or three changes of clothing.

Pyrexia is the chief and often sole symptom present, and although, speaking generally, there is a relationship between the temperature curve and the other symptoms present, yet the pyrexial severity is not always an indication of the urgency of the symptoms or of the prognosis in any given case. Its height in a large number of cases seems to depend upon the capacity of the individual to nervous excitability.

The chief characteristic of the pyrexia of this fever, when compared with that of others, is the variability which exists in its amount and duration in different cases. The daily curve may vary between a continuously high temperature and an intermittent one. One point, however, is common to all cases, in that the daily maximum and minimum temperatures tend to form waves of intensity of varying character and duration. These waves in individual cases have a tendency to resemble their primary wave, though they usually decrease in length and severity as the cases progress.

In length they vary from three to fifty, or more days (average ten to fifteen).

The average number of waves in an attack was three (one to seven). The interval between waves is marked by a period of apyrexia, without other morbid condition, lasting from one to ten or even more days (average three to four), or simply by a comparative abatement of pyrexial and other symptoms of variable duration and degree.

The pulse is usually firm and slow at first (80 to 90), even out of proportion to the number of the respirations, and the amount of pyrexia present. In malignant cases, where there is lung stasis, it is rapid, and becomes small, thready, and then intermittent before the overburdened heart gives out entirely. In long-continued cases it often becomes constantly increased in rate (110 to 120). In such cases cardiac irritability is of common occurrence, giving rise to attacks of palpitation on the slightest exertion, or even under the influence of some trifling emotion. Hæmic murmurs are met with during convalescence. Organic cardiac disease is said to arise in some cases, but the writer has only met with it in four instances

(fatal on the 19th, 62nd, 111th, and 150th days), and in these the condition might have existed previously. In the first two cases the immediate cause of death was pericardial effusion (similar in onset to the characteristic arthritic effusions). Swelling and œdema of the ankles after standing is common during convalescence. The writer has only once met with phlegmasia dolens following an attack of this fever. The blood has been microscopically examined by Dr. Thin and numerous other observers, but no malarial organisms have been found present. The spleen can nearly always be made out on percussion and palpation, and occasionally is considerably enlarged. During the first acute stage it is tender to pressure, and may be painful. The specific micro-organism has been isolated from the spleen during life. Epistaxis occasionally occurs early in an attack; intestinal hæmorrhage is limited to spots of fresh blood in the stools in cases where the lower bowel is affected.

About the beginning of the third week, or earlier in severe cases, bronchial râles may be heard on auscultation in nearly 95 per cent. of cases. In acute cases basal congestion of the lungs is a most common symptom. In severe cases, and especially in those who have previously suffered from pleurisy, pneumonia, or have organic heart lesions, this is apt to pass on to double lobular pneumonia of varying amount, most marked, in the writer's cases, on the right side. A nervous cough unaccompanied by expectoration is occasionally present; while in mild intermittent cases emaciation and night sweats may be combined with bronchial râles and crepitations, and give rise to a wrong diagnosis of phthisis. Pleuritic effusion without suppuration is not uncommon, and often leaves permanent adhesions behind.

The tongue is usually thickly coated with whitish-yellow fur on the dorsum, pink at the tip and edges, moist, swollen, flabby, and indented laterally by the teeth. In very severe cases only does it tend to become dry and brown. Occasionally it becomes red, glazed, raw, and with the epithelium denuded in patches. There is usually foulness of breath, tenderness on pressure in the epigastric region, nausea, and occasionally vomiting, with other signs of gastric derangement. In non-malignant cases constipation is the rule; in the writer's experience constipation has been marked in 81 per cent., diarrhœa in 4 per cent., both conditions in 3 per cent., and a normal condition in 12 per cent. of such cases. In *fatal* cases, owing to the frequent involvement of the lower bowel, diarrhœa has been present in some 50 per cent. of those that were noted. The condition of the tongue is a valuable indication of the suitability of the diet given, and of the permanency of any amelioration of symptoms. A fall in the temperature rarely proves permanent if the tongue remains coated.

Albuminuria is rare even in fatal cases, though a form of large white kidney has been met with in very prolonged cases.

The action of the virus on the nervous system may be regarded as one of the special characteristics of the fever, of which indeed many of the symptoms already mentioned may be more or less the result. Severe headache with shifting pains in the back and limbs is rarely absent in the initial stages. Later on constant or repeated attacks of facial or occipital neuralgia may be present. The tendon reflexes are almost always increased. In a large number of cases, generally late in the attack, or even during convalescence when pyrexia has ceased, other nerves may be affected, and obstinate lumbago, intercostal neuralgia, or sciatica may be set up. In rare cases the acute stage is accompanied by general cerebro-spinal irritation, characterised by mental irritability, delusions, sleeplessness, cutaneous hyperæsthesia, of variable extent, girdle pains, &c. Severe pain and hyperæsthesia of the soles of the feet is a not uncommon condition. Paralysis, partial or complete, of certain muscles is a late symptom. The extensors of the foot and the deltoid are most commonly affected. The muscle slowly atrophies, and as slowly regains, first its function and then its proportions. These symptoms appear to have a special predilection for those who have previously suffered from rheumatism or rheumatic fever elsewhere.

The rheumatic and neuralgic symptoms have often a relation to chills received during an attack of the fever.

Epididymitis and orchitis (usually single) occur at a late stage in a few cases, mastitis very rarely. The testicle swells in from twenty-four to forty-eight hours to the size of an orange, is extremely painful, while there may be some redness of the skin and effusion into the tunica vaginalis. It is often long in disappearing.

Abscesses occasionally occur, but are probably unconnected with the disease in question.

Diagnosis.—Serum from the blood of a patient suffering from this fever gives an agglutinating reaction with cultures (alive or dead) of the micrococcus melitensis, comparable with the serum diagnosis test of enteric fever with the bacillus typhosus. It may thus be distinguished from enteric fever, which condition many of the severe cases simulate at first. Wright's serum sedimentation test is extremely simple and needs no special bacteriological or microscopical knowledge. The absence of entorrhagia, pea-soup stools, rose spots, and the more usual presence of constipation, profuse perspirations, extremely remittent or even intermittent pyrexia and rheumatoid symptoms, are aids to diagnosis, while later on in an attack there is no difficulty in distinguishing between the two diseases. From malarial fevers it may be differentiated without difficulty by an absence of blood parasites, paroxysmal symptoms and its non-amenability to quinine. From the hectic pyrexia of phthisis, empyæma, liver or pelvic abscess, bone caries and other suppurating diseases we can rely upon local symptoms and serum diagnosis, but must bear in mind that phthisis is not an uncommon *sequela* of this fever. The effusion into joints and neuritic symptoms must be distinguished from rheumatic fever, subacute rheumatism, synovitis and neuralgia.

In cases which commence with very mild pyrexia the fever is often overlooked, and such cases treated for dyspepsia, &c.

Prognosis.—The mortality does not usually exceed 2 per cent., the majority of which occur during the first month or six weeks of the attack. The duration

of the pyrexia is most variable (14 to 300 days or more) and averages as much as 60 to 70 days, while the average time spent in hospital by 844 of the author's cases was nearly ninety days.

The previous existence of cardiac, pulmonary or tubercular disease, or concurrent existence of organic, cardiac or venal affection, anæmia, phthisis, syphilis, &c., or great nervous excitability, all indicate serious prognosis. Hyperpyrexia following temperature allowed to remain continuously high, and pneumonic states or congestion and heart failure, account for most of the deaths. Excessive diarrhœa, vomiting or symptoms indicative of the "typhoid state" are serious. Except when complicated by other concurrent disease, convalescence is usually rapid, continuous and ultimately complete. A subnormal temperature lasting for a few days, accompanied by a *clean tongue* and returning appetite are the surest signs of approaching convalescence.

Morbid anatomy.—The morbid appearances seen in sixty-two cases were confined to intense congestion of the lungs and other internal organs in acute and rapidly fatal cases, combined with enlargement and softening of the spleen, even to semi-liquefaction. Those met with in chronic cases, dying at a late stage in the disease, are indicative of long continued irritation of the tissues (microscopically).

Prophylaxis consists of proper treatment and disposal of infected excreta, the provision of water-tight drains, adequately flushed, ventilated and disconnected, and other measures to prevent the fæcal contamination of ground in or around dwellings or camping grounds. The avoidance by individuals, when possible, of infected areas, especially from May to September inclusive, and the choice of sanitary dwellings, and other details connected therewith, and with the preservation of health generally in the countries in which the disease prevails.

Treatment.—The patient must be first of all removed from any insanitary surroundings, be confined to bed as long as acute symptoms persist, and arrangements made for suitable nursing and dietary. After making sure that the case is not one of enteric fever, any constipation should be relieved by a good dose of calomel and compound jalap powder, or when doubt exists, by an enema. In malignant cases an ice bag or sleeping draught is often indicated when the early headache or "fever pains" are severe. Cotton sheets and pillow slips, and flannel or flannelette bed clothes, should be worn, and a good supply be available to enable them to be changed during profuse sweating.

During acute stages the dietary should be roughly on the same lines as is recommended for enteric fever, and be graduated according to the *severity of the pyrexia and the condition of the tongue.* The too general tendency to over feed during high temperature, when digestion is feeble or almost in abeyance, and to under feed when pyrexia is low, should be guarded against, peptonised and other easily absorbable food-stuffs being especially useful for malignant cases with continuously high temperature. During the later stages, when the temperature has become intermittent, and reaches normal or subnormal each morning, patients often derive great benefit from a good breakfast which would upset them later on in the day. The patient is apt to feel very "down in his luck" when, as is not unusual, the pyrexia is subnormal in the early morning. A glass of milk at 6 or 7 a.m., with possibly a little whiskey, is beneficial at that time as an almost routine practice. Where much dyspepsia or vomiting are present, bismuth subnitrate and peptonised foods are useful. As these patients are often for many weeks without vegetables, the writer has made a point of giving his patients fresh lemonade and fruit as seem suitable. Cooked fruit, such as apples and prunes, are useful also to combat the constipation which is an almost constant feature of this disease. Beyond this, dietary is based upon general principles. Stimulants, stimulating expectorants and cardiac tonics, such as digitalis and strychnia, may occasionally tide over a severe crisis of temporary anxiety, when high temperature is combined with signs of feeble heart's action and stasis in the pulmonary circulation. At such times care must be taken not to add to the embarrassment by over-loading the stomach with undigested food.

The skin needs much attention, and may with advantage be sponged once or twice a day with tepid water to which acetic acid or ammonia have been added.

Pyrexia should be regulated, and in most cases it is best to interfere whenever the temperature exceeds 103°F., but not otherwise. Nothing succeeds so well as cold or tepid water sponging, and many complications and fatalities may be avoided by a judicious and systematic employment of such treatment. With possibly the occasional exception of phenacetin, antipyretic drugs should be avoided in all cases.

Marked diarrhœa should be checked. When due to irritation of the large gut, injections of starch and opium give relief. Beef tea should be stopped when diarrhœa is present. In long-continued cases bed sores must be guarded against. The lungs should be carefully watched, and any symptoms of bronchopneumonic stasis or congestion dealt with at once, especial care being necessary where cardiac disease co-exists. Bromide of potassium and morphia internally may be necessary if peripheral neuritic symptoms are severe, with hot fomentations followed by opium and belladonna locally, and *flannel next the skin.* Such patients, and those suffering from joint effusions, should sleep between blankets, the joints being wrapped in cotton wool. Excessive hyperæsthesia of the soles of the feet may be relieved by soaking them in cold water, or wrapping them in cold-water bandages. During the late and more chronic stages nothing is so efficacious as open-air treatment, except during damp (or sirocco) weather. Patients should be carried out and lie on couches in the open air or on balconies if the air is warm and dry, but must be carefully protected from chills, or neuritis may result. All bodily or mental fatigue or excitement must be avoided.

During convalescence physical and mental work must be only gradually resumed—as much time as possible being spent in the open air. A tonic, such as Blaud's pills, compound tincture of gentian and nitrohydrochloric acid (freshly prepared) or quinine (gr. ii. to v.) twice or thrice a day, is beneficial.

Patients should not be sent away for change of air

BERI-BERI.

Beri-Beri has come to be accepted as the designation of a specific multiple neuritis occurring in many tropical, sub-tropical and even temperate climates. The Dutch in Sumatra first drew attention to the disease, and since then it has been carefully studied in Brazil, Japan, India, Ceylon, China, the Straits Settlements, British North Borneo, and in a few of the islands of the Pacific, whither it has been recently introduced. The disease is not, however, confined by any means to the East Indies, nor even to the Tropics. In America it has been met with at Panama, at Havana, and amongst the fishermen on the coast from Newfoundland to New York, and in several lunatic asylums in the States; in Africa cases are reported from Lake Nyassa, the Upper Congo, the railway workmen on the Congo railway; in Australia Beri-Beri has been seen amongst the Chinese immigrants and Australian natives. Recently we have been made acquainted with an epidemic of the disease in a lunatic asylum in Dublin, Ireland, and it is known to occur in similar institutions in North Germany and France. Nowhere, however, is it seen to prevail with the virulence observed in the East Indies and in Brazil. The early planters in British North Borneo were ruined by the mortality it caused amongst the coolies imported from China, and the opening up of the country was paralysed for a time. Throughout the Dutch East Indies and in the Malay Peninsula the workers in the plantations and mines are subject to frequent epidemics of this terrible scourge.

On board ship, and more particularly in ships engaged in the coolie traffic, or in ships carrying native crews, it is frequently seen; and once it has occurred on a ship it would seem as though the disease stuck to the vessel and re-infected fresh crews again and again.

Many theories have been advanced as to the cause of Beri-Beri. That it is a special toxin which causes its peculiar symptoms there can be no doubt, but the origin of the poison has not been ascertained. The Dutch observers in Sumatra, Pekelharing and Winkler, described and figured a parasite which they met with in the blood of Beri-Beri patients when first affected; this observation, however, has not been corroborated, and we are at present unaware of the source of the toxic influence. Malaria, insufficient food, food deficient in nitrogen—such as rice—overcrowding, damp dwellings, and many other suggestions have been made as regards the ætiology of the disease, but they are merely suggestions, no definite conclusions as to the origin of the toxin having up to the present been arrived at.

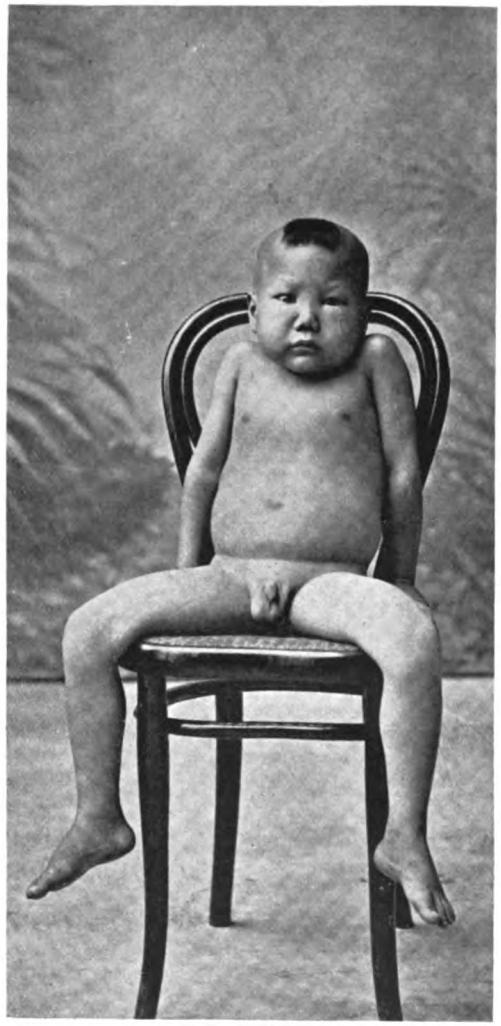
The signs and symptoms of Beri-Beri in the early stages are:—Slight œdema along the crest of the tibia; a puffy, pasty face; difficulty in certain movements, observable only at first when the patient walks quickly or attempts to go upstairs; some paræsthesiæ or anæsthesia of the lower extremities; palpitation of the heart; a slightly quickened pulse, or rather a pulse which remains within normal limits whilst the patient is at rest, but which, upon the slightest movement, goes up to 90 or 100 beats per minute; a slight increase of dulness of the heart's area to the right; a marked contrast between the violent impulse of the heart and the small and feeble pulse; a prolonged first and reduplicated second sound of the heart. Shortly after the invasion of the disease the knee jerk will be found at first to be exaggerated and then to disappear. The superficial reflexes are usually present but the deep reflexes disappear. The muscles show, when tested electrically, the re-action of degeneration. The gait of the patient is affected in various ways according to the particular group of muscles involved and to the degree in which they are involved. The muscles of the leg are usually extensively and deeply implicated and the movements of the feet and legs proportionately perverted. When walking, the patient lifts the feet high, the toes are the last to leave the ground, and when the foot is brought down it descends on the floor with a flop. The finger tips feel numb, and being anæsthetic, feeble and perhaps ataxic, the patient cannot pick up a pin or fasten his clothing. The affected muscles are always tender, and at times extremely painful when handled or compressed. The general health, as evidenced by a clean tongue, a good appetite, and regular evacuation, may remain good. The classification into the wet, dry and mixed forms of Beri-Beri is unscientific, as any one may merge into the other, or the conditions may alternate.

Sudden death, occasioned by a paralysis of the heart in Beri-Beri, may occur at any stage of this disease.

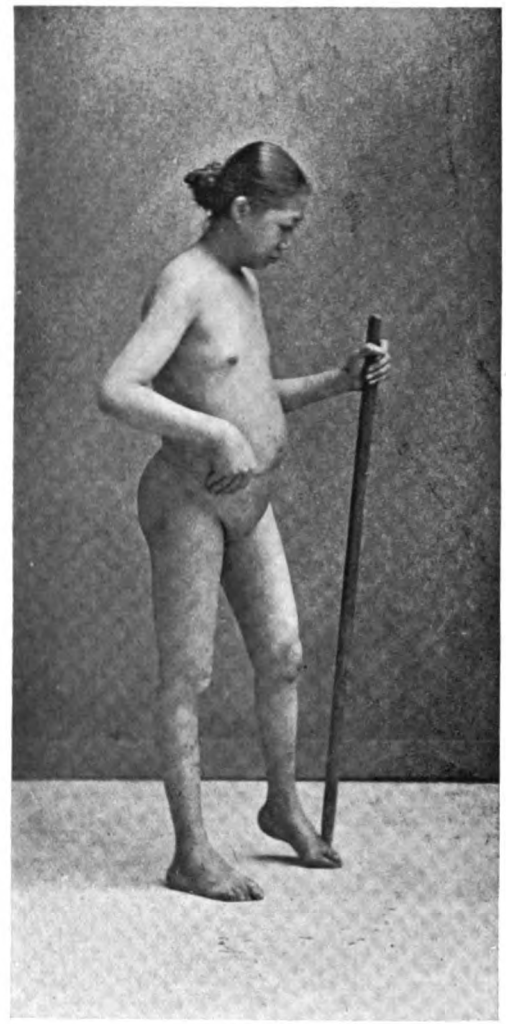
In Beri-Beri the patient may succumb to a paralysis of the organs of respiration, or of the larynx, or to œdema of the lungs.

When the motor nerves, the sensory nerves, and the vaso-motor nerves are attacked at the same time, paralysis and cramps, anæsthesia and paræsthesia, œdema, vomiting, diarrhœa, &c., may appear together.

The treatment of Beri-Beri consists primarily in removing the patient from the house or ship, and if possible from the district in which he contracted the disease. Nitrogenous, preferably animal, food should be supplied in moderation. Cardiac tonics, such as digitalis, are advisable, and general hygienic measures combined with massage, when the muscular tenderness has subsided. The death rate is very high in cases that have not been removed to a healthy neighbourhood, but if Beri-Beri is treated early and under good sanitary and dietetic conditions, permanent recovery is the rule.



A.



B.

BERI-BERI.

Photographs by Dr. J. C. GRAHAM, Deli, Sumatra.

A. Boy, 5 years old. Died after two months' illness. Photograph shows the characteristic anxious look, the paralysis of the anterior tibial muscles, and the general œdema.

B. Woman, 20 years old. Recovered. Photograph shows the paresis and wasting of the tibial muscles, and of the extensors of the forearm.

Both cases occurred in a place usually free from Beri-Beri, and both were in good circumstances; neither overcrowding nor underfeeding were factors in the disease.

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during the acute stages or until a sure diagnosis has been made. Nor is it justifiable to send them in an early stage away from the comforts and attentions of home or hospital to the fatigues of travelling or the doubtful resources of foreign hotels, or away from skilled and friendly advice and health for the sake of an over-estimated benefit of "change of air." During the spring and summer months patients able to bear the voyage should be sent home to England to some dry inland place which is bracing without being bleak or exposed. During the autumn and winter they are better in a warmer climate where they can get out of doors more often, an elevated situation, with cheerful and sanitary surroundings being chosen, where the benefit of change can be accompanied by suitable dietary and freedom from physical discomfort and mental work and worry.

THE TREATMENT OF CRAW-CRAW.

By Dr. J. Emily.

(*Translated from "The Archives de Medicine Navale," January*, 1899.)

Marchand Mission, Fashoda, Soudan, December 8, 1898.

Craw-craw is a skin affection well known to medical men engaged in practice in the French Congo. The disease usually manifests itself on the lower part of the legs, the ankles, the back of the feet and the toes, but it is not uncommonly seen to affect the backs of the hands and fingers likewise. It is ushered in as a small reddish-brown coloured spot merging circumferentially into violet, and accompanied by an overpowering tendency to scratch; the spots, which at first are mere papules, become excoriated by scratching, and from their surface a serous fluid exudes. Pustules soon form which in turn suppurate, and coalescing leave a deep sore with almost perpendicular edges. The surface of the ulcer is covered with pale granulations, secreting a somewhat thick tenacious pus. The zone around the sunken patch shows the skin to be inflamed and of a deep "wine-dreg" colour, shading off at the circumference to a light red.

The pus by its constant flow keeps up an irritation, which extends the ulcer by causing its edges to slough until it may attain the size of half an orange. The pus cakes and beneath the crust matter collects and burrows, causing marked induration of the skin for some distance round. When craw-craw attacks parts where the subcutaneous tissues are thin, as on the fingers and shins, it causes pain of an excruciating character.

Subsequent investigation will perhaps enable us to confirm for certain if this affection is caused by a special microbe. It has already been affirmed that this microbe exists, and that in fact it is a gonococcus. Whatever it is, one may say with certainty the disease is contagious, and one may try to establish how it is contracted and how developed.

Many persons believe the disease to be caused by mosquito bites, especially on the wounds left by sand-flies. The latter are very numerous on the coast of the Gabon; they infest the caravan routes between Loango and Brazzaville; and carried by our infantry, perhaps in the wrappings of our bales of goods, they have been conveyed into Central Africa. Mosquitoes also are very numerous in these regions. All who live in the countries referred to, Europeans and natives, suffer with innumerable mosquito bites, and the feet and the hands are also bitten by the sand-flies. Nethertheless I have never observed craw-craw on the negroes of the Lower Congo, and it was rarely that I had to treat a case amongst the Soudanese Infantry which formed the escort of the mission to which I belonged. In the same way but few Europeans were attacked by the disease.

It is none the less true that every little injury to the epidermis opens a means of entrance to the pathogenous medium, and thus facilitates the outbreak of craw-craw. All these mosquito bites which one scratches and tears open, all the sore places left by the sand-flies, are so many spots by which the insidious microbe may enter. It seems, then, to reside only in the irritating liquid which these parasites inject beneath the epidermis.

Besides, once the microbe is established anywhere, the patient himself helps to spread it. The nails and fingers are the agents of the dissemination. A craw-craw spot is touched, and the same hand is used to allay the irritation caused by bites of mosquito or sand-flies, and thus one may inoculate oneself *ad infinitum*. A craw-craw spot is never found isolated. The hands, the feet, the lower extremities are covered with the same, and sometimes the whole body. I have observed them on the thorax, the back, and even on the face.

There is no disease more obstinate. Once craw-craw has developed anywhere, the subject is liable to constant relapses. In my observations I shall give as examples the case of two officers of the mission, who having contracted the affection, one at Loango and the other by Brazzaville, had eruptions of craw-craw in the basin of the Nile, at Tamboura, a year and a half afterwards.

Although, as I stated above, this skin disease appears mostly on the extremities, parts of the body less exposed are also attacked. My colleague, Dr. Spire, colonial doctor serving at Oubanghi, was able to confirm the existence of craw-craw on the penis and scrotum. Since then I have myself observed this localisation. One might imagine one had a phagedenic chancre to deal with. Inguinal gland enlargment is not uncommon, and at first sight, when one is not on the alert, one may fall into an error of diagnosis. Nevertheless the relapses, the course of the disease, and above all the presence of ulcerations of the same nature on other parts of the body, do not leave one long in error.

In conclusion, I will say that the germ of the craw-craw pustule does not seem to me to be inoculated either by the mosquito or by the sand-flies. These parasites create points of entry which the nails of the patient enlarge, thus facilitating the dissemination. But it is not in these parasites that the cause is to be found, and the specific microbe, if it exists, must be sought elsewhere.

The anæmic state of the victims, some natural predispositions, the forced want of cleanliness, the abundant sweat and the irritating dust, may perhaps suffice to explain the genesis of these pustules, which have much resemblance to the Annamite sores and all the special ulcers of hot countries.

This affection, exhausting on account of continual suffering and continual suppurations, has forced more than one European patient, already enfeebled by the climate, to abandon his business or official duties and to return to Europe. Even in its most benign form it is a considerable hindrance to walking, sometimes preventing it entirely. This consideration, in regions where excursions only can be made on foot, makes such an illness to be dreaded by everyone.

The patients I have treated were subjected to continual change of residence so that it was impossible to submit them to rational and prolonged treatment. But in all the districts where I have stayed some time, at Libreville, Loango, Brazzaville, and later on in nearly all the stations of Oubanghi, I had the opportunity of seeing how vain and illusory were the means used to cure craw-craw. Not only my companions who were obliged to be on the move did not get cured, but those in residence who could take every care of themselves were not more fortunate. This was not for lack of trying various remedies. While some praised permanganate of potash, others preferred corrosive sublimate, and others again used carbolic dressings. A small number derived benefit from iodoform powder. Prolonged baths in these various solutions or damp compresses applied on the sores, constituted the method of treatment. But the results were always unsatisfactory, and the illness, even if cured in the long run, was liable to break out again. In this condition several of us arrived in the Nile valley, still suffering with craw-craw contracted in the Lower Congo. My conviction was confirmed that the therapeutic methods employed to treat this pustule were insufficient. I then set myself to seek other means.

Observation No. 1.—D———, Sergeant of Marine Infantry, 26 years old; in the Colony eight months; diabetic; has already had several attacks of severe fever, one being of a bilious hæmaturic form. The patient was very anæmic. He had made a stay at Madagascar, but never had any skin disease. Came to see me on my arrival at Fort Hossinger, on July 18, 1897, having suppurating pustules on both legs, and on the right foot. He himself diagnosed the illness as craw-craw. It broke out about a month previously. The patient treated himself with spongings of a solution of sublimate of 1 in 1,000, and by damp dressings of wadding soaked in the same preparation, with no perceptible amelioration. The zone of inflammation, of a coppery red verging on violet, which surrounded each pustule, had a little diminished in circumference, and the painful irritations were less acute at the diseased parts, but not a sign of cicatrisation manifested itself anywhere. The suppuration had not dried up. I then tried baths and dressings of permanganate of potash, which treatment is much recommended in the Lower Congo. At the end of a week, not being able to see any progress, I resolved to change the dressings.

After having carefully washed and cleansed out the adjacent parts and the depths of each ulcer (which I made to bleed slightly) with a plug of wadding steeped in solution of sublimate of 1 in 1,000, I liberally powdered each with boric acid. Over this I placed a square of aseptic gauze on which I spread a thin layer of boricated vaseline. I covered this with a piece of dry cotton wool over which I again applied a bandage of ordinary calico. The latter covered up all the little dressings applied to each craw-craw ulcer, so that in fact my dressing had the appearance of a high boot reaching to the knee. A very tight bandage kept all in place and the patient was sent off with the order not to walk too much.

On paying his visit next day, the patient declared that the bandage was not at all painful, having only smarted slightly the first few minutes; the itching had disappeared, so I did not touch the dressings. In the meantime the patient had to take to his bed, having been seized with hæmoglobinuria for the second time in three months, and he had to keep his bed for eight days. The dressings kept in place, causing no inconvenience; I therefore did not touch them till the day the patient got up, that is to say nine days after having applied them.

All the craw-craw ulcers, without exception, were completely healed. Their surfaces were now covered by a slightly pigmented cicatrix, but perfectly smooth and soft.

During the two subsequent months that the patient passed near me no new outbreak occurred.

Observation No. 2.—Lieutenant M., of the mission, was attacked by craw-craw at the Station of M'Bamou near Brazzaville, in September, 1896, three months after his arrival in the Lower Congo. From the very commencement the illness took a serious form. The patient, incapacitated for a long time, had to be carried in a chair to survey the installation works of his station, his feet, hands and legs being covered with large pustules. The treatment employed was as follows: First prolonged tepid baths of sublimate of 1 in 1,000, then the application of compresses soaked in the same solution on all the bad places. The dressings were renewed daily, but no good resulted, not even a diminution of the itching which was intolerable. Before this failure the patient had tried "liquor of Van Swieten," and replaced the same by a solution of 1 in 1,000 permanganate of potash, which he employed in the same manner. Amelioration very slow. The patient dare not try iodoform, which he knows he cannot stand. This drug, instead of helping the cicatrisation of wounds received in battle in the Soudan, had only irritated them and had brought on erythema of the skin over the parts where it had been applied.

At last, after prolonged treatment of more than a month, some craw-craw ulcers disappeared; but these broke out afresh, and walking became impossible.

Just at this time the patient was obliged to go into the bush at the head of his infantry to suppress a partial revolt, in consequence of which he had a relapse. He again began the permanganate of potash treatment and partial but not complete disappearance of the pustules resulted. It was in this condition that Lieutenant M. embarked at Brazzaville with me on

a steamer bound for Upper Oubanghi. I continued to dress each craw-craw ulcer, as soon as it appeared, with permanganate of potash. This lasted till our arrival in the Nile basin in July, 1897. The patient was relieved, but new eruptions on the legs, the arms and the hands, broke out continually and were always very troublesome and at times very painful.

On August 10, Lieutenant M. pointed out to me a craw-craw ulcer which had broken out on the second phalanx of the index finger of the left hand, and two others which had appeared on the lower part of the right calf. Immediately I proposed the boric acid dressings to him, and he willingly acceded. I applied them in the same manner as in the preceding case. At the end of eight days, on removing the bandages I found the sores were quite healed, with a cicatrix hardly tinted and which appeared white by contrast to the black scars left by craw-craws previously cured.

Since then no relapse. Lieutenant M., during the course of new walking expeditions, has had numerous blisters and excoriations made by boots; whereas previous to the boric acid treatment the slightest abrasion had turned to craw-craw, the same now healed rapidly and easily without any appearance of craw-craw.

Observation No. 3.—Captain G., of the mission, was attacked with craw-craw some days after his arrival at Loango in June, 1896. The eruption was confluent and very painful on the feet, legs and hands. The irritation was unbearable and the patient could not resist scratching; he applied dressings of Van Swieten's liquor, then of permanganate of potash, then of iodoform. After two months of ineffectual treatment applications of glycerolated starch gave great relief. Walking, previously impossible, became comparatively easy and the patient began on foot the march of 500 kilomètres to Brazzaville. On the way he had slight relapses which he could not properly attend to.

In January, 1897, I went on board a steamer at Brazzaville, bound for Bangui, with the patient. He had outbreaks of craw-craw, which I treated with permanganate of potash, having neither glycerine nor starch in my medicine case. Obliged to separate from my patient in February, I did not see him again until ten months after, in October, 1897, at Fort Hossinger. Being obliged to start at once for Soueh, a tributary of the Bahr-el-Ghazel, he asked my advice for several craw-craw ulcers on his calves and feet. He told me that since our separation he had never left off having fresh eruptions, and at last, worn out by the futility of his efforts to cure them he had finished by treating them with contempt; but the route between Zemio and Fort Hossinger (nearly 400 kilomètres) had caused all the old sores to break out again. Walking was most painful to him, and he begged me to dress the sores before he resumed his peregrinations. I gave him all the information I could as to treatment, and at the same time I told him of a new dressing which he was to apply as soon as he arrived at his goal, 80 kilomètres further. At the same time I asked him to let me know how he got on. Taking into consideration the inevitable friction on a long march, I increased the quantity of boricated vaseline and wadding, and on each leg I made a bandage which reached to the knee. Eight days subsequently I received a letter in which the patient explained how pleasantly he was surprised on arriving at the Soueh Station, on unbandaging the limbs to find them quite healed. Walking, a little painful on the first day, was effected afterward without inconvenience, and there was no occasion for a second dressing.

This patient, whom I saw afterwards, had, like the previous case, made long and painful excursions, subsequently, he had had blisters and excoriations on his feet, but not a single craw-craw. The cure has lasted now fourteen months.

Observation No. 4.—During my stay in Fort Hossinger, in September, 1897, Lieut. G., then on the M. Bima route, 50 kilomètres from me, wrote that he was suffering from craw-craw and asked me for medicaments. I at once sent him Van Swieten's liquor, powdered boric acid, boricated vaseline, sterilised gauze, cotton, &c. These were accompanied by a note with a detailed explanation as to the manner of applying the dressing. Some days afterwards Mr. G. wrote to me that he had followed my instructions and that the sores were much better. I heard no more of him till his return to the station, six weeks after. Incomplete cure.

Some craw-craws certainly were healed but others still suppurated. I then made a fresh bandaging myself, during the course of which, the patient, who could hardly bear the operation of cleansing the depths of the ulcer with the plug, wished to prevent me making the papules bleed and thus relieve them of their evil contents. At last with difficulty I was able to apply my dressings in the usual way. At the end of six days, without my having to intervene a second time, cicatrisation was complete at every point.

The cure remained permanent until the unfortunate death of this officer, six months after, at the Equator, where he was the first to hoist the French colours.

The perusal of these observations will show that it was the dressings that yielded such good results.

As soon as a patient suffering with craw-craw comes to me he is treated in the following manner:—The part affected is shaved, if necessary, and then soaped and scrubbed vigorously with a brush with solution of sublimate. This toilet completed, the craw-craw ulcers are mopped out with sublimate dissolved in boiled water (1 in 1000). First I open the ulceration by removing the scabs with irrigations and sometimes with the help of the spatula. Mostly there was then revealed a granular uneven basis, full of pus. Then with a wet plug, I completely clean out the sore. Not only do I remove all traces of pus, but I do not rest satisfied till I bring a little blood to the surface of the flesh. This is very painful but does not take long. This done, I liberally powder all the ulcerations with pure powdered boric acid. The cavity is completely blocked with this powder, and the edges quite covered. Over this I apply a large square of sterilised gauze on which I have spread a layer of boricated vaseline. I finish the dressing by covering up with absorbent cotton wool, then ordinary cotton, and keep the whole together with the help of a slightly tight bandage.

This dressing is not painful. The action of the boric acid on the raw surface of the sore produces a slight smarting which does not last long, subsiding entirely in a few minutes. It need only be applied once. It is left in place five or six days, at the end of which time, when removed, I have always found the craw-craw healed and the cicatrisation quite completed.

I have also noticed that in the vicinity of the cicatrix the epidermis regains almost its normal colouring. It contains hardly any of the highly pigmented elements, which are found in such considerable quantities on all the scars of the sores of hot countries, and which were never lacking when craw-craws were cured by other means. This advantage is not to be despised from the point of view of appearance, without taking into consideration that it is unpleasant to have certain parts of the body covered with large blemishes, giving one the impression of syphilitic sores.

The results which I have obtained, thanks to this new mode of dressing craw-craws, seem to me sufficiently encouraging to be made known.

The fact of the cure in some days, and without relapse for more than a year, in an affection so difficult to heal and so liable to relapses, seems to indicate that boric acid is a real specific against the still unknown pathogenic agent which engenders craw-craw. For it is to this therapeutic medium alone that I ascribe this power of healing. Corrosive sublimate alone does not yield the same results. In the three cases which form the object of my first observations, the sluicings and applications with the solution of this medicament could not arrest the disease, nor above all prevent relapses and successive outbreaks. If I make use of it to clean out the sores, it is but to utilise its powerful action against the common microbes of suppuration; whereas, a craw-craw ulceration healed by sublimate or permanganate of potash does not prevent the appearance of new craw-craws. I have not seen one single relapse after the boric acid applications. Does it not seem that it relieves the whole organism of the parasitic generator of the disease?

As to the bleeding of the papules, I think that also is of great utility. If in my fourth observation the result obtained was not an immediate cure, I attribute that only to the fact that Lieut. G. did not dare to cleanse out his craw-craws vigorously enough.

I believe that it is necessary to alter by energetic massage these evil-looking sores, which, having suppurated for so long a time, show no natural tendency to cicatrisation. This little local bleeding must decongest the diseased tissues, and whilst relieving the blood vessels of a viscid liquid, it sweeps away all the germs, it determines the establishment of new healthy elements, and gives birth to a more active phagocytosis.

In conclusion, the complete closing-up, by means of vaseline and cotton wool, prevents the intrusion of fresh germs from the exterior, it renders further inoculation impossible, and thus contributes to a definite result—a complete and rapid cure.

Quinine in Malarial Hemoglobinuria.

Hæmoglobinuria is neither aggravated nor produced by the administration of quinine in doses of any size. While some practitioners in malarial districts believe that quinine does aggravate or produce this condition it can be positively stated that the vast majority of practitioners in those districts do not hold to any such belief. The pathological condition of the kidney in malarial hæmoglobinuria is found in conditions quite distinct from malarial fever, yet the administration of quinine in the latter does not induce a hæmoglobinuria. The study of the plasmodium in the peripheral circulation shows that the full-grown, vigorous organism contains fine masses of hæmoglobin from the red cells that had been set free by the necrobiotic process engendered by the toxin. Every paroxysm increases the amount of freed hæmoglobin that has to be stored within the organs whose function it is to care for the same. The internal viscera must receive it, and when it is thrown off they hold the legacy of its colouring matter within the intracellular tissue. The hæmoglobinuria is a symptom of the malaria and not a distinct entity.

The bright-red urine is rarely a hæmaturia, as hæmorrhage is quite uncommon in malarial fever. The blood colouring-matter and not the red corpuscles produces the glow in the cheek of the sufferer. In the later stages, when the kidney is congested, red cells may be present in the urine. The writer cannot understand why malarial hæmoglobinuria has been singled out for such an explanation while they have not attempted to apply it to other forms. In paroxysmal and toxic hæmoglobinuria no such explanation has been attempted, and he therefore holds that by the rules of logic there is no therapeutic evidence that quinine is guilty.

It has been shown that the largest doses of quinine have failed to produce either a hæmaturia or hæmoglobinuria, and that it neither produces a necrobiosis of the red cells nor a congestion of the kidney. Cases are cited in which 3 dr., 10 dr., and as high as 55 dr. have been taken at a single dose, and in none of them were there any signs of hæmoglobinuria. It has produced cramps, vomiting, paleness of face, dilated pupils, irregular slow pulse, ringing of the ears, syncope, deafness, dumbness, blindness and death, but there is no satisfactory evidence of its ever having produced hæmoglobinuria. As quinine and toxin are in the blood together when patients are under treatment, the author asks which is likely to be most potent in the production of hæmoglobinuria? The toxin produces hypertrophy of the spleen, destroys the red cells, causes chill, and being poisonous may irritate the kidneys, thus engendering the hæmoglobinuria. The quinine has never been proven to produce any such conditions, and therefore must be deemed the least causal agent in the production of this symptom, for which it should be used as a remedy.—*Journ. Am. Med. Assoc.*, Jan. 14, 1899.

Such a generous response has been made to the appeal of the Seamen's Hospital Society, for funds for the establishment of a new School of Tropical Medicine, that it is anticipated that the full amount will be forthcoming by the time the banquet over which Mr. Chamberlain is to preside is held on May 10 next. The dinner itself promises to be a notable function. Besides the Colonial Secretary, many men of eminence in the medical and scientific world are expected to attend, together with a strong body of shipowners. Sir Donald Currie is the chairman of the Dinner Committee, and he has associated with him the Marquess of Lorne, Lord Lister, Lord Rothschild, Lord Strathcona, and the Lord Mayor of London.

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THE

Journal of Tropical Medicine

MARCH, 1899.

LEPROSY IN INDIA.

THE paper recently read on Leprosy in India at the Imperial Institute by Mr. Acworth, C.I.E., formerly Commissioner of Bombay—which we shall publish in our next issue with the discussion that followed—gives the history of the origin of compulsory segregation in India, and shows very clearly that the system now introduced by the Imperial Government in 1898 was largely attributable to the success which first attended that system in Bombay. The picture drawn by Mr. Acworth of the condition of the streets of Bombay before compulsory segregation was established sufficiently justified him, we think, in his action in introducing that measure, especially as it was based on the strong opinion of Dr. Vandyke Carter as to its utility. In every part of the city of Bombay lepers were more or less in evidence in every stage of the disease, and exhibiting in the public streets every one of its hideous deformities. On the occasions of Hindu festivals they crowded the temples on the seashore, lining the streets in scores and hundreds. They occupied the verandahs and courts of empty houses; they washed and drank from the public taps; they scattered their rags about shops and markets, and instead of using, like Job, potsherds to scrape themselves withal, they used rough stones from the heaps of road metal stacked at the street sides. Similar scenes present themselves in all leprous cities where no compulsory segregation exists. In Calcutta, for example, the streets, markets, bazaars, the doors of the rich man during times of festivals, marriages and feasts, and the entrances and approaches to the principal temples were always thronged with lepers. Wherever begging or the soliciting of alms could profitably be carried on there would scores of lepers be seen exhibiting their loathsome sores and deformities. Compulsory segregation cleared Bombay of this loathsome nuisance, and it would clear other cities in a similar manner. In 1890, through Mr. Acworth's influence, the Matoonga Leper Asylum was built on the outskirts of Bombay, and when completed covered several acres of ground. Besides the wards and its adjuncts, it included storehouses, a dispensary, a school, a Hindu temple, a Mahommedan mosque, a Roman Catholic church, and a hospital. In fact, the asylum was practically a small village, whose inhabitants were confined to the boundaries of the village, and whose wants, physical, social and spiritual, were provided for, there being only one restriction, viz., that the lepers were not to go beyond the boundary.

From every point of view this treatment of the leper is the most humane that can be devised, and even those who have expressed themselves as opposed to the view that leprosy can be stamped out by segregation, and are opposed to compulsory segregation, are but half-hearted in their opposition, for they would at least substitute voluntary segregation for compulsory.

Mr. Jonathan Hutchinson, in the discussion on the paper, indicated that he would keep the lepers in asylums by kindness and not by compulsion; but is it true in any sense that kindness and compulsion are divorced from one another? Those who have practical experience in dealing with lepers know that kindness alone will not keep the leper in the East from wandering, unless

the disease causes total disablement. If this object is to be obtained, compulsion has to be added.

The fish theory is a popular one in the West Indies and in other countries, and under the able advocacy of Mr. Jonathan Hutchinson it assumes a most plausible aspect; but examined closely in its bearings it neither applies to India nor China, for it is not on every coast or part of it that leprosy exists, and when the disease prevails in inland places it has not that wide prevalence which is necessary to fit in with the theory. The error is to suppose that the cause of leprosy and its extension can ever be discovered by statistics. They give us the incidence of the disease, but beyond this, without inquiry into the individual cases and the adoption of the synthetical process in regard to these, they lead no further, and all the theories based on them are in the same position, viz., not proven. Leprosy, as Vandyke Carter has shown, has a very intimate relation with family life. In his enquiries he found that out of 1,564 lepers, 64 per cent. had a family taint in the direct line, *i.e.*, father, mother, or grandparents; 14 per cent. in the indirect line, *i.e.*, among aunts and uncles; and 21 per cent. in the co-equal line among brothers and sisters. Every inquiry of a really searching character, when it has resulted in arriving at any positive facts, has shown that prolonged intercourse with a leper usually results in the infection of the healthy; and further, that the infection is slow, and that there is no widespread distribution in space such as was concluded by the Leprosy Commission sent out to India. This conclusion was based on the mistake of dealing with the statistics as a whole, and not with the analysis of the statistics which plainly indicated that leprosy retained a firm hold on certain communities, and from these communities gradually spread to others. Leprosy focuses and attaches itself to certain parts of India, and in those parts, notwithstanding the high mortality of lepers, their abnormally low birth-rate, and their inability to protect themselves from floods and famine, the incidence of leprosy goes on increasing. It is the leper and the things in connection with the leper that are dangerous to others, and we believe that there is no other feasible method of dealing with the disease than by compulsory segregation.

As was very opportunely pointed out by Mr. Cantlie in the discussion, in opposition to Dr. Abraham's view of non-communicability, leprosy is spread in the Eastern Pacific littoral and in the islands of the Archipelago by the Chinese coolie. Mr. Cantlie remarked that the aboriginal tribes were free from leprosy until they came in contact with the Chinese coolie, who is the carrier of leprosy, and if steps are not taken to minimise the evil the islands of the Pacific will share the fate of the Kanakas in the Sandwich Islands. As regards the contagiousness of leprosy, when contact is prolonged and intimate, he believed there could be no doubt. Mr. Cantlie quoted one case where a European contracted leprosy from his boy, who, whilst travelling on a Chinese junk, for some considerable time had to share the same room and the same covering with his Chinese boy, who was a leper. This reminds us of the Dublin case, in which the brother of a soldier who returned from India became a leper by sleeping in the same bed, although he had never been out of Ireland.

Mr. Cantlie, we believe, is right in his contention that it is a duty devolving on the European Powers in the China seas to attempt to stem the spread of the disease by a careful and rigid inspection of all coolies embarking for foreign countries; but we would go still further, and urge that, in addition to these precautions, similar measures as those proposed to be taken by the Indian Government be adopted by other European Powers with Asiatic possessions.

LIVERPOOL SCHOOL OF TROPICAL MEDICINE.—The office of demonstrator of tropical pathology in the newly-formed school of tropical medicine in Liverpool has been filled by the appointment to it of Mr. H. F. Annett, M.B., B.Ch., Vict., D.P.H. Mr. Annett was a student of the Liverpool School, and gained, among other distinctions, the Lyon-Jones Scholarship and the Holt Fellowship in Pathology. He has been an exhibition scholar, and has spent the last two years in study in Professor Koch's laboratory in Berlin. The post of lecturer in tropical diseases has not yet been filled.

Article for Discussion.

ARE ASIATICS WHO TAKE TO EUROPEAN FOOD AND DRINK RENDERED THEREBY MORE LIABLE TO DIABETES?

MEDICAL practitioners in the eastern tropics, who have not had their attention drawn to this subject, may be surprised at such a question being raised. Seeing, however, that there is a certain amount of what seems positive evidence in regard to the connection between the ingesta of certain Asiatics and diabetes, it is advisable to elicit information thereupon.

Many of the more "enlightened" natives of India, the Straits Settlements and China, as is well known, affect "European" ways of living, and amongst other attempts in this direction, take to "English" food and drink. In the Straits Settlements, more particularly in Penang and Singapore, the Chinese, who from long residence have become well nigh indigenous to these parts, make considerable pretensions towards being "European," and, without dispensing with either their "cues" or national costume, build and furnish their houses after the Western model. They also eat and drink "English chow," and to all appearances live as Europeans. This, however, is only true as regards outward appearance, for every Europeanised Chinaman has a part of his house apportioned "China fashion," and in his family life reverts to his native food. In many, also, of the coast parts of China, in Hong-Kong and wherever a European or American concession brings the Chinese merchant in contact with the foreigner, he is wont to follow the dual method of living. "Butcher's meat" is partaken of in place of the rice, preserved fish, and fat pork, which he naturally affects; sweet champagne and claret are drank in place of tea, or sham-shu (rice spirit.)

In but few instances, however, is the diet "single;" more often "native" food is partaken of at one or two meals, and "foreign" food and drink is indulged in as an "extra," or in place of one meal. Seeing that butcher's meat is accommodated in much smaller space than is rice, a larger quantity of the former has to be eaten to satisfy the appetite than is the case with the European accustomed to such a diet. A Scotchman used to a bulky diet of oatmeal makes light of an ordinary helping of meat; a German accustomed to quantities of thick soup and bulky vegetables is regarded, by English folks, as having an inordinate power of consuming the more concentrated foods—beef and mutton. Examples in this direction might be multiplied, but it is an easily accounted for phenomenon, and so it comes about that the Oriental not only eats meat, but must eat it in quantity to get satisfaction from so concentrated a form of food.

That diabetes is common amongst the better class of Chinaman who consult the European doctor is a well-established fact. It is not common—in fact it is a very rare disease—amongst the coolie class nor even amongst the richer class who live in the native fashion; it is almost exclusively confined to those who have taken to European food and drink. These men also, from their knowledge of Europeans and their ways, most frequently consult a European doctor, and it is amongst one's better-off private patients that one meets with natives affected with diabetes.

An analogy in this direction will bring the matter home to practitioners in temperate climates. Diabetes is a disease so prevalent amongst the Jews in Western Europe that its inroads are much dreaded by the Jews as a class. Many of them have been long indigenous to the countries of their adoption, although their ranks, in England especially, are constantly being added to by oriental Jews who have either amassed wealth in some part of the British Empire, or seek the capital to pursue their calling. Is this prevalence of the tendency to diabetes amongst Jews on a footing with the Asiatic who, after adopting European food and drink, acquires the disease? Is this tendency a token of the oriental origin of the Jew, in that he frequently fails in metabolising the meat and wines or beer of western Europe?

Collective evidence on this subject should afford interesting information, not only in regard to the disease itself, but also as regards the *rationale* of the diet in tropical countries. It is not amongst hospital patients, but amongst the Europeanised private native patients that information on this important subject is to be sought for.

Medical practitioners in India and Egypt should have ample opportunities of obtaining evidence as regards Hindoos and Arabs on this subject, and in the United States of America the effect of a meat diet on the negro should be readily ascertainable. I am not aware that diabetes is a frequent disease amongst sugar-fed negroes, but my experience of negro patients who live on European food is that they are as pronouncedly liable to become diabetics, as are the Chinese under similar conditions of food and drink.

J. C.

Reprints.

SPLENIC ENLARGEMENTS: SPLENITIS AND PERISPLENITIS.[1]

By James Cantlie, M.B., F.R.C.S.

Reprinted from *The Clinical Journal.*

Gentlemen,—The patient before us is a woman, aged 41, who has been an inmate of the hospital since March, 1898. Her appearance at the present moment indicates anæmia of a marked character, and the distension of her abdomen with ascitic fluid, combined with the enlarged and tortuous veins over the abdominal wall, suggest visceral disease. Percussion and palpation of the abdomen reveal a contracted liver and a big spleen. Auscultation shows the presence of a systolic bruit over the apex of the heart, and the thrill felt at the pulse is indicative of the state of the circulatory disturbance. The woman confesses to a free use of alcohol, and it seems an easy conclusion to come to that the patient is suffering from cirrhosis of the liver and subsequent enlargement of the spleen due to alcoholic causes.

In dealing with this very ordinary group of clinical signs and symptoms I propose to consider the following points:—

(1) The normal and pathological relative anatomy of the spleen.

(2) The association between enlarged spleen and hepatic disease.

(3) The anatomy and meaning of distended surface veins on the abdomen.

(4) Perisplenitis, its frequency and anatomical consequences.

(5) The treatment of splenic enlargements.

I. *The normal and pathological relative anatomy of the spleen.*—It is the rule when the spleen is being examined to have the patient lying on the back. In this position by palpation and percussion it is no doubt quite easy to find the spleen when it is greatly enlarged; but when the organ is of normal size, or even moderately big, it is impossible to determine, even approximately, while the patient is supine, its exact position or actual size. We are accustomed to regard the spleen clinically as being an organ presenting relations with the anterior aspect of the abdomen, but a very superficial study of the splenic relations will show that this is a clinical fallacy. The spleen is situated far back in the hollow of the ribs; it is accommodated in the angles of the ninth, tenth, and eleventh ribs. The long axis of the spleen runs parallel with the tenth,—"the splenic rib,"—and the inner (upper) end of the organ reaches to the head of the rib. The outer (lower) end of the spleen comes no farther forward than the level of the posterior axillary line, so that to attempt to percuss out the spleen from the front with the patient supine is, in the case of the normal spleen, an absurdity. Such being the case, it is necessary to enquire in which position is it expedient to place the patient so as to determine the exact limits of the spleen. With the patient in the horizontal position and rolled over on the right side, equally fallacious signs of splenic relations will result as the organ moves forwards with the altered position of the body, rendering correct location impossible. Again, with the patient prone it is impossible to percuss out the spleen from the back, as the whole organ leaves its position (unless adherent owing to old perisplenitis) and rolls forwards to the anterior part of the abdomen. It is, therefore, only when the body is in the erect position that the spleen can be percussed out properly, for the organ is maintained in its normal position only when the body is so disposed. The spleen does not descend when the erect or sitting position is assumed, as will be readily understood from a study of its anatomical relations.

Relations.—The spleen lies on the cardiac curve of the stomach, inclined towards its posterior and upper part. Externally and above is the diaphragm. Below the spleen is the pleuro-colic fold of peritoneum presenting a cup-shaped pocket for its support. So exactly does the cup fit the spleen that it is impossible for the organ, when the erect position is assumed, to sink downwards; even when considerable enlargement is present this pleuro-colic fold supports the spleen, and renders enlargement downwards impossible. The only direction along which the spleen can travel during change of position is forwards; and not only so, but it is only in a forward direction that the spleen can expand when it is enlarged. It cannot enlarge downwards until the supporting power of the pleuro-colic fold is overcome; hence it is only when the enlargement is great that the spleen passes downwards towards the left flank.

[1] A Clinical Lecture delivered to the London Post-Graduate Class at the Central London Sick Asylum, December 1, 1898.

In all splenic enlargements the organ primarily expands forwards. As the growth increases the spleen speedily crosses the middle line of the body, but after a time, and owing to a continued increase—not to a diminution—in size it recedes from the right of the middle line of the body, and falls more towards the left flank. The sole determining cause for these relations is the pleuro-colic fold. At first the power of the fold is sufficient to maintain the spleen even when enlargement is great, but after a time the spleen comes so far forwards that it overlaps the anterior border of the fold, and pushing it back drops down by its own weight towards the left iliac fossa. In the patient under consideration this very circumstance took place. Some two months ago the spleen crossed the middle line of the abdomen, but now it falls short of the middle line by some four inches, not because the organ has diminished, but because it has increased in size, and by its increasing weight overcame the supporting power of the pleuro-colic fold, and dropping down towards the left side receded from the middle line of the body. One, therefore, must not assume that a recession of the spleen from the middle line always means a decrease in size, for it is evident the opposite condition may obtain. I would advise you, therefore, when percussing out the normal spleen, or even when you suspect enlargement, to do so with the body in the erect or sitting position; also to search for early enlargements of the spleen high up in the abdomen in the left hypogastric or epigastric regions, or, at any rate, above the umbilicus. You will only find the spleen in the left lumbar or iliac regions when the organ has attained large proportions and receded from the middle line of the abdomen, owing to the inefficiency of the pleuro-colic fold of peritoneum.

II. *The Association between Enlarged Spleen and Hepatic Disease.* — It is regarded well-nigh as an axiom that a cirrhosis, tumour, abscess, or allied disease of the liver necessarily involves a corresponding and consequent enlargement of the spleen. This assumption is not borne out by clinical experience.

(*a*) When the liver is cirrhosed, be the disease in the stage of hypertrophy or fibroid shrinking, it might be expected *à priori* that the venous obstruction would lead to distension of the spleen. It can only be by *à priori* argument, however, for clinical experience shows that in only 50 per cent. of the cases are the two conditions affiliated; that is to say, in half the cases of cirrhosis of the liver there is no enlargement of the spleen. This fact, which can be proved by carefully sifting *post-mortem* records, disturbs the even tenor of our well assorted beliefs and calls for explanation. A cirrhosed liver does not evidently always mean one in which the venous circulation is encroached upon; but seeing that half the evidence is for and half against, it may be naturally asked which is the normal and which the variation; or does splenic enlargement depend on some factor totally unconnected with cirrhosis of the liver, and outside the liver influence? I have no hesitation in saying in this patient the latter is the cause. The patient is suffering from cardiac valvular insufficiency, which has caused at times a pronounced pulmonary congestion accompanied by hæmoptysis. The associated venous fulness of the liver, combined with the unresiliant and inexpansible nature of the hepatic vessels, consequent on fibroid thickening of the tissues through which the portal vessels run, is calculated to produce a splenic engorgement and hypertrophy. In this case I can well believe that had there been no cardiac disease there need have been no splenic enlargement. This is of course a mere assumption; but seeing that in half the recorded cases of cirrhosis of the liver no splenic enlargement exists, and that even in many cases where it does obtain cardiac disease exists as well, the number of splenic enlargements directly dependent on contraction of the hepatic tissue is still further reduced.

Hypertrophy of the spleen, therefore, in uncomplicated cirrhosis of the liver is the exception and not the rule.

(*b*) Fatty degeneration of the liver is not a direct cause of enlargement of the spleen. When the liver is fatty, splenic hypertrophy is the exception. Of course, when other organs are similarly affected to the liver, the effect upon the circulation may be such that the spleen does enlarge to some extent, but such a condition cannot be attributed to the liver alone.

(*c*) Hydatid disease of the liver, even when the hydatids bulk largely, does not necessarily carry with it a hypertrophy of the spleen. Quite the opposite obtains, for in many cases the spleen is actually reduced in size. The spleen itself may be the seat of hydatids, and yet the splenic tissue is not increased in bulk.

(*d*) Amyloid disease of the liver is almost invariably associated with a similar condition of the spleen; yet it not infrequently happens that an amyloid spleen is actually small, in spite of being combined with what would seem to be an obstruction of the portal circulation in the liver.

(*e*) Abscess of the liver is but seldom associated with an enlargement of the spleen. Even when the abscess is huge, and occupying the greater part of one lobe, a splenic enlargement is quite exceptional. In the hepatitis antecedent to abscess, splenic fulness may be found, but when pus has formed the spleen usually returns to its normal size.

(*f*) Cancerous infiltration of the liver may be unattended by splenic enlargements. It is only when the portal or splenic venous trunks themselves are encroached upon by infiltrated glands, or by direct interference of the cancerous growth, that splenic engorgement is to be expected.

All these statements go to show that passive splenic enlargements dependent on hepatic disease are not the rule but the exception, and that the cause and effect usually accepted as existing between hepatic hypertrophy or cirrhosis and enlarged spleen will not bear clinical tests.

(*To be continued.*)

SMALL-POX was very prevalent in Johannesburg last year. The percentage of deaths was 25·7 amongst Europeans and 29·2 amongst the coloured patients. A special note is made of the fact that no deaths occurred amongst persons vaccinated within the last seven years.

Recent Literature on Tropical Medicine.

OPHTHALMOLOGY IN ITS TROPICAL BEARINGS.

THE BACILLUS OF ACUTE CONJUNCTIVITIS.—At the last meeting of the Ophthalmologial Society of Paris, M. Morax exhibited pure cultures of Weeks' bacillus on gelatinised serum, thus refuting, in the most practical manner, the contention of Weichselbaum and Muller that the bacillus only develops in the presence of other microbes. The colonies were punctiform, translucent, and almost imperceptible when confluent; isolated colonies varied in diameter from 1 to 2 mm. The bacilli also develop in bouillon-serum, rendering it turbid. A case of acute conjunctivitis contracted in Egypt yielded Weeks' bacilli of precisely the same characters as those obtained from cases in Paris. Pure cultures instilled for several hours into the conjunctival sac of a rabbit had no effect beyond producing a temporary inflammatory reaction: cultures heated to 100 C°., or filtered through a Chamberland filter, provoked no reaction whatever.

TRICHIASIS IN EGYPT.—M. Lakah, of Alexandria, describes an operation for trichiasis which, in his hands, has been uniformly successful (*Annales d'Oculistique*). He adopts Krone's method, which consists in making an incision in the lid parallel to the free border, dissecting down through the muscular fibres, and then cutting away fine shavings from the cartilage until it becomes flexible and can be bent into the required position, in which it is retained by fine sutures.

For the treatment of trachoma he relies almost entirely on scarification and subsequent lavage with corrosive sublimate solution, finding by experience that this procedure leads to the highest percentage of complete cures.

PROTARGOL IN CATARRHAL OPHTHALMIA.—MM. Fromaget and Degos have just published (*Ann. de la Policlin. de Bordeaux*) a series of cases in which protargol was employed in the form of a 1 in 10 ointment. They found it produced a rapid and permanent cure in all cases of acute and sub-acute catarrhal conjunctivitis; in purulent ophthalmia it was not so successful; in phylctenular ophthalmia it seemed to be quite useless. One of its most marked advantages is the fact that its application is quite painless.

Judging from a recent discussion at the Ophthalmological Society of Paris, protargol has been found most useful in cases of acute conjunctivitis and vernal catarrh, but appears to be on the whole inferior to nitrate of silver in trachoma and purulent ophthalmia; it has been but little employed in this country.

XEROSIS OF THE CONJUNCTIVA.—Nesnamoff (*Wratsch*, No. 49, quoted in *Revue Gén. d'Opht.*, January, 1899) describes his method of treating xerosis, treatment directed partly to removing the diseased condition of the conjunctiva and partly to stimulating the lachrymal gland. With the former object he washes the conjunctiva and cornea first with a solution of chloride of sodium and then with ether; this has the effect of removing the fat from the diseased spots so that they become capable of being wetted by the tears. To increase the lachrymal secretion he employs inhalations of ammonia.

Xerosis of the conjunctiva appears to be due to a reduced state of nutrition of the eyeball, and in Russia is found, especially during and after the long Easter fast. It is common in the tropics, both in its light form, associated with night blindness, and in the graver form spreading to the cornea and producing kerato-malacia. It attacks debilitated persons exposed to strong glare. Herbert, of Bombay, notes its excessive prevalence in that city among poor natives run down by want of food and dysentery (*Trans. of Oph. Soc. of United Kingdom*, 1898). In China it is by no means uncommon, and my friend and teacher, Mr. Treacher Collins, tells me he saw many cases in Persia. In this country it is occasionally seen in Schools (Sydney Stephenson).

SOUTH AFRICA.

ELEPHANTIASIS.

DR. BEN BLAINE in the *South African Journal* writes as follows on elephantiasis:—

"In the *South African Medical Journal* for October, 1897, there was published a paper on the occurrence of elephantiasis in South Africa, read by Dr. Black before the Cape Town Branch of the British Medical Association, and also the subsequent discussion. There seemed to be some doubt in the minds of those present whether the case reported by Dr. Black was really one of elephantiasis, and whether the disease was to be found in South Africa.

"The disease is certainly very rare in this part of the world. Out of considerably over 40,000 patients I have seen during the last seven years, I can only recall two cases of elephantiasis. I have a vague recollection of having seen a third case, but am unable to trace it. In July, 1891, a native woman was admitted, suffering from enormous hypertrophy of the skin of the left arm and breast, the fingers standing out separately. The disease began in the breast, and had been present for some months. The patient was in other respects healthy. It might be objected that the want of symmetry is against the idea of the case being one of elephantiasis; but, on reference to Liveng, I find that he states that in the early stages the disease may be confined to one limb. The case is not fully reported in my journal, but I had no doubt of its nature.

"The next case was admitted in December, 1897. The patient was a young native adult male, about 23, who came from the Kentani district, Transkei. The disease affected the scrotum. He stated that he was about seven years old when the scrotum began to swell, and it had gradually increased to its present dimensions. The whole of the scrotum was affected. The skin of the penis, more particularly the prepuce, was hypertrophied, giving the organ a club shape. The prepuce could not be withdrawn, and projected considerably beyond the glans. The surface was smooth. The scrotum had a circumference of 25 inches and the measurement from perinæum to pubes was 17½ inches. The skin could not be pinched up between the fingers. All over, the natural lines of the skin were much exaggerated. At the lower and back part were several distinct small elevations varying in size, the largest being about the size of a grape. These were sessile. Here also were some cicatrices and a fissure-like ulcer, with a watery discharge.

"The patient died soon after admission, from tuberculosis. On making an incision into the scrotum, the chief hypertrophy was seen to be in the skin and subcutaneous connective tissue, which contained a large number of veins irregularly enlarged.

"A specimen which was removed for examination was unfortunately spoiled.

U.S. AMERICA.

ABSCESSES OF THE LIVER.

DR. EDWARD E. FIELD, in a paper read before the Virginia State Medical Society, in December, 1898, said that among the predisposing causes of abscess might be mentioned:

(1) Alcoholism.

(2) Residence in tropical climates.

(3) Any abnormal condition of the parenchyma caused by malaria, syphilis, new growths, cardiac insufficiency, renal insufficiency, hyperæmia of liver, anæmia of liver, tuberculosis.

It was reasonably certain, he said, that syphilis and tuberculosis did not directly act as exciting causes of abscess, but that the gummata of the former and the broken-down nodular masses of the latter offered a suitable nidus for the development of any of the pus-forming germs which might be present in the systemic or portal circulation.

While some authorities claimed that in abscess in infants

tuberculosis was the exciting cause, the foregoing seemed the most rational explanation. It had also been pretty clearly demonstrated that malaria was only a predisposing cause, as the organism of Laveran seemed to cause a degeneration of the hepatic cells, and had never been known, *per se*, to produce pus under any other circumstances.

Renal insufficiency from sclerosis, causing an imperfect depuration of the blood and a consequent increase of hepatic activity and hyperæmia, and cardiac lesions inducing congestion, predisposed to abscess. The effects of alcohol were too well-known to need further comment.

Residence in hot climates was undoubtedly the most frequent predisposing cause of hepatic abscess. Europeans resident in India were often attacked with abscess through disregard of the hygienic rules of the tropics, although the disease was also prevalent among the natives.

Men seemed to be more subject to the disease, owing to their greater exposure to alcohol and syphilis, the proportion being about thirty men to one woman.

Among the exciting causes of liver abscess should be mentioned in the order of their relative frequency: (1) Dysentery (mainly tropical dysentery), in which the *Amœba coli* was generally present as a causative factor; (2) extension of inflammation from adjacent structures; (3) pylephlebitis; (4) phlebitis of umbilical vein; (5) suppurating hydatids; (6) actinomycosis; (7) trauma; (8) suppuration of gall bladder; (9) typhoid ulcer; (10) tuberculosis.

Tropical dysentery seemed by far the most frequent cause of liver abscess.

Treatment.—When the diagnosis of abscess had been satisfactorily made, the author said the only rational treatment was to evacuate it as soon as practicable.

First, a point of fluctuation should be ascertained, if possible, or even a probable point of suppuration, and an aspirating needle introduced under strict aseptic precautions. A rather large needle should be used, as liver pus was generally very thick, and would not pass through a fine needle. The needle should be pushed deeply into the liver and the piston pulled back, when, if pus did not flow, the needle should be slowly withdrawn, in order that any pus which might have been traversed by the needle might drain into its lumen. Under no circumstances should the piston be pushed down during the removal of the needle, lest pus be forced into the peritonæum or pleura, but the vacuum should be maintained.

If no pus was found, the needle should be withdrawn until the point was near the walls, and, its direction being changed, it should be reintroduced as in the previous manner.

This procedure could be repeated several times without danger, with careful asepsis. It should be done under anæsthesia, as it was quite painful, and if the pleura was punctured, might give rise to unpleasant reflex symptoms. It was claimed by some writers that even if no pus was found this procedure would often relieve the existing symptoms.

Having located the abscess, the needle should be left *in situ* and a dissection carefully made down until the peritonæum was reached; if the incision was below the costal line, and if adhesions to the liver were satisfactory the liver should be opened and the abscess explored with the finger, due regard being had for the adhesions. If there were no adhesions and the case was not extremely urgent, the wound should be packed with iodoform gauze for forty-eight hours, or until adhesions were sufficiently strong to prevent infection of the peritonæum. If the case was urgent, the capsule of the liver should be sutured to the edges of the wound before opening the abscess.

If the abscess was covered by the ribs, it would be necessary to resect about three inches of at least one rib, and, after stitching together the right leaflet of the diaphragm and the capsule of the liver, to open the abscess as in the preceding case. In either event the hæmorrhage would be considerable, but could usually be pretty easily controlled by packing around the tube, which should be of glass, five-eighths of an inch in diameter and fenestrated. After washing out the abscess with hot saline solution or sterilised water a heavy dressing should be put on and an abdominal binder applied. The after treatment should consist in irrigation and dressing the wound at least once a day. In order to cause the tube to drain properly, it should be packed loosely with sterilised gauze for its capillary effect. Owing to contraction of the liver after evacuation of the abscess, the direction of the tube would often be so changed as to necessitate sometimes the use of a curved tube.

The bowels should be kept open. Tonics and a generous diet should be given to combat the large drain from suppuration. It was well to put the patient in a rolling chair as soon as possible after the operation and wheel him into the open air.

Every precaution should be used to prevent bedsores. The patient should recline on the right side as much as possible to facilitate drainage.—*From New York Medical Journal and Georgia Journal of Medicine and Surgery.*

AUSTRALIA.

At the Third Ordinary General Meeting of the Royal Colonial Institute, Sir Horace Tozer, Agent General for Queensland, read a paper on "Queensland Progress" from which we extract the following remarks on the tick pest.

"The most recent pest is, however, the tick (*Ixodes bovis*), which fortunately only affects horned cattle. This insect has an extraordinary life history. In the first stage, that of the ovum, it may remain four months, from which it emerges into visible life in the larval form, and is so small that it appears to the eye like a grain of sand. In this stage it has existed for quite nine months, and never increases in size till attached to a congenial host, invariably horned cattle, though it has been known to fecundate on a horse. After a period of fifteen days in this minute form it begins to expand, till in a few days it reaches the size of an ordinary pea of dull grey colour, when it drops off, and within a short time deposits no less than 2,500 eggs, which in their turn perform similar prolific functions. I have seen a portion of the flank of a beast so covered with ticks that a point of a penknife could hardly be inserted without removing a female tick, or the tiny male insect, which in every case is found nestled under the comparatively huge frame of his larger though not better half. It made its first appearance in Queensland in the end of 1894 in the Gulf Country. The Stock Department was not prepared for its arrival, and then knew very little of its life history. They were lulled into a sense of security by official information from Natal that 'red-water' was not caused by ticks; and treating it as a local disease of a malarial nature, restrictions against travelling were withdrawn, in consequence of which the disease very rapidly spread to the eastern seaboard as far south as the Tropic of Capricorn. It has, however, never made a footing on the western plains, for the sun is its greatest enemy, so that even after four years it is still confined to the more humid districts on the eastern seaboard. Whether it will eventually spread over the southern Colonies depends more on natural conditions. In America none are found north of a particular range of mountains; the chances are that the tick will in time expand over the southern coastal districts, by which time immunity from ill effects will have been secured by inoculation. The Department has now two gentlemen, eminent in their profession—Dr. Hunt, a pathologist, and Mr. Pound, a bacteriologist—and though their conclusions are by no means final, nor their investigations completed, yet it is tolerably certain—

"1. That the tick is identical in all respects with that existing in the Southern States of North America, Argentina, the West Indies, Buenos Ayres, South Africa, India, Ceylon, Java, South Australia, and West Australia.

"2. That dipping is not of much permanent practical utility.

"3. That the disease, of which the most marked symptoms are fever and red-water, can only be contracted by the operation of the tick or inoculation with the blood of a recovered animal.

"4. That it leaves no injurious effect whatever when the animal has quite recovered.

"5. That cattle, the progeny of tick-recovered animals, are immune.

"6. That ticks are carried by so many agencies that quarantine by proclamation or by fencing is only a postponement of the danger.

"7. That it will never extend into the principal pastoral country, as it cannot exist there for any time.

"8. That, comparatively with drought, the losses, though serious in some coast districts, have been small.

"9. That the fever is caused by a micro-organism associated with the tick, but the present unsettled complication is when or where this micro-organism becomes so associated. This difficulty has arisen recently from the discovery of a number of healthy cattle covered with these ticks, which, when removed to another tick-infested locality where more ticks of the same species abound, at once contracted the fever. The harmless ticks were the direct progeny of others which had been on cattle infected with the fever, and caused many deaths.

"It is, however, satisfactory to note that inoculation with the blood of a recovered animal practically ensures immunity from the disease, and it is hoped it will be eventually considered as of no moment, as it is in the other places I have referred to, where no special precautions are used or required."

FRANCE.

"Liver-Cough" an Undescribed Symptom of Hepatoptosis.

In the *Journal de Médecine Interne* of October 15, 1898, M. Louis Vène relates the following case:—A woman had suffered from cough for eighteen years, which since she came under observation (in 1891) was dry and incessant. Drugs were useless. She was examined by a laryngologist and by a number of physicians, including Professor Simon Duplay. The diagnoses of nervous cough and of hysterical cough were made. She happened to break her clavicle, and a bandage was applied for a month, which compressed the thorax and upper abdomen. During this period the cough was never heard, which was attributed to pre-occupation. M. Vène's attention was attracted to a peculiar symptom—the patient never coughed at night. She said, "I sleep very little, perhaps two or three hours a night; but whether I sleep or not I never cough lying down whatever be my position in the bed." Examination showed that the liver extended to the level of the umbilicus in the right hypochondriac region. Hepatic dulness began at a lower level in the upright than in the recumbent posture—at the seventh rib. The thorax was compressed with a flannel bandage extending from the nipples to the umbilicus. The effect was remarkable. The patient had not a single fit of coughing. This result was maintained by bandaging from the pubes to the nipples with rigid material—cotton or flannel—but corsets and complicated elastic belts proved useless. In none of the various observations on hepatoptosis is cough mentioned as an important symptom. But since Hippocrates "liver-cough" has been known, and, curiously, in 1831, when hepatoptosis was unknown, J. B. de Larroque, physician to the Necker Hospital, in a work on "Certain Abdominal Diseases which Simulate, Provoke, and Maintain Diseases of the Chest," distinguishes "liver-cough" due to hepatitis and "liver-cough" coming on in the standing position caused by the weight of the organ. As to the manner of production M. Vène suggests that as the liver is (according to Faure) suspended from the vena cava, which is connected with the diaphragm, excitation of the latter produces the cough. He concludes that before making a diagnosis of hysterical cough the absence of any pathological reflex from the abdominal organs in connection with the diaphragm, and particularly the liver, should be ascertained.—*Medical and Surgical Review of Reviews*, Jan., 1899.

U.S. AMERICA.

Quinine in Malaria.

At the outset H. A. Hare says no one can deny that, so far as the infecting organism is concerned, quinine acts as a specific. He then cites a number of authorities, and deduces the following facts: (1) Quinine sometimes produces hæmaturia in malarial disease; (2) malarial disease often congests, irritates, or inflames the kidney; (3) quinine is capable of doing likewise.

The author does not, however, advise that no quinine be given in malarial nephritis or hæmaturia, but that it be given wisely. Its administration during an attack of hæmaturia is equivalent to "shutting the door after the horse is stolen." In hæmoglobinuria occurring with the paroxysm, there is probably less danger than when true hæmaturia is present, since the kidneys are not so clogged by blood-clots. In a prolonged hæmoglobinuric attack, indicating that the malarial poison is destroying the blood-corpuscles independent of the chills, quinine may be needed. If given, cholagogues, followed by a brisk purge, should be used to aid in eliminating the colouring-matter through the liver and bowels, and to relieve the kidneys. When, through the frequency of the intermittent paroxysms, quinine must be administered, the same attention to the bowels should be given, and the kidneys flushed with diuretics, as the vegetable potassium salts. In cases of severe hæmaturia associated with jaundice and general hæmorrhages from the stomach, bowels, and nose, the virulence of the infection calls for quinine, though the contra-indications are stronger than ever. This malignant form comes on either suddenly with the access of a malarial attack in a patient already broken down, or as an attack of hæmaturic jaundice, without any evidence of another dose of malarial poison. Quinine will be needed in the first of these, but not in the second, which should be treated by other measures for relief of the dyscrasia and hæmaturia.—*Medical Record*, iv., p. 7, and *Merck's Archives*, January 1899.

Medical News.

Dr. Rogers will continue to officiate as Imperial Bacteriologist to the Government of India, during the absence on leave of Dr. Lingard, or until further orders.

The Meteorological Service of India.—It may not be generally known that this is the most important meteorological organisation in the world, and also covers the largest area.

The Plague and the Plague Policy in India.—The Plague, unfortunately, is still making steady—if slow—headway in Southern India, and the returns during the past week from Bombay have been very disappointing. The Punjab Government has just revised its plague regulations, and now every local administration has declared that in fighting the pest medical considerations must give way to social ones.

After some discussion in the Transvaal Medical Society, the following resolution was carried:—"In the opinion of the Medial Council the Government should prohibit the importation from infected places of everything mentioned in the list appearing in the report of the Venice Conference, with the exception of body clothing and luggage, which must be disinfected; and, further, that no infected ship should be allowed, under any conditions whatever, to come into port."

It is with profound regret we see the telegraphic announcement of the death from plague of Major Evans, Professor of Physiology in the Calcutta Medical College. It is supposed that Major Evans contracted the disease whilst performing a *post-mortem* on some one who had died of plague.

News and Notes.

The Medical Faculty of the University of Aberdeen has decided to institute a Lectureship on Diseases of Tropical Climates.

The Edinburgh University Court is advertising a Lectureship on Diseases of the Tropical Climates. The lecturer will be required to give a course of from twenty to twenty-five lectures, the course to include demonstrations and other practical work. One course is to be given during the winter session, and one during the summer session.

The Amount of Salt in Various Seas.—In a ton of water from the Caspian Sea there are 11 lbs. of salt; in the Baltic 18 lbs.; in the Black Sea 26 lbs.; in the Atlantic 31 lbs.; in the English Channel 72 lbs.; in the Mediterranean 85 lbs.; in the Red Sea 93 lbs.; and in the Dead Sea 187 lbs.

Mosquitos and Malaria.—William S. Dodd, in a letter dated August 30, 1898, addressed to the Editor of the *Medical Record*, refutes the hypothesis that malaria is spread by means of mosquitos. The town of Cesaria, in the interior of Asia Minor, which lies very high and dry, has no mosquitos, but during the past ten years many cases of malaria have occurred there.

Correspondence.

THE DISTRIBUTION AND HARMFULNESS OF THE ANCHYLOSTOMUM.

To the Editors of "The Journal of Tropical Medicine."

Sirs,—Burke said that you cannot indict a whole nation. Is it allowable to condemn the fæces of a whole nation? According to Dr. Rogers (*Journal of Tropical Medicine*, October, 1898), Dobson has shown "that upwards of 80 per cent. of the healthy inhabitants of most parts of India harbour this worm (the anchylostomum) in numbers varying from units to hundreds"; and again, "it has been proved by Dobson and myself that from 60 to 80 per cent. of the inhabitants of Assam, Bengal, and many other parts of India, harbour the anchylostomum in numbers varying from a very few to 100 or even more." If the term anchylostomiosis be used in the sense of harbouring the parasite, Rogers states that "three-quarters of the healthy people of India suffer from anchylostomiosis," which is absurd. It will be observed that here is no rough answer as to the prevalence of the worms in India or of the diagnosis of anchylostomiosis by symptoms. We are told the very number of worms the inhabitants shelter.

If the population of Assam, Bengal, and many other parts of India be estimated at fifty millions—I regret being away from all books of reference—allowing one minute for the preparation and microscopic examination of each inhabitant's fæces, working ten hours a day continuously, it must have taken 180 years for Messrs. Dobson and Rogers to attain their results, *i.e.*, ninety years have been spent by each one in this work, "which is absurd." Throughout the paper references are made to the examination of 50+7+72 =129 cases. It is just possible that despite the definite statements, "units to hundreds of worms," we have an example of that abuse of the science of statistics for which medical men evince so much partiality. It is very kind of Dr. Rogers not "to wish in any way to disparage Dr. Galjey's work" in St. Lucia; I must confess that I wish in every way to disparage such statistics as Dr. Rogers's.

Dr. Rogers deals rather with the harmlessness than the harmfulness of the anchylostomum. Most practitioners in the Tropics are aware that, although the parasite be present, it may not be the *causa vera* (in Newton's sense) of the disease, and all will agree that before dosing the patient with thymol, a diagnosis be made. But is it really necessary to repeat these copy-book maxims in every scientific paper? The statements are very properly made in the text-books, and doubtless it cannot be too often impressed upon anyone that diagnosis precedes treatment. But don't repeat these statements as a new gospel every time practitioners are addressed. Probably a healthy man can make use of two or three hundred anchylostoma without evidence of disease. The trouble is that your healthy man so seldom consults you. Having got him, are you able to assure him that he is never likely to be ill? I suppose a couple of hundred anchylostoma is not to be regarded as a prophylactic measure.

As Dr. Manson says, it is as a complication in other diseases that worms are to be dreaded with populations that live in physiological starvation. I am not convinced that even the trichocephalus is quite as innocent as is pretended. But *post-mortems* are difficult to obtain here, and I will not commit myself.

I am, dear Sirs,
Yours truly,
M. D. Eder.

Palmira, Republic of Colombia,
January 7, 1899.

RESIDENTIAL TRAINING HOME FOR MEDICAL MISSIONARY STUDENTS.

To the Editors of "The Journal of Tropical Medicine."

Sirs,—Your issue of this month contains a well-timed and interesting leading article on medical missionaries. Let me add to it an item of information of which the writer apparently is quite unaware. He says: "the Guild (of St. Luke) is about to establish a Residential Home for those who desire to enter seriously on the training which will fit them to be medical missionaries. While attending the ordinary course of instruction at one of the medical schools, these students would have facilities given them for special training in tropical medicine and be surrounded with such counsels and influences as would guide and encourage their missionary aims. In Edinburgh a college of this type has existed for many years and has, unquestionably, done good work. It is the ambition of the Guild of St. Luke to establish a similar home in London."

The information I desire to add is, that such a Home already exists, and has been in active operation for the last thirteen years. The Medical Missionary Association of London was established in 1878, and in 1885 it opened its Residential Training Home for the very purposes which your article describes. A few months ago I had the pleasure of showing the Provost of St. Luke's Guild over the Home, and explaining to him our methods of working. The Home consists of two large houses, the freehold property of the Medical Missionary Association, and has been adapted to suit the purposes of such an institution. It is situated at 47 and 49, Highbury Park, in one of the healthiest parts of the

north of London. Its former students are now scattered in many parts of the heathen world, and to-day, besides eleven students resident under its roof, there are nine outside who have spent three years or more in residence with us, and who are now completing their hospital work outside. The Home of the Medical Missionary Association has only one feature which could distinguish it from the proposed Home of the Guild of St. Luke. It is *inter-denominational*, whereas, if I understand aright, the proposed new Home would be on *strict denominational* lines. The larger proportion of our students are members of the Church of England, but there are also Presbyterians, Wesleyans, Baptists, &c., whereas the new Home would be limited, if I mistake not, to Church of England men. If such is not the case, then there hardly seems any good reason for its coming into being.

Let me conclude by saying that the Committee of the Medical Missionary Association is in thorough sympathy with the suggestion of your article, that intending medical missionaries should be versed, as far as possible, in tropical medicine, and should be "adequately trained for scientific observation." Six of our present students are University of London men; two passed their primary Fellowship during this year, another is already F.R.C.S., and four others are aiming at the same. The day is past for intending medical missionaries to be other than men who, for the very highest reasons, are determined to be thoroughly abreast of their profession, and who, while looking forward to take their full share of privilege in seeking to bring their patients into believing, living union with Christ, are ready also to do their part in elucidating some of the many problems associated with the diseases of tropical lands.

JAMES L. MAXWELL, M.D.,
Secretary of Medical Missionary Association, London.
49, Highbury Park, N.,
December 17, 1898.

[Crowded out of January and February numbers.]

BIRTH.

SEAL.—At Darjeeling, on February 22, 1899, the wife of Charles E. Baldwin Seal, M.R.C.S.Eng., & R.C.P.Lond., of a daughter.

Communications, Letters, &c., have been received from:—

C.—Dr. Albert Cook (Mengo, Uganda); Dr. Frank Cantlie (Swatow).

F.—Dr. Jean Fonstanos (Greece).

H.—Staff-Surgeon R. Hickson, R.N. (Mediterranean Station).

M.—Surg. W. B. Maurice, R.N. (Mediterranean Fleet); Mr. K. Munro (Westminster); Dr. G. E. Murray (Johannesberg); Capt. Mulvany, I.M.S. (London).

S.—Dr. Stephens (Mediterranean Station).

T.—Major Tyrrell (Punjab).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Medicale.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
New York Medical Journal.
Pacific Medical Journal.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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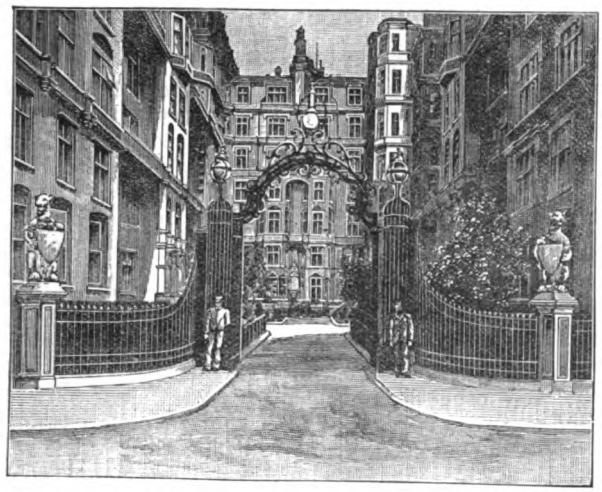

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SPECIAL ACCOMMODATION FOR INVALIDS.

Original Communications.

LEPROSY IN INDIA.[1]

By H. A. Acworth, C.I.E.

The statement of objects and reasons appended to the Bengal Bill No. III., of 1895, which ultimately became the Bengal Lepers Act No. V. of 1895, begins with the following words :—

"The expediency of segregating pauper lepers under proper safeguards, and of forbidding lepers generally from exercising certain trades and callings connected with human food, drink, and clothing, was urged by the Leprosy Commission which visited India in 1890-91. Experience in Bombay having shown that these recommendations can be successfully carried into effect, the accompanying Bill has been prepared, &c."

On July 30, 1896, a Bill was introduced into the Legislative Council of the Government of India, which became in time the Lepers Act No. III. of 1898. The statement of objects and reasons attached to that Bill opens with these words :—

"The expediency of segregating pauper lepers under suitable safeguards, and of forbidding lepers generally from following certain trades and callings connected with the bodily requirements of human beings, was urged by the Leprosy Commission. . . . Last year an Act was passed by the Bengal Council &c. . . . The present Bill has been prepared on the lines of the Bengal Act."

On the introduction of the Bill in the Government of India Legislative Council, the mover, Mr. Woodburn, now Sir John Woodburn, Lieutenant-Governor of Bengal, made the following observations :

"In 1890-91, a Leprosy Commission visited India, and a couple of years later submitted a report to the Government of India, pressing on it very earnestly two questions, the segregation of lepers, and the restraint of lepers in certain callings in which they were brought into immediate contact with the food and clothing of their neighbours. The Government of Bombay had already taken action in that direction, with the help of a very munificent donation from Sir Dinshaw Petit. They constructed in 1890 a lepers' asylum in Bombay. That asylum, I believe, contains accommodation for about 300 lepers, and the result has been to free the city of Bombay from the beggars who extorted alms by the exhibition of their sores. The unfortunate creatures subjected to this dreadful malady have now been removed to a hospital, in which that comfort and attention are given to them to which their pitiable condition gives them a just claim. The Government of Bengal followed that example last year, and passed through their Local Council a Bill for the two purposes I have mentioned; the segregation of lepers, and their prohibition from certain callings."

The object of the writer in making these references is two-fold; first, to point out that the legislation undertaken in the Councils of Bengal, and of the Viceroy, was avowedly based upon recommendations made by the Leprosy Commission; secondly, that both Governments acknowledged that an example set in Bombay had encouraged and induced them to undertake that legislation. Sir John Woodburn's account of the action taken in Bombay is hardly accurate; but the error is one which has no interest except for the present writer, who will be excused, perhaps, for reverting to it further on. It will suffice to say here that the Government of Bombay never erected a leper asylum in Bombay, and that though Sir Dinshaw Petit offered a lakh of rupees for the purpose, it was never spent.[2]

Now is it true that the Leprosy Commission recommended the segregation of lepers; meaning, of course, their compulsory segregation? In both Councils their authority was appealed to in justification of legislative measures for the enforcement of a system of compulsion; and the word segregation, as used in these Councils, must clearly be understood in this and no other sense.

Among the definite conclusions at which they arrive, the Leprosy Commissioners say :—

"3.—Though, in a scientific classification of diseases, leprosy must be regarded as contagious, and also inoculable, yet the extent to which it is propagated by these means is exceedingly small."

If this be so, the writer must confess that he fails to understand on what principle, save perhaps that of heredity, segregation, or voluntary isolation, can be defended as of any value.

[1] Read at the Indian Section of the Society of Arts.

[2] The asylum, built in 1893, was at first known as the "Acworth Asylum," and was so styled in Government resolutions. The writer requested (and wrote accordingly to Government) that the name might be changed to the "Matoonga" Asylum, after the suburb in which it was built.

Going on to "Practical Suggestions," the Commissioners say that segregation "may be voluntary or compulsory, and in either instance partial or complete. Complete segregation has never yet been possible." They then consider the examples set by the Sandwich Islands, and Norway, and go on to say:

"For India, complete compulsory segregation may be considered to be absolutely impracticable. Neither do the conclusions given . . . as to the nature of the disease, justify any recommendation for absolute segregation. The presence of a leper in a healthy community is a source of danger no greater than the presence of an individual suffering from tuberculosis. Both diseases are contagious in an equal and minimal degree. The amount of ulceration which exists in both diseases is to some extent a measure of the danger of contagion.

"It is impossible for the same reasons to advise compulsory partial isolation. Voluntary isolation is, therefore, the only measure left for consideration. Among civilised communities the separation of those suffering from many diseases other than leprosy is encouraged. The voluntary isolation of the leper is, therefore, no exception to this custom. For this reason the Commissioners recommend the adoption of a voluntary isolation as extensive as local circumstances allow. Further, by permitting marriages among lepers, the plan suggested might be the more easily carried out."

The Commissioners then go on to recommend that the crowding of lepers into large towns should be discouraged, for a variety of general reasons, but not because there is any risk of their diffusing the contagion of leprosy; that municipal bye-laws should be passed, preventing "vagrants suffering from loathsome diseases from begging in or frequenting places of public resort, or using public conveyances;" also that leper asylums should be built near towns, and that the "authorities should have the power of ordering lepers infringing the regulations, either to return to their homes, or enter an asylum." They also recommend that lepers should be debarred from following occupations which concern the food, drink, and clothing of the people, but guard themselves by adding that they recommend this "quite apart from the dread of a possible infection."

"In no case," say they, "would the Commissioners suggest an Imperial Act, especially directed against lepers as such, for these are far less dangerous to a community than insane or syphilitic people."

The writer would here observe that he does not presume to state any opinion of his own on the questions of the hereditary or contagious character or otherwise of leprosy. For information on these points, he goes to the leaders of medical science; though they, unfortunately, may speak with an uncertain voice. All that the writer, and those who, like himself, have been concerned with the question of leprosy in its administrative aspect, look for, or are capable of discussing, is a working hypothesis. It was, he believes, Dr. H. Vandyke Carter—*clarum et venerabile nomen*—who spoke of contagion as a "good working hypothesis." Dr. Vandyke Carter was, nevertheless, for many years a non-contagionist, though the writer is not sure whether in later life the views of that most eminent man were not modified or changed.

The references which have been made to the report of the Leprosy Commissioners make it abundantly clear that the legislation which has been undertaken in the Bengal Council, and in that of the Viceroy, would never have had their concurrence, and that to have quoted them, and their recommendations, in support of it, seems to indicate a misapprehension of their views.

The writer must repeat that if the contagiousness of leprosy may for practical purposes be disregarded, if it is not a "good working hypothesis," he is at a loss to comprehend why lepers should be singled out as the objects of any of the measures which the Commissioners deem to be justifiable. It seems to him that the Commissioners, however convinced they may have been of the accuracy of their opinions, had not what is commonly called the courage of them. They repudiate the risk of the disease being diffused by contact, and then recommend, or suggest, or half suggest, a series of measures which cannot be justified on any other grounds. The words "half suggest" are meant to indicate the recognition of the value of isolation, coupled with the whittling away of the value of it by making it voluntary. The Commissioners must have been aware, by the experience gained on the spot, that in India voluntary isolation is no isolation at all, and that a voluntary asylum has almost no effect whatever in relieving the streets and public places of a town from the presence of lepers. Dr. G. A. Hansen, in an article in *The Lancet*, in October, 1893, dwells on the contradictory character of this part of the report.

The Committee of the National Leprosy Fund, in commenting on their Commissioners' report, expressed their "entire dissent from the conclusion that segregation was either impracticable or undesirable." "They entertained a precisely opposite opinion," and "would be sorry if the Government of India were encouraged by the report of the Commissioners to refrain from taking the necessary steps in the direction of such segregation of lepers as may be found possible." They then expressed their concurrence in views formerly expressed by Dr. Vandyke Carter, and which favoured the erection of leper asylums, the establishment of leper colonies or villages, in all of which compulsory segregation should be enforced, together with the strict isolation of leprous subjects retained at home at the express wish of their friends.

This last provision has been enforced in Norway since 1885, when a law to that effect was passed at the instance of Dr. Hansen, the Inspector-General of Leprosy, and it illustrates the view of that authority that separate sleeping accommodation, a separate food apparatus, and strict cleanliness of house and person, are "usually isolation enough to prevent the spread of leprosy."[s]

It is interesting to note that the first name signed to the committee's memorandum is that of George N. Curzon, then Under-Secretary of State for India, and now Viceroy of India. Of the medical men who also signed it, two only failed to append minutes of dissent from the views of the committee as to compulsory

[s] Hansen, *Lancet*, October, 1893—"Hansen and Looft on Leprosy," 1895.

segregation. Mr. Jonathan Hutchinson, Sir Dyce Duckworth, Sir Joseph Fayrer, Sir Andrew Clark, Sir Guyer Hunter, Sir James Paget, all dissented from those views.

Such being the array of expert knowledge and talent in favour of the Commissioners' views upon the question of compulsory segregation, is there any likelihood that, circumstances remaining as before, any legislation in that direction would have been yet undertaken in India? The writer is firmly convinced that there is not;[4] and that in spite of the minute of the committee of the National Leprosy Fund, the Governments in India would not have moved if circumstances had remained as before. If, indeed, either the report of the Commissioners or the minutes on it had supplied the necessary stimulus, it may be alleged with some confidence that the Government of Bengal would not have waited for two years, or the Government of India for three years, before dealing with the question. It was the stimulus supplied and the example set in Bombay which were really the parents of legislation at Calcutta and at Simla.

The question of leprosy as an administrative problem had become urgent in the city and presidency of Bombay some considerable time before the death of Father Damien (April, 1889), or the foundation under the chairmanship of H.R.H. the Prince of Wales, of the National Leprosy Fund (June, 1889), but it was the creation of this fund, and, in particular, the association with it, and the energetic leadership and control, of His Royal Highness, which furnished the momentum, under which a solution of the question was rendered possible. Our great English moralist says:—

> "How small of all that human hearts endure.
> That part that Kings or laws can cause or cure."

There may be some truth in this, as a general proposition, but it has no correct application to the present question, or to our Kingly Commonwealth of England. On the contrary, it may be confidently asserted that it was to the personal influence and capacity of H.R.H. the Prince of Wales, that we owe it that the question of leprosy in India was made ready and ripened for solution, instead of being allowed to linger on as an unmanageable administrative difficulty for an unknowable series of years.

By the census of 1881, the number of lepers in the Bombay Presidency was 10,095, and in the rest of British India, 108,858, or a total of 118,953. By the census of 1891—which was after the period which the writer is about to consider—there were 10,187 lepers in Bombay, and 94,510 in the rest of British India, to which, since 1881, Upper Burma, with 3,504 lepers, had been added. There was a great diminution in 1891, as compared with the figures of 1881, in Bengal and Madras, the numbers being, 76,079 for Bengal, and 11,967 for Madras in 1891, against 92,181 and 14,088 in 1881. In Bombay the numbers had remained nearly the same.

The census figures can only be taken as an approximation to the truth, for there can be little doubt that numbers of persons who were affected only with what is called white leprosy, had been included as lepers, and, on the other hand, that some true lepers had escaped enumeration. In the early stages of the disease, true leprosy is often very difficult, and to a layman impossible to detect. The writer has seen many cases in which the only indication of leprosy has been a small patch of discoloration on the skin, very often covered by the clothes. He remembers one case in the Matoonga Asylum, in which the patient was a fine, robust, healthy-looking young woman of the Maratha caste, who showed no sign of the disease, excepting a small light-coloured patch, called by the doctor an anæsthetic patch, high up on the inside of the left thigh. In another case, a female child of six years' old, there was one such patch on one arm, and another on the buttock (right hip). Photographs of the latter case were included among a number which he sent to the Medical Secretary of the National Leprosy Fund. Such cases as these would never have been included among lepers by any census enumerator.

Without going into the classes into which true leprosy is divided, nodular or tuberculous (lepra tuberculosa), anæsthetic (maculo-anæsthetica), and mixed, if a separate division is now recognised under the title of mixed, the writer would point out that in Western, and he believes in other parts of India, two kinds of so-called leprosy are popularly recognised. One of these is black leprosy, and called in Marathi Raktapiti, and the other white leprosy or Kor. The former is true leprosy; the latter, Vandyke Carter says, has "hardly anything in common with true leprosy." . . . "It is included among a group of skin diseases, known as 'Kushta.' The leprosy of the Jews was clearly the same as the 'Kushta,' of Hindu writings. . . . The expression leprous as snow can only refer to some skin disease, probably 'Kor.' It cannot possibly be true leprosy. 'Kor' and 'Raktapiti' are totally distinct." "What is called 'White Leprosy,'"[5] says Mr. Jonathan Hutchinson, "is usually leucoderma, a disease which has no relation to true leprosy."

White leprosy, therefore, kor, or leucoderma may be eliminated from consideration altogether. Every measure contemplated or effected in India for dealing with leprosy has had relation to black leprosy, or raktapiti.

There were several leper asylums in the Bombay Presidency in 1889. There were two in Belgaum, one at Ratnagiri, and one at Trombay, close to Bombay. This was a very small one, accommodating about twenty patients, and had been established by the charity of a Parsee gentleman of the All Bless Family. There was also at Byculla, in the heart of the city of Bombay, a leper dharmsala, attached to a dharmsala for indigent persons. They were established by Sir Jamsetjee Jeejeebhoy; an endowment provided for a small daily issue of food and money, and in 1889 this dharmsala contained from 150 to 200

[4] The writer says "*yet* undertaken." He has no doubt, seeing the position he occupied on the Committee of the National Leprosy Fund, that the able and resolute man, who is now Viceroy of India, would not have waited long after his arrival to initiate a measure for the segregation of lepers.

[5] H. Vandyke Carter, Trans. Medical and Physical Society, Bombay, 1871; No. XI., p. 74.

lepers from all parts of the Presidency. Dr. Vandyke Carter had noted in 1876, in a report on leprosy in Kattiawar, that it was customary to send bad cases from that province to the leper dharmsala at Byculla.

It may be added that there was a leper ward attached to the Central Jail at Yerrowda, near Poona, to which leper criminals, if on long terms of imprisonment, were sent. The lepers here were isolated, and did not mix with the healthy prisoners. Compulsion, of course, is part of a prison administration, and there was no difficulty in enforcing it at Yerrowda; but the fact that it was enforced showed that where it was possible to resort to it, the necessity for compulsory isolation had already been recognised as a "good working hypothesis." The case was the same in Madras, where a ward for criminal lepers had been attached to the leper asylum in that city.

In every part of the city of Bombay lepers were, in 1889, more or less in evidence, in every stage of the disease, and exhibiting in the public streets every one of its hideous deformities. On the occasions of Hindu festivals they crowded to the temples on the sea shore, lining the streets in scores and hundreds. They occupied the verandahs and courts of empty houses. They washed and drank from the public taps. They scattered their rags about shops and markets, and instead of using, like Job, potsherds to scrape themselves withal, they used rough stones from the heaps of road metal stacked at the street sides.

For some years a sense of this evil had been growing. In 1883 the Roman Catholic Bishop of Bombay had applied to the Government for the grant of one of the old forts of Sion, Matoonga, Warli, or Sewri, to turn into an asylum for lepers. His request was not complied with, but the Government intimated that they would gladly see an asylum established in Bandora or in Kurla, outside the limits of the city. A year or two afterwards the tiny asylum at Trombay, already referred to, was established, and also the asylum at Ratnagiri. In February, 1888, the Municipal Commissioner (Mr., now Sir Charles Ollivant) called on the Health Officer for a report with reference to the leper dharmsala at Byculla. Early in 1888 the Inspector of the Education Department drew the attention of the Government to the fact that a colony of lepers had established themselves in close proximity to some large schools; and being also a member of the Bombay Municipal Corporation, he brought the matter forward in that body, desiring to know what powers the Health Officer required to deal with the evil.

The Commissioner of Police in June, 1889, reported that lepers were on the increase, that he could not deal with them, because there was no place to which they could be sent; that it was useless to place them before the magistrates as vagrants, for the magistrates would not send them to jail; that there were 430 lepers in the city at the last census, and at the time of writing probably twice as many; that if Section 424 of the Municipal Act was put into force he could clear the city of them in a few days, supposing there was any place to send them to; and that the necessity of clearing the city of them and isolating them was urgent. The Municipal Commissioner, in sending on this letter, said that the existing provisions of the law were totally inadequate.

In August, 1889, the Government of Bombay directed the Municipal Commissioner to remove the lepers from the place complained of by the Educational Inspector. They added that it was the duty of the municipality, under Section 424 of the Bombay Municipal Act, to remove persons suffering from dangerous diseases to hospitals or asylums, and if there were no suitable hospitals or asylums in existence they should be provided.

To issue such an order as this from the Secretariat was easy enough; to carry it out was quite another matter. Supposing the estimate of the Police Commissioner as to the number of lepers to be accurate, and that there were 860 requiring removal, it would have cost, on the basis of the expenditure afterwards incurred at Matoonga, and which was rigidly economical, at least three lakhs of rupees to build an asylum for them, even supposing the land to have been obtained free, and a monthly expenditure of Rs. 8,600, equal at 4 per cent. to a further capital of nearly 26 lakhs, to maintain them. Moreover, the law quoted by Government gave no adequate power to any authority. Section 424 of the City of Bombay Municipal Act, III. of 1888, runs nearly as follows:—

"The Commissioner, or any Police Officer empowered by him in this behalf, may, on a certificate from the Health Officer, or any duly qualified medical practitioner, order the removal of any person suffering from a dangerous disease, and who is without proper lodging or accommodation, or who lodges in a building occupied by more than one family, to a hospital where such diseases are treated."

This is an extremely valuable provision in its way, and is constantly acted upon in cases of small-pox, cholera, and so on, but its futility in cases of leprosy consists in this, that though it gives power of *removal*, it gives none of *detention*. A person suffering from cholera or small-pox is generally glad to be removed to a hospital, and glad or not, is physically incapable of leaving it until he has recovered; then he is allowed to leave it, and no one tries to detain him. But the case of a leper was quite different. He was as well able to leave the hospital the hour after he was taken there as he was when he came in, and if he wished to do so no one could prevent him. The section was, therefore, useless as applied to lepers, and the orders of Government dead at their birth. The Corporation realised this, and simply "recorded" the order, without attempting to take action on it.

Before matters had reached this stage the foundation of the National Leprosy Fund, and the public action in connection with it of H.R.H. the Prince of Wales, had stirred into movement the *non possumus* of India. In June, 1889, the Supreme Government issued a Resolution on the subject of leprosy, which, together with a draft Bill for dealing with lepers, was circulated to the various provinces for opinion. The main features of this Bill were as follows:—

A leper was defined to be "a person certified by a medical practitioner, having from the local government general or special authority, to be suffering from leprosy." The writer may observe in passing that in his opinion this definition is preferable to that which was subsequently embodied in the Bengal and

Government of India Acts, which became law. The Bill empowered local governments to establish retreats, to which lepers might voluntarily apply to be admitted for life or a term of years, being liable to punishment if afterwards they escaped from them, and to which vagrant lepers might be sent by magistrates for detention. Provision was made for the inspection and management of these retreats, in which, by a further provision, the sexes were to be separated from each other. Local bodies were empowered to spend money on such retreats, and local governments were authorised to devote to them any monies which they held at their disposal for expenditure on hospitals or asylums.

It may be observed here that the Government of the Central Provinces had, in the previous year, asked the Government of India to include powers for the compulsory detention of lepers in the Municipal Bill for those provinces which was at the time before the Supreme Government.

The reports on this draft Bill were on the whole unfavourable to it. Particular stress was laid on the circumstance that it contained no adequate provision for dealing with rich lepers. The Bill was not proceeded with.

In January, 1890, the Government of Bombay laid their hand upon an Act, to which their attention had been drawn some time before by their solicitor, and on the strength of which the asylum at Matoonga was afterwards established. This was Bombay Act VI. of 1867. It does not appear that the Government realised how this Act might be used to supplement or supersede the insufficient coercive provisions of the City of Bombay Municipal Act. The writer thinks that this discovery was left for him to make. But it was useful, and used for other collateral purposes.

The Act is entitled "An Act for the better sanitary regulation of the City of Bombay," and the preamble states that it is intended to have special reference to vessels using the port. It gave Government power to establish by notification sanitaria "for the segregation of persons suffering from any infectious disease dangerous to life," and to notify what diseases should be held to be such for the purposes of the Act.

On January 8, 1890, the Government issued a notification, declaring "black leprosy" to be an infectious disease dangerous to life, and further declaring the asylums at Trombay and at Ratnagiri to be sanataria under the Act. Three weeks later they required of the Police Commissioner of the city that he should deal with the evil, inasmuch as "places had been provided for the treatment of leprosy."

Now the Trombay Asylum would hold comfortably 18 or 20 patients, and at a pinch, 25; the Ratnagiri Asylum would hold at the outside 80. Moreover, Ratnagiri was distant 120 miles from Bombay by sea, and was almost inaccessible for a sick person, certainly for a crowd of them, by land. There were, ex-hypothesi, at least 800 lepers in Bombay. Even if Ratnagiri could be reached, yet the asylums there and at Trombay would together hold only an eighth of them. The action of the Government opened no way out of the impasse. The Police Commissioner was ordered to make bricks without straw.

At this juncture, Sir Dinshaw Petit offered one lakh of rupees for the erection of a leper asylum, on the conditions that it should be called after his name, and that the Corporation or Government, or both, should equip and maintain it. The Corporation agreed to the terms, but hoped Government would contribute. On March 25, 1890, the foundation-stone of this asylum was laid at Trombay by an illustrious prince, who was then visiting India, and whose early death no long time after threw the whole empire into mourning. Plans and estimates were ordered to be prepared, the accommodation to provide for 1,000 lepers.

It may be observed here that no part of this money has ever been spent, and that this scheme has never advanced from that day to this. It was found, to begin with, that accommodation for 1,000 lepers in buildings sufficiently solid, and, if I may use the word, "pucka," to satisfy the Government engineers, would cost nearer five lakhs than one, and the necessary funds were not available. While the plans were being considered, and during the pause which ensued on this discovery, the asylum at Matoonga, intended at first as a temporary expedient to bridge over the period till the new asylum at Trombay should be ready, was started, enlarged, and gradually acquired its present permanent character, and for various reasons, among others, the much greater convenience of the situation, has superseded the Trombay scheme altogether.

The writer succeeded to the office of Municipal Commissioner of Bombay on May 1, 1890. It was by that time becoming very plain that a long time must certainly elapse before the asylum at Trombay could be built, if ever it was built, and the question of what to do with the lepers of the city was every day more and more pressing.

The writer has no intention of dwelling at any length upon the measures which he took to dispose of the difficulty. It was soon clear to him that the best, indeed the only, course was to elicit from the public charity—that charity which the natives of Her Majesty's Indian Empire always, and everywhere so nobly show when the necessity for it has been made clear to them—funds sufficient to provide a temporary home for vagrant lepers, until the expected permanent accommodation was ready. It would be egotistical, and would interest nobody except himself, to recount the measures he took towards this end. It suffices to say that by a sustained and organised effort funds were raised, funds in the issue considerably exceeding the sum offered to Government by Sir Dinshaw Petit. But the raising of these funds was the least and the easiest portion of the undertaking. Two things presented themselves as difficulties requiring preliminary solution, one the question of maintenance, the other the far greater and wider question of segregation. As regards the first the writer applied to Government and to the Municipal Corporation to guarantee a certain monthly sum per head of lepers received into the asylum. In each case a generous response was made. Government and the Corporation each agreed to contribute Rs. 10 per head up to the number of 150 lepers, or a joint total of 300. In mentioning this, the writer must needs mention the names of Lord Harris, then Governor of Bombay, and of Mr. Pherozeshah M. Mehta, now the Hon. Pherozeshah M. Mehta, C.I.E. Lord Harris was the first, and a very liberal, subscriber to the fund, and though the

writer cannot assert, yet he has no doubt, that it was primarily to his influence that he owed the consent of Government to the large contribution they made towards maintenance. He owed a similar debt in the Corporation to Mr. Mehta, and here he has a full knowledge of all that passed, and is able to state that, generously disposed as the Corporation undoubtedly were, yet it was to Mr. Mehta's commanding influence and ability that he is principally indebted for the readiness of their response to his appeal.

The question of segregation seemed for a long time to be an insuperable difficulty. The chief object to be aimed at was the purgation of the city. To this end a voluntary asylum would have been useless. If lepers were to come and go, the streets would never be clear of them. At all times the majority, on each of the numerous occasions of a festival would infest the streets as before, begging and exhibiting their deformities. It was absolutely necessary, if any good was to be done, that the real crux of the leper question which had never yet been grappled with, and on which every scheme had hitherto been wrecked, should be resolutely and decisively faced, and disposed of.

It was clear that in the Municipal Act no help was to be found. After other vain explorations in the arcana of legislative enactments, Act VI. of 1867 (Bombay), was turned to, and after careful study, it seemed to give all the necessary powers. Under the third section of the Act, the Consulting Officer of Health, or in his absence, the Health Officer of the port, had the power to remove any vagrant who might be suffering from a disease notified under Section 12, to a sanatorium notified under Section 1; and any person so removed was "*bound to remain there until one of the said Officers of Health certified that he might go abroad without danger to the public.*" Now, over the Health Officer of the port the Municipal Commissioner had no control, but under Section 76 of the Municipal Act the Health Officer of the municipality was made Consulting Officer of Health for the purposes of Act VI. of 1867. The Health Officer of the municipality was subordinate to, and was indeed the head of one of the departments under the Municipal Commissioner. The writer, who filled the latter office, began therefore to see his way sufficiently clearly to justify him in proceeding to collect subscriptions. As soon as a certain sum had been realised, building was begun. The site chosen was at the village or suburb of Matoonga, within the limits of the city, but on its further boundary, and near the head of the harbour. Though not far from one of the main roads leading northward out of the city, it was completely concealed from it, and was, in fact, so effectually isolated, that, as the Health Officer observed, it was "anatomically twenty miles from the city." In former days troops had been quartered at the place, and the long high embankments or plinths upon which the barracks had stood were very suitable for the erection of wards, and their existence saved much expense. The ground belonged to the Municipality, and it was not the least of the services of the Corporation to the cause that they never demurred to its occupation, which the writer had no authority to have effected without their previous concurrence. But in all matters connected with the leper asylum, the Corporation of Bombay have shown a large and noble liberality.

The asylum was at first intended to be of a temporary character, but if it was to resist the monsoon of Bombay, strength was essential. Economy also was a primary requisite. The plans and the character of the structure were the work of the Municipal Chief Engineer, Mr. Rienzi Walton, now one of the Engineering Inspectors of the Local Government Board, without whose talent and energy the buildings could not have been erected so quickly as they were, or on such satisfactory lines. Though at first called temporary, and constructed to a great extent of iron (angle and T irons with brick noggin), so as to be easily removed, they may, as a matter of fact, defy the wear and tear of centuries.

The wards, with all their adjuncts, which now include, besides lavatories, storehouses, dispensary, and so on, a Hindu temple, a Mohammedan mosque, a Roman Catholic church, and a very useful hospital ward (built at the cost of the benevolent Bai Dinbai, widow of Mr. Nusserwanjee M. Petit), cover several acres of ground, and are surrounded by a high fence of barbed wire.

On November 6, 1890, sufficient work had been done, though building had only begun on August 19 previous, to provide accommodation for somewhat less than 100 lepers. Those who were concerned with it will not forget that day, the first on which compulsory segregation was applied to lepers in India. The police had received due warning, and the necessary orders had been issued by their Commissioner, whose valuable co-operation had been readily secured. On the morning of November 6, 40 or 50 lepers were brought in by the police from the streets, and conducted to a locality where they were to be inspected by the Health Officer (Surgeon-Major Kirtikar). They were all carefully examined, the necessary certificates were issued, and they were removed in carts (afterwards carefully disinfected) to the Matoonga Asylum. On the next day similar action was taken, and so on, until the available space was filled. As the asylum grew, further removals took place, and when the writer left India in April, 1895, the full number of 300 was rather exceeded; there were about 310 patients in the asylum.

Two sets of prophecies attended the establishment of the Matoonga Asylum; one, that it would be so popular that it would soon be over-crowded; the other, that it would be impossible to coerce the lepers into staying there. Both have proved wide of the truth, though the former is nearer to the facts than the latter.

The asylum became in course of time sufficiently popular to keep it filled, and even a little more than filled. It did happen now and then that a leper seeking admission had to be turned away, but these were occasional cases. Room was always found for lepers sent there by the police. It must be understood that after the first few months the intervention of the police was confined to sending to the asylum casual vagrants arrested for begging, and who would, if they had not been lepers, have been placed before a magistrate.

In its earlier days, the asylum was distinctly unpopular. The wholesale sweeping up of lepers from the streets, and the confinement of the asylum were both resented. The writer was once disposed to attribute the diminished number of lepers in the city, which was disclosed by the census taken in February, 1891, four months after the asylum had started, to an exodus caused by the coercive measures which had been adopted; but though he still thinks there was some ground for that belief, he has since come to the opinion that he exaggerated it, and that though a few lepers may have left Bombay on that account, yet the real fact was that both he himself and all the other authorities of the city at that time had over-estimated the number of lepers within it. He has already referred to the Police Commissioner's estimate that there were at least double as many in 1889 as there were in 1881, and no one put the number at less than 800 to 1,000. But the writer greatly doubts whether 500 would not have been an over-estimate. The writer never thought or said at any time that he believed leprosy to be on the increase in India; but he did believe that with the improvement of communications both by land and sea, the natural gravitation towards a great city, that is, towards a convenient centre for begging, had added to the number of lepers in Bombay itself. He still thinks it had done so, but not to anything like the extent which he at one time supposed to be the case.

The Matoonga Asylum, however, built to contain 300 patients, might reasonably be enlarged so as to contain 500.

For some weeks after the asylum was established, it was thought advisible to keep a small guard of police there. But in spite of this, and in spite of the fact that it was surrounded by an 8-foot fence of barbed wire, there were several cases of escape from it in the first few weeks, perhaps a dozen from first to last, and there was a good deal of discontent within the asylum. The truth is that confirmed habits of vagrancy are difficult to eradicate, and that lepers, like tramps in this country, prefer a life of freedom and irresponsibility, though it be one in which their sustenance is precarious, to regular habits, and sufficient food and clothing, coupled with restraint, however lightly applied. Perhaps the vagrant leper may often have passed a day or two without a full meal, but then he very often got opium, sweetmeats, and other luxuries, and forgot his sufferings in the enjoyment of them. It is true that when the pinch of privation was severe, large numbers would have voluntarily sought the asylum; but after good feeding for a day or two they would have left it again, and resorted to their former wandering habits. For this reason a voluntary asylum in India will never clear a place of lepers, and as a matter of fact, never has done so. There is, for instance, a most admirable leper asylum at Madras, constructed on the best lines, and managed to perfection; but when the writer visited it (in March, 1891) he found the streets of Madras full of lepers.

One difficulty which was felt at the Matoonga Asylum, when first started, arose from the opium-eating habits of the patients, and though the writer for a short time adhered to the resolve not to allow the issue of opium, he had at last to give way, and small and diminishing quantities were allowed under the strict control of the medical officer. It was found possible to reduce the quantities by degrees, and the habit was gradually almost or altogether eradicated.

Every effort was of course made to render the asylum as comfortable a residence as possible. The diet was on the most liberal scale, the clothing was excellent, tobacco was allowed to both men and women who cared to have it, and the strictest regard was paid to cleanliness. At the same time, every remedy, medical and surgical, by which the misery of the patients could be alleviated, was at their disposal. A medical officer lived on the spot, and he was under the control of a doctor of superior rank and attainments, who daily spent from two to three hours at the asylum. The latter again was under the orders of the Health Officer to the Municipality. The whole asylum was directly ruled by the Municipal Commissioner.

After a few weeks, it was found possible to remove the police guard; and as the reputation of the asylum grew among those whom it was intended to serve, as they learned how greatly health and comfort were promoted by residence there, and how ample a measure of freedom they might still possess within its wide boundaries, attempts to escape came entirely to an end, and the difficulty of finding room for those who wished to come in was substituted for the difficulty of retaining them there.

All classes of natives were admitted, and there were from time to time a very few Europeans. A question soon arose as to providing for the spiritual requirements of the inmates. The writer did not feel at liberty to do this out of the general funds collected for the erection of the asylum. He therefore made special appeals to Hindu, Mohammedan, and Christian gentlemen; and out of the funds by them contributed for the purpose, he built a small Hindu temple, properly fitted up, a small mosque, and a small Roman Catholic church, and suitable arrangements were made for the attendance of priests of the different religions. The writer's action in this respect exposed him to some comment, and he was accused of furthering the cause of idolatry; but for such accusation he cared and cares little. The inmates of the asylum were, without having committed any crime, prisoners for life, and he felt it to be his clear and sacred duty to provide, so far as he could, for all their legitimate requirements; and he thought, and thinks, that the claims of a Hindu or a Mohammedan for a place of worship according to his belief was a requirement as legitimate as that of a Christian for a church.

Perhaps the most painful feature of the asylum was the large number of children which it contained. These amounted at last to 30 or 40, from five or six years of age upwards. Every species of game and toy that could be devised was provided for them, and added to from time to time; and though education would be of little use to them, it was thought well to occupy their minds by giving them some schooling. A small school was accordingly built; schoolmasters were easily found among the lepers, and four hours' schooling a day, excepting during the holidays, was

insisted on. Examinations were periodically held with as much show and circumstance as possible, and public prize-givings, often attended by many of the principal ladies and gentlemen of the city, succeeded to them. It was easy to raise money for these prizes from the benevolent, and it was contrived, contrary to all sound educational principle, that all the children should receive some prize or other. The last prize-giving which was held before the writer left India was presided over by the Right Rev. Dr. Mylne, Bishop of Bombay, to whom the writer was greatly indebted, not only for his kindness in consenting to preside but also for the excellent speech which he made to the children in the Marathi language, a feat which not many of the chaplains under him could have emulated.

But for the institution of the school the condition of the children would have been far more miserable than it was. It gave them something to think about, and take a solid and abiding interest in, saved them from brooding, and gave zest to their play hours. The writer considers that if they were kept tolerably happy, it was chiefly the school to which they owed it.

During the years following the establishment of the Matoonga Asylum, the writer had some friendly controversy with the Army Sanitary Commissioners on the question of compulsory segregation; that is to say, they condemned it in some of their reports, and he replied to them in his annual Administration Report. It might seem that a layman can be but ill-equipped for such a combat with leaders in the medical profession, but, as before remarked, what the layman has to do is not to solve medical problems, but to seek a hypothesis upon which action may be based, and believing the problem before him to be one of an administrative character, the writer maintained and preferred his own opinion, even to that of the Army Sanitary Commissioners. The latter, in reviewing the sanitary condition of Bombay in 1889-90, dealt with the question of leprosy, and deprecated compulsory segregation on the grounds that the benefit to the community at large would be small, the expense would be great, and the interference with liberty would cause great injustice. On the last two points a practical adminstrator is probably a better judge than a medical man; at all events, they are clearly questions for the former. As regards the first, if it is correct, it is difficult, nay, impossible, to understand why voluntary isolation should be recommended by the Leprosy Commission, or any interference with lepers in their trades, even though those trades do concern human food, drink, or clothing. If isolation is *per se* of value, it cannot matter, medically, whether it is voluntary or compulsory. How the isolation is to be effected does not seem to be a question for the profession. Given isolation as an end to be achieved, the method by which it is to be achieved is a question for administrative, not medical, science. Suppose the Army Sanitary Commissioners to have been in favour of compulsory isolation, would they have thought it part of their function to prescribe to the writer the agency which he was to employ to enforce it, or to dictate whether such agency was to be that of the police or of the municipality?

The Army Sanitary Commissioners, in a later report, recurred to the subject, and said they had thought, and were still of the opinion, that compulsory segregation would cause much mischief. To this the writer replied in 1894, that the Matoonga Asylum had, at the date of writing, been in existence for more than three years; that it was idle to employ hypothesis when facts were at hand, and that if any mischief had been caused by compulsory segregation, it would be possible to prove it from the example of the Matoonga Asylum. He added that the Bengal Government were then considering a Bill for the compulsory segregation of lepers (the Bill which has since become law, and which had been sent to the writer for comment and opinion by the Bengal Government), and that a fact of that sort, which showed that the responsible administrators of another province believed that an example had been set in Bombay which it was wise to follow, was worth a thousand theories to the contrary.

It need hardly now be repeated that the example set at Matoonga has germinated and spread, and that not only have the Bengal Government passed an Act on the subject, but that the Government of India, in direct opposition to the advice of the Leprosy Commission, have passed "an Imperial Act, especially directed against lepers, as such," though the expression "directed against" does not seem a happy one.

A plain and simple narrative has been given of the establishment of the Matoonga Asylum, and it will be seen that it was incorrect to say that that asylum was built by the Government of Bombay out of funds contributed by Sir Dinshaw Petit. It was built by the writer out of funds collected by him. He claims no credit for having built a leper asylum. Any energetic person in the position he then occupied could have collected money and built an asylum; but such a structure, if it had been no more than a hospital or home, would have been as useless, considered as a means of delivering the city from an intolerable pest, as every other asylum in India at the time was. The real peculiarity which differentiated Matoonga from all other asylums, and which furnished first Bengal, and then the Supreme Government, with an administrative model, was that the principle of compulsion was in it first applied. There had been scores of leper asylums for years all over India, but none of them had brought this great question an inch nearer to solution. The Matoonga Asylum solved it, and has done so in the teeth of the medical profession. The writer hopes he may be pardoned for saying that he could not peruse Sir J. Woodburn's speech (which he first saw in January last) without some feeling of astonishment; and that he had a right to expect that his services to the country in this connection would not have been so completely forgotten.

Having regard to the fact that from the Matoonga Asylum has sprung the whole leprosy legislation of India, it will not be thought that too much time has been spent upon that asylum. The writer wishes now to say a few words on the general question of compulsory segregation, to which India has been committed. The serious fact in connection therewith is that the medical profession are almost unanimously opposed to it. It is almost impossible to deal with the question without some reference to the further

question of contagion, with regard to which, however, the writer feels his incapacity, and would decline disputation with a medieal man as altogether *impar congressus*.

The writer may be here allowed to digress for a moment to recount an incident which made at the time a considerable impression on his mind. When the Leprosy Commission first came to Bombay, at which time the Matoonga Asylum had been established, they courteously called at the Municipal Commissioner's Office, and the writer pointed out to them the Act under which leprosy had been notified as "an infectious disease, dangerous to life." On his calling their attention to the words, one of them asked, with a smile, "Who was the bold person who called leprosy an infectious disease?" The only reply that could then be made was that the word infectious must be read in a popular and general sense, as meaning communicable; but the suggestion that leprosy could not, with propriety, be termed infectious, caused a very uneasy feeling; for, if it could not, the whole fabric of the Matoonga Asylum and its regulations would fall to pieces. Under no other law than this Act was it humanly possible to enforce segregation—it could not be enforced unless leprosy could be notified under the Act—and if leprosy was not an infectious disease, the notification already issued was bad *ab initio*, and all that had been done under it lawless and actionable. The Matoonga Asylum was open to any respectable visitor, and who knew how soon some sharp solicitor might get hold of an aggrieved leper, and bring an action for false imprisonment, on the ground that leprosy was not an infectious disease, and, therefore, not notifiable under Act VI. of 1867? Luckily, nothing came of it, and doubts were dissipated by time.

But is leprosy not an infectious disease? Drs. Hansen and Looft, in their monograph on leprosy, published (at least the translation was) in 1895, seem to use the term "infectious" as synonymous with communicable. That is to say, the Norwegian word that they use has been translated "infectious." They say that there is no course open but to assume that leprosy is infectious, that leprosy may be "caught" by inoculation, that as bacilli cannot be destroyed the only thing to do is to prevent "infection," and that this can only be done by isolation.

In a contribution to the *Journal of the National Leprosy Fund* Dr. Beavan Rake (one of the Leprosy Commissioners) speaks of "infected foci." Other instances of the use of the word by medical men may be quoted, sufficient, probably, to justify the use of it in connection with leprosy in an Act of the Legislature.

To what extent the medical world is divided on the question of the contagiousness or otherwise of leprosy, the writer is hardly in a position to judge. It is noticeable that the Leprosy Commissioners, while they discard the theory of hereditary transmission, consider that, scientifically, leprosy must be regarded as contagious, though the extent to which it is propagated by contagion is exceedingly small, and that in the great majority of cases leprosy originates *de novo*, that is, "from a sequence or concurrence of causes and conditions which are related to each other in ways imperfectly known." In referring to this oracular pronouncement, Dr. G. A. Hansen remarks (*Lancet*, October, 1893) that it would have been more straightforward to say at once that it was not known how leprosy originated, than to pretend to know something about it; and to a plain mind the criticism seems not unfair. The Commissioners further remark that leprosy is not originated by insanitary surroundings, though it is "indirectly influenced" by them as increasing susceptibility to the disease. This again does not seem to take us much further. Of course, a weak man is more easily knocked down than a strong one.

There appears to have been much difference of opinion on the question how far the measures adopted to combat leprosy in the Middle Ages were the real agents in expelling, or almost expelling, the disease from Europe. It has been, no doubt, popularly supposed that the isolation of lepers, which was at one time generally, and often harshly and cruelly, enforced in England and in Europe, was the cause of this successful result; but many, perhaps most, medical authorities seem to think that improved hygienic conditions were in reality responsible for it. Dr. Vandyke Carter, however, concurred in the common and popular view. It is difficult to mention this name without paying a tribute to it. Of all Indian authorities upon leprosy he is surely by far the foremost. It is refreshing and ennobling to read his many contributions to the literature of the question, to study the masterly capacity with which he arrays his facts, the extraordinary acuteness with which he sifts them, the combined clearness and caution of his conclusions, above all, the earnest unwavering persistence with which he strives after truth; never the advocate for a moment of any special fad or theory, firm in his opinions, because they are based on facts and sound deduction, but always ready to yield to wider knowledge and sounder reason, continually striving after the light with a large and liberal recognition of the claims of practice as well as theory. Dr. Vandyke Carter did not believe that hygienic improvement accounted for the decline and practical extirpation of leprosy in the Middle Ages. He says,[6] in 1871:—"The ban under which lepers were put in Europe had the greatest influence in eradicating the disease—it may be said it was thus stamped out. To mention the old harsh leper laws, except with deprecation, might not be acceptable, yet, if the only alternative to their partial imitation be the hope thus expressed in the report of the late London Committee" (Committee of the Royal College of Physicians, June, 1862), "that a marked change in the habits of the population of India will ensue upon the increase of divers industries, the improved cultivation of land, the less frequent recurrence of famines, and the consequent general amelioration of their condition from year to year, and that better food, better clothing, better housing, with greater personal cleanliness, will lead to the abatement of leprosy; then, indeed, the outcome of a most elaborate official inquiry does seem disproportionate." He goes on—"To realise the

[6] *Transactions, Bombay Medical and Physical Society*, No. XI., p. 74.

needs and remedies of India on this subject, some practical acquaintance with the country, which the Committee of the Royal College of Physicians have not enjoyed, is an essential pre-requisite." He might well say so. To those of us who have been brought into close contact with leprosy in India—and what district officer or resident in a great town has not?—it seems the merest trifling to argue that we are to wait till the general improvement of the physical condition and sanitary surroundings of more than 300,000,000 of people has extirpated the disease.

(To be continued.)

YELLOW FEVER—ITS MODE OF DISSEMINATION.

By W. Loudon Strain, M.B., C.M.

Surgeon to the S. Paulo Railway, Visiting Surgeon to the Hospital Samaritano of São Paulo, Brazil.

When yellow fever has been endemic in a large city for a number of years, outbreaks occurring with painful regularity every hot season, it is almost an impossible task to trace the mode of infection in any one particular case. Cities like Rio de Janeiro, Santos and Havanna, have for so long been subject to recurring epidemics, that the popular mind considers them infected places to which it is dangerous to go during the months when the fever prevails, and if a case occurs in a person who has visited one of these places, all attempts to trace the source of infection are likely to prove futile. The water, the soil, the air (especially the night air), the buildings, the river or the sea, are all supposed to have become infected and contain the germ of yellow fever, and this supposition of a general infection of a city and all it contains makes the task of getting at the truth an almost impossible one.

In the years 1892 and 1893, I had the opportunity of watching closely the beginning and spread of an epidemic of yellow fever in the city of São Paulo, Brazil, where the disease then appeared in epidemic form for the first time. The facts observed by myself and others during this epidemic are of sufficient interest and importance to warrant their being made more widely known.

The city of São Paulo contains about a quarter of a million of inhabitants and its growth has been very rapid. Twenty-five years ago it contained only about 30,000 inhabitants. It is situated just within the tropics, the tropic of Capricorn being said to pass through one of the suburbs. It is about 2,500 feet above sea level, and only 50 miles from the seaport town of Santos, which for many years has had the unenviable reputation of being one of the unhealthiest ports in the world, chiefly owing to the terribly devastating epidemics of yellow fever. The climate of São Paulo is very moderate, both as regards heat and cold. Water of excellent quality, but often deficient in quantity, is brought into the city from the neighbouring hills. Drains were put down over twenty-five years ago, but what with scarcity of water for flushing, bad workmanship and an antiquated system, the drainage is not much to boast of. Enteric fever has been endemic for many years and every summer brings a fresh outbreak of this disease. For some years prior to 1892, "imported" cases of yellow fever were known to have occurred in the city, but in no instance did any of these prove the starting point of even a limited epidemic, and before March of 1892, no well authenticated case had been observed of the disease appearing in a person who had not recently been to Santos, Rio or Campinas. São Paulo and Santos are in direct communication by rail, the journey occupying about two and a-half hours. Santos is the only port for the enormous imports and exports of the whole State of São Paulo. For thirty years or so yellow fever has been a fearful scourge, and although for the last few years no severe epidemic has prevailed, yet the disease is by no means stamped out. Last year (1898) in the months of May, June and July, a considerable number of cases occurred, strange to say these being the cool months of the year. The improvement is generally believed to be chiefly due to the construction of a magnificent quay wall, doing away with what formerly was a foreshore of pestilential mud, composed in great part of the sewage of the city. The town itself, streets, drains and habitations, still leave much to be done in order to stamp out the remains of yellow-fever infection.

In order to show clearly the connection between Santos and the appearance of yellow fever in São Paulo, it will be necessary to describe briefly the state of affairs in Santos at the time of the appearance of the disease in São Paulo and for two or three years previously. The years 1889-90, and 1891-92, were black years in Rio and Santos. Yellow fever appeared with an intensity that seldom has been seen. Hundreds and thousands fell victims to the disease and the death rate was simply appalling in both places. From Rio the disease spread inland to small towns and villages, but always to places in close railway connection with Rio. In the State of São Paulo a fearful epidemic prevailed in Campinas and later in Rio Claro, both important railway centres where cargo from Santos had to be handled in large quantities in transhipment from a broad to a narrow-gauge railway. Each of these towns served as a new centre of infection, spreading the disease to other places still further inland, but always following the railway lines. During the last four or five years the disease has been spreading in the interior of this State to places hundreds of miles from the sea coast, and last year, when Sanarelli wished to make his first trials in serum treatment, it was to the town of S. Carlos in this State that he came.

The years 1889-92 also witnessed a remarkable "boom," and the imports became so great that the port of Santos was utterly unable to cope with them. Scores of vessels lay in port for months unable to discharge cargo, and many of them lost every member of their crew, and, waterlogged, sank in the river. Every available space was utilised for storage, and even the streets and pestilential river banks were piled up and littered with cargo. Such was the state of affairs prevailing in Santos in these truly awful years.

During the hot season of 1891-92, I treated a large

number of cases of yellow fever in São Paulo, in hotels and private houses, in every instance the victim being a fugitive from Santos, Rio or Campinas. In no instance did there occur a case in these houses or amongst the people in close contact with the sick, unless where, within the previous week, a visit had been paid to one or other of these infected centres.

In April, 1892, I saw the first case in my practice where the infection had been acquired in São Paulo.

Fraulein S., aged 35, German, governess, was first seen by me on April 3, 1893, on the sixth day of her illness. She had been four months in São Paulo, having come from Germany to act as governess in a well-to-do Brazilian family. Her trunk, with her clothing, was detained in Santos, owing to the block in the port, and when she received it the contents were almost completely spoiled from damp and apparently soakage of dirty water. She had the assistance of a mulatto boy in unpacking her box and doing what she could to save some of the contents. Within a few days of the receipt and unpacking of the box both fell victims to yellow fever. Fraulein S. died on the eighth day of disease, with all the characteristic symptoms of yellow fever. She was seen by several colleagues, amongst them by Dr. Domingos Freire, of Rio, of *micrococcus xanthogenicus* fame.

The boy was not seen by me, but from information furnished by the colleague who treated him there can be no doubt that he also fell a victim to yellow fever of a virulent type.

No other case occurred in connection with these two, and it is worthy, of note that the house is isolated, surrounded by its own grounds and in a good part of the town. Also that every care was taken in the disinfection of the rooms occupied by the patients and in the burning of the trunk and its contents.

At this time numerous outbreaks were occurring along the railway between Santos and São Paulo, and also from São Paulo inland for two or three hundred miles. Two of these outbreaks are interesting as showing clearly the source of infection. At a small village a few miles from São Paulo, an industrial company was erecting a distillery, and the machinery came *via* Santos, where it was delayed for a long time. On arrival at its destination a serious outbreak of yellow fever occurred amongst the *employés* and eight or nine of them died. Since then until now no further outbreak has taken place.

In the town of S. Carlos, which is a long way in the interior and a place of importance as a centre of coffee-farming, some railway contractors had a depôt for material, all of which passed through Santos. A man who lived in the depôt was the first victim of yellow fever in that town, the second being the man who succeeded him. Since then the town has been decimated on more than one occasion, the last being in 1898, when Sanarelli visited the town, as I have previously observed.

Returning now to the progress of events in São Paulo, the next appearance of the disease was in the beginning of 1893, in a district called Santa Ephigenia, at that time noted as being one of the dirtiest and least salubrious parts of the city. Overcrowding in filthy, cheap hotels and boarding-houses, was very prevalent and notably in the small area affected by the epidemic.

In a small house in the Rua Santa Ephigenia, a German, who lived there with his family, had stored in the lobby and in the very small courtyard at the back of the house a number of packing-cases containing pieces of machinery for wine making. These had been lying for months in Santos. They also remained for some weeks in his house without anything in the way of sickness occurring that could be attributed to their presence. On opening the cases the material used for packing, straw, &c., was found to be rotten and smelling most offensively. The empty cases, straw, &c., were stowed away in a corner of the courtyard for some time, until they were ordered to be burned. Within a few days of opening the first cases I was called to see the son-in-law, whom I found suffering from a moderate attack of yellow fever from which he recovered. In all, four cases occurred in this house, two being fatal, including the owner of the machinery himself, the other two being his grandchildren. The family left the house in terror, had the house thoroughly cleaned and a big bonfire of all that would burn and might harbour infection. As far as this house was concerned no further case occurred, but the evil had already spread and later on threatened to assume serious epidemic proportions. The next case was that of a German cabinet-maker, whose small workshop was built against the wall of the courtyard of the wine-maker's house. He died from yellow fever, but the exact date I have not been able to ascertain, but it was within a week or so of the outbreak in the Rua Santa Ephigenia. On the other side of the courtyard were the stables and coach-house of a gentleman whose house faced a street parallel to the Rua Santa Ephigenia and whose grounds extended to the wall of the courtyard. This gentleman's house was in charge of an old Portuguese couple who acted as caretakers during his absence. His coachman died first of yellow fever, and his widow, on leaving the place, made a gift to the old couple of the blankets, sheets, &c., that had been in use at the time of her husband's death. Both these old folks died of the disease, but at some considerable time after the coachman's death. I saw them both with my colleague, Dr. Mello Oliveira, to whom I am indebted for the particulars.

Meanwhile numbers of other cases occurred, but for some time the cases observed by me and those of which I had reliable information were limited to one block about 150 metres square, or to houses in the next blocks that were *vis-à-vis*, or almost so, to the infected houses, separated only by the breadth of narrow streets. Cases kept appearing in this very limited area from February till May, 1893. Since then, so far as I am aware, no further case has been observed there. I am unable to state with accuracy the number of cases that occurred in this area, but personally, I either saw or had reliable information of over twenty cases.

Numerous cases appeared in other parts of the city, but in almost every case there was a history of having cargo from Santos. One case I saw was a post-office official whose duty was to receive the mail-bags at the station, and amongst these, bags from Santos and Campinas, where epidemics were raging.

From these observations it will be seen that railways

running out from an infected centre play an important part in spreading yellow fever. That the infection is mainly carried in merchandise is also well known, but that infection should be thus carried, I believe it must have actually been contaminated with the emanations from a yellow-fever case, this being most likely by means of the sewage. In Santos the sewers even now discharge straight into the river through outlets in the quay wall, and before the quay wall was built the sewers discharged their contents on the muddy banks. The shipping has always been the greatest sufferer, and it is quite notable that yellow fever as an epidemic often prevails amongst the shipping for some weeks before it appears on shore. Sailors, rightly or wrongly, believed this to be owing to washing down decks with the contaminated river water, and it is now an established rule on many vessels never to wash decks in Rio or Santos harbours. Merchandise, then, that has been contaminated with sewage containing the yellow-fever infection is capable of spreading the disease provided it finds a suitable place. What constitutes a suitable place? The old theory of its being only a coast-line disease was only true until railways and rivers opened up the interior. A few still cling to the idea of its malarial origin and consider quinine administration to be recommended. In São Paulo city, malaria, at least in its common forms, is quite unknown, and so it is in many places in the interior where yellow fever has been epidemic. As to altitude, he is a bold man who limits the appearance of the disease to so many hundreds of feet above sea level. São Paulo is 2,500 feet above sea level and only fifty miles from the sea; the disease has been higher than this even, and, given an infected and insanitary port with such conditions as I described in Santos in 1890-92, and rapid railway communication, and the disease may spread as high and as far as it finds a suitable developing ground. The essential thing to constitute a suitable place is *filth*. In the interior towns of this State where yellow fever has prevailed and become endemic almost, the sanitary conditions were, and in some cases still are, of the worst possible description. Giving Campinas water and a system of drains only took place after several devastating epidemics. Prior to this water was got from shallow wells or running streams; house refuse of all kinds, including human and animal excreta, were accumulated round the dwellings until, as one colleague told me, to cleanse the town it would be necessary to remove at least twelve inches deep of the superficial soil. Where yellow fever finds such a lodging place it comes to stay, and year after year it appears with painful persistence, and there is, to my mind, no doubt that the specific micro-organism finds its *nidus* in the soil loaded with organic impurities; that during the cool months it remains quiescent in the superficial soil, to recrudesce with the return of heat and moisture. Now as to the method of individual infection, the mode of introduction of the germ into the system, I agree with those who consider that infection takes place by the digestive system and not the respiratory, that the infection is eaten and not inhaled. In the case of the first outbreak in the Santa Ephigenia district, the cases containing the machinery and the infective material were stowed for some weeks in the house and yard and no case appeared; but as soon as unpacking began then came the first case of fever. The likelihood is that food was eaten with contaminated fingers, or which is also very probable, that the house fly was the bearer of the infection, implanting such minute particles of living infective material as their little legs can carry. Only those living in tropical regions can know what a plague flies can be, and how they swarm in myriads, apparently in the most indiscriminate manner over the foulest filth or over your own bread, butter, sugar, cold meats, salads, fruit, or even take a bath in your milk.

For years I have had a strong suspicion that the common fly plays an important *rôle* in the dissemination of yellow fever, and it is a popular saying in this country that when there are many flies there is much fever. I remember on one occasion being asked to see a British sailor, recently arrived in São Paulo from Santos, a fugitive, during one of the bad epidemic years. The poor fellow was already three days ill with yellow fever and lying in one of the filthiest dens I have ever been in. His bed was surrounded with the beds of other members of the crew, as thickly as they could pack them in. It was a sweltering day, and the poor fellow was practically abandoned. He had had copious black vomit, which all went on the floor, and as I entered the room a cloud of flies rose from their loathsome feast and departed through the open window to enter perhaps a neighbour's house and have another feast on some bread, butter or sugar.

An old colleague and friend, Dr. Lane, of this city, informs me that the great Campinas epidemic of 1889 had its origin in a bakery. The first case was that of a woman who came from Santos. She sickened and died in the baker's house, which was also his shop, and all the early cases that occurred are said to have been customers of this bakery. In the Santos and Campinas epidemics I have never known of a case of yellow fever acquired in one who took the precaution to eat nothing in the infected places; but I have seen cases where the person never slept a night in Santos, travelling up and down from São Paulo every day, but in every case one or more meals had to be taken in a restaurant. I have observed in Santos epidemics that, amongst my countrymen, at any rate, young bachelors form the largest number of victims in epidemic years. There may be several reasons why this should be, but one important one to my mind is that the married men have their meals more often in their own homes, where, presumably, more attention is paid to cleanliness and keeping food stuffs covered up than in the restaurants where the bachelors have their meals. I know of one very popular restaurant in Rio where in a corner of the kitchen, not four yards from the table where the cooks are preparing the food, a bucket is placed, partially screened off, which is used as a closet by the *employés* of the restaurant.

Sanarelli[1] has informed us that "the virus of yellow fever does not reside in the digestive tube," but as a result of his investigations he states that it is found only in the circulation and in the tissues, and he has, contrary to all expectation, never found

[1] "Etiology and Pathogeny of Yellow Fever."

the bacillus in the contents of the gastro-intestinal tube. Just as in many of his experiments on, and examinations of, the blood of yellow fever patients, Sanarelli failed to discover the *bacillus icteroides*, not because it did not exist, but because for some reason or other he could not succeed in finding it, so in the contents of the intestinal canal, the evidence as to the spread of the disease by sewage, that is fæcal, contamination, is too strong to be completely annulled by Sanarelli's simple negative. He has also given us no suggestion as to the method of spread of the disease, but also agrees that it is by the *via gastrica* that the infection is introduced.

Some of our local *savants* incriminated the water supply, others found some very mythical relation between the spread of the disease and the subsoil water, and still another thought it advisable to advise sterilising the water used in making bread and beer. The Government and its sanitary authorities in São Paulo, however, were more practical, and under the guidance of the late Dr. Cesario Motta, took steps to close all wells whose waters might be contaminated, constructed an excellent isolation hospital and insisted to a certain extent on cleanliness of habitations and disinfection of rooms where the sick had been, and since that small epidemic of 1892-93 no further epidemic manifestation has occurred in any district of this city.

XEROSIS AND NIGHT-BLINDNESS.

By M. T. Yarr, F.R.C.S.I.

Major, R.A.M.C.; Fellow Medical Society of London; Member Ophthalmological Society, &c.

Xerosis of the Conjunctiva, with its almost invariable concomitant, Night-Blindness, is a common disease in tropical and sub-tropical countries, and by no means unknown in England. By xerosis is meant a disease of the interpalpebral portion of the conjunctiva, mainly affecting a small area on the temporal and nasal sides of the cornea, by which the conjunctiva becomes dull, greasy, covered with a dirty grey foam, and incapable of being wetted by the tears. Microscopical examination of the xerotic patches shows that the epithelium is greatly thickened, and that each epithelial cell is filled with minute drops of oil: to this fact, and also to the adhesion to the surface of the epithelium of the Meibomian secretion, is due the peculiar greasy, frothy appearance so characteristic of the disease. Rod-shaped bacilli, with rounded ends, the so-called bacilli of xerosis, are found in profusion on the surface of the epithelial cells. Mr. Sydney Stephenson, in this country, has never yet failed to find them in cases of epithelial xerosis, not only in cover-glass preparations, but also in cultures made from the frothy patches.[1] As these bacilli, however, are also found in the conjunctiva under certain other conditions, we are hardly as yet justified in doing more than noting their apparently invariable presence in xerosis.

A severe form of xerosis, in which the disease spreads to the cornea (Keratomalacia), which frequently suppurates, is unfortunately not uncommon in the tropics, especially in the native quarters of great towns such as Bombay, Calcutta, and Hong Kong, and seems to be the local expression of some obscure general disease; patients, mainly children, emaciate rapidly, suffer from diarrhœa, and often die. Of this class of cases, Captain Herbert, of Bombay,[2] exhibited some interesting photographs and microscopic preparations at the May meeting of the Ophthalmological Society last year. He attributes xerosis to faulty feeding: it is so common in Bombay amongst the poorer classes that in four months, June to September, 1896, he was able to tabulate one hundred consecutive cases.

A condition somewhat resembling xerosis is often seen associated with ectropion and chronic trachoma, and has been called "secondary" xerosis by Leber,[3] but the resemblance to xerosis is only superficial.

Of the visual phenomena associated with xerosis, the most constant and remarkable is *night-blindness*. The expression "night-blindness," though convenient, is not a very happy one, inasmuch as the defect of vision develops when illumination is diminished from any cause, in an artificially-darkened room, for example. We are all familiar with the difficulty experienced in seeing objects at first on entering a darkened room. In night-blindness this defect of vision, transient in the normal eye, is persistent, owing to the retina having lost its power of "adaptation," and no longer acting except to strong stimuli—a condition known as "torpor retinæ." Förster's photometer furnishes a means of estimating the light-sense, and hence the degree of night-blindness, but the apparatus is clumsy, and seldom used in this country. The amount can be estimated sufficiently well for all practical purposes by testing with faint test-types in a darkened room, and comparing the types read by the patient and those read by the surgeon.

It is now generally recognised that night-blindness is an almost constant symptom in xerosis, both probably being expressions of malnutrition of the eye. Mr. Sidney Stephenson, in the able and exhaustive paper previously alluded to, quotes many striking examples of this association. Bitot examined twenty-nine cases of night-blindness, and in every case found spots of xerosis—the so-called hemeralopic spots.[4] Villemin confirmed this by investigations amongst soldiers at Strasburg.[5] Kuschbert and Neisser described an outbreak of night-blindness in a Breslau asylum in which xerosis was present in every case,[6] and many other instances could be given. It must be remembered that the symptom must be looked for, as patients often fail to draw attention to it. I saw many cases of xerosis, both mild and severe, when in charge of the Government Civil Hospital in Hong Kong, but never detected night-blindness, as at the time I was not aware of its being an almost invariable symptom. Cases of night-blindness without xerosis and without any other discoverable cause (such as retinitis pigmentosa) undoubtedly exist, but are extremely rare; I have only seen one such case, at present under the care of Mr. Treacher Collins at Moorfields.

It has been suggested[7] that night-blindness is due to molecular changes in the retinal pigmented layer, brought about by impoverishment of the blood in the choroid, and that the xerotic patches are due to

degeneration of the superficial layers of the conjunctiva forming a *nidus* for the xerosis bacillus. "In this view both night-blindness and xerosis would be attributable to a common cause, viz., impaired nutrition, while the former would be proximately due to dazzling, and the latter to the bacillus." (Stephenson.)

Mr. Stephenson believes that reduction and transposition of the red and green fields of vision is another constant phenomenon in xerosis, having found it in every case which he investigated fully.

Dr. Schtschepotzew, of Kiew, mentions dilatation and paresis of the pupils as a frequent symptom in the epidemics which prevail in Russia.[8] No fundus changes can be detected.

The ætiology of xerosis is still obscure. Its frequent association with malaria, and widespread prevalence in malarial countries, have led many to believe in its malarial origin. Stoeber, in 1841,[9] first noted night-blindness as a frequent symptom in malarial fever. Quaglino[10] drew attention to the same fact thirty years later. Segard[11] found numerous cases of xerosis amongst malarials at Tamatave; Sulzer also[12] refers to the frequent association of the two diseases; and Laveran, in his classical work on malaria, endorses his experience. On the other hand, xerosis is found in countries where malaria is unknown, for instance, in schools and asylums in England.

On the whole it appears probable that xerosis is an expression of a state of general malnutrition which may be due to several causes, chief among which are malaria and insufficient or improper food. It is most frequently found amongst the insufficiently-nourished classes, and is stated to occur in an epidemic form in Russia during and after the long Easter fast.[13] Dazzling by bright light often seems to act as an exciting cause; hence its frequency amongst sailors, by whom it is often attributed to the malign influence of the moon.

Cases of xerosis always improve *pari passu* with improvement in the general health: with good food, fresh air, and general tonic treatment most cases do well. Keratomalacia must be treated on the same principles as other forms of corneal ulceration. Nesnamoff (see my note in the March number of the JOURNAL) treats the xerotic patches by washing the conjunctiva and cornea first with normal saline solution, and then with ether. This removes the fat from the diseased spots, and they become capable of being wetted by the tears. He also employs inhalations of ammonia to increase the lachrymal secretion, but the utility of this procedure is questionable. The real cause of xerosis is not deficiency of the lachrymal secretion, and it has never been known to occur after extirpation of the lachrymal gland.

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TYPHOID FEVER IN INDIA.

By OSWALD BAKER.
Surgeon-Lieut.-Colonel, I.M.S.

As the *role* of the bacillus of typhoid fever in India is of considerable importance, the following case, to which some remarks are appended, may possibly be of interest.

On March 8 I was consulted by a military man, who had only a few days previously arrived in England from Burma, whence he had been invalided in consequence of a somewhat prolonged attack of what was said to be malarial fever. Before describing his symptoms he handed me a copy of his medical case and attached certificate, such as is always furnished to military officers who are invalided from India to Europe. This medical history of the patient's illness was a model of brevity and precision; its leading points were these:—

"*Disease.*—Malarial fever. *History, &c.*—Patient was admitted into hospital on December 12, 1898, having had fever in his quarters for some days previously. After admission his temperature was normal in the morning, but rose to 102° or 103° in the evening. It remained like this for about three weeks. On and after January 1, it was normal throughout."

I further learned from the patient himself that during his stay in hospital he had an attack of phlebitis in the right leg, and also that he suffered slightly from diarrhœa. The affected limb was still an inch greater in circumference, both at the thigh and calf than that of the opposite leg at corresponding situations. There was no enlargement of the spleen or other obvious manifestation of disease beyond a trifling amount of anæmia, nor were there any malarial crescents to be found in the blood.

On reviewing these data it was difficult to resist the impression that the diagnosis of the illness the patient had undergone was at fault. Although diarrhœa is of frequent occurrence in malarial fever, phlebitis of malarial origin is, on the other hand, practically unknown. I have certainly never met with an instance of plugging of the femoral veins in malaria in my own practice, nor have I ever read of its occurrence in the practice of others. Moreover, the character of the fever in the case under consideration, taken by itself, was not suggestive of malaria, for, although in quotidian ague there are daily remissions and exacerbations of temperature, it is seldom these changes manifest the precision and regularity noticed in this case. I felt convinced therefore that the attack had been enteric and not malarial fever and, desiring to obtain confirmation of this belief, I sent some of the patient's blood in a sedimentation tube to the Clinical Research Association, for submission to Widal's test. On the following day I received the Association's report, which was as follows:—"This blood has a strong clumping activity when it only forms 5 per cent. of a mixture with typhoid bacilli; it therefore gives the typhoid serum reaction."

There was, then, no longer any doubt in the matter, the illness had obviously been an attack of typhoid fever.

The case, therefore, furnishes fairly conclusive

evidence of an error in diagnosis, a mistake which is, I believe, of frequent occurrence in India at the present day. There can be no doubt that enteric in India, although productive of great mortality, owing to its high degree of prevalence, runs, in many instances, a milder course than its congener in this country, and that typhoid spots and bowel complications are much more rarely seen. There is not always in India, and probably the same may be said with regard to other tropical countries, the same amount of constitutional disturbance present and, occasionally, sufferers from the disease are not conscious of being at all seriously ill. They sometimes continue to go about their daily work and take ordinary food, aware, perhaps, that they are having a little fever in the evening, which they regard as malarial or climatic, but to which, beyond paying it the compliment of taking a little quinine, they give but slight attention. I do not mean to say that extreme cases of this kind are of frequent occurrence, but I am satisfied that they do occur, just as I am convinced that the usual concurrent signs of typhoid met with in this country, such as rash, diarrhœa, abdominal tenderness, and grave constitutional disturbance, are by no means constant features of the Indian cases. In one of the worst cases it was my lot to attend whilst in Burma, which, with its relapses, covered a period of four months, there was not, from beginning to end, a vestige of an exanthem or a trace of diarrhœa.

That a case of typhoid presenting no definite typhoidal symptoms should, when viewed from a clinical standpoint only, escape recognition, and should, in a country where malaria prevails, be set down to malaria, is not at all surprising. Indeed it would, paradoxical as it may appear, be wrong rather than right, having regard to prevailing views, to make a correct diagnosis under such conditions, for there exists in the continued and remittent fevers of India ample scope for a diagnosis which would more closely fit in with preconceived medical opinion.

When, however, it has become the established practice to appeal to Widal's test for diagnosing doubtful cases, the confusion which now prevails between typhoid and the other endemic fevers found in India will be at an end. At the present time it is more than probable that mild attacks of enteric are frequently mistaken for malaria or for other varieties of tropical fever.

Without venturing so far as to say that all the cases for which Crombie, in his recently-delivered able address on the Unclassified Fevers of Hot Climates, suggests the name of Urban Continued Fever are probably mild attacks of typhoid, I am decidedly of opinion that the majority of them are of this nature. If I were still practising in India I should certainly consider every case of continued fever of about three weeks' duration presenting regular morning remissions of temperature, although not associated with any other definite typhoidal symptoms, to be an example of mild typhoid. Crombie admits that the fever to which he has applied this designation might be called "Bastard Typhoid;" and Manson, in his valuable manual on "Tropical Diseases," says that, "in cases in which there is the slightest doubt, it is an excellent rule to regard all doubtful fevers as being possibly typhoid."

THE ETIOLOGY AND TREATMENT OF BLACKWATER FEVER.

By L. W. Sambon, M.D. (Naples).
London.

Of late years much attention has been directed to the study of blackwater fever, on account of its extreme prevalence and deadliness in the newly opened up regions of intertropical Africa.

In 1893, at a meeting of the Epidemiological Society, Dr. Manson read a masterly paper on "African Hæmoglobinuric Fever," in which he stated that he believed it to be a disease "*sui generis*." Three years later, Bastianelli, in the *Annali di Medicina Navale*, endorsed Tomaselli's theory, which ascribes the hæmoglobinuria to quinine poisoning; but, at the same time, he admitted that there was a hæmoglobinuria which could not be attributed to quinine, and which he believed to be a consequence of intense æstivo-autumnal infection.

At the last meeting of the British Medical Association, I again endeavoured to prove that blackwater fever is a specific disease, and I pointed to the fact that it is clinically, and probably also etiologically, akin to the *redwater fever*, or *Texas fever* of cattle. I likewise observed that *paroxysmal hæmoglobinuria*, and *epidemic*, or *infantile hæmoglobinuria*, possibly belong to the same group. At the same time appeared Koch's statements supporting the quinine theory of blackwater fever. Then followed a lively discussion and many valuable papers were published by Powell, Poole, Reynalds, Gray, &c., which have added much new and important information to our knowledge of the disease.

Finally, Dr. Crosse read a paper at a recent meeting of the Epidemiological Society, upholding the old theory, which ascribes hæmoglobinuria to an intense infection of ordinary "malaria."

The Colonial Office, fully aware of the fact that blackwater fever is seriously opposing African development, appointed in conjunction with the Royal Society a commission consisting of Drs. Daniels, Christophers and Stephens, to enquire into this deadly scourge. I trust that this Commission, which is now workng in British Central Africa, may soon produce overwhelming evidence of the specific nature of the disease; but, meanwhile, I believe we have sufficient information to recognise that blackwater fever is due neither to ordinary malaria, nor to quinine poisoning, but is a distinct disease, just as much as beri-beri or dysentery, which were at one time believed to be manifestations of malaria, and of which we have as yet no definite etiology.

It is not possible to form any idea on blackwater fever unless we have a clear conception of the various diseases comprehended under the generic and inappropriate term "*malaria*." Many of those who have lately discussed the etiology and treatment of blackwater fever proved themselves to be absolutely ignorant of the recent progress in the knowledge of "malarial diseases" and some, furthermore, confounded hæmaturia with hæmoglobinuria.

At one time typhus, typhoid fever, relapsing fever, dysentery, liver abscess, elephantiasis, beri-beri, undulatory fever and a host of other diseases, were con-

founded with malaria, but now we know that they are distinct diseases. Localisations of summer-autumn parasites in the lungs or gastro-intestinal mucosa, may simulate pneumonia and cholera or typhoid fever, but usually, the cases described by authors under the names of typho-malaria, malarial pneumonia, malarial dysentery and malarial scorbutus are cases of mixed infection.

Soon after the introduction of Peruvian bark, Morton and Torti distinguished the "malarious fevers" from a host of febrile diseases by noticing that they yielded to quinine; but it was not until after Laveran's discovery of the malarial parasites, and the subsequent admirable researches of the Italian school, that we were able to frame definite notions concerning these diseases.

Recent text books mention three varieties of "malaria": tertian, quartan and summer-autumn fever. Tertian and quartan fever are two distinct diseases which can now be successfully and unmistakably isolated, but summer-autumn fever is still involved in much obscurity, and the obscurity is probably due to the fact that we are confounding several varieties of hæmatozoa under the most inappropriate name of *summer-autumn fever*.

Recent studies in malaria have taught us that each disease has its peculiar parasite—that each parasite has its peculiar life history and gives rise to peculiar symptoms. The symptoms caused by any particular parasite do certainly vary in intensity, but never in kind. The characteristic periodicity of successive paroxysms will always be the same in each disease according to the life cycle of its peculiar parasite, and the accompanying phenomena will follow suit.

While this is absolutely true, the practitioner will not find it always as evident as stated, because he will most probably meet one of the following complications:—

(1) A double or multiple infection of the same variety of parasites—the two or more broods attaining maturity at different times.

(2) A mixed infection of two different varieties of parasites (tertian and summer-autumn fever).

(3) A malarial infection and one or more concomitant infections of a totally different nature (tertian and typhoid, or summer-autumn fever, together with anchylostomiasis and tuberculosis, &c.).

Theories of Blackwater Fever.

The theories concerning the etiology of blackwater fever are the following:—

(1) Intense summer-autumn infection (Bastianelli, Bignami, Crosse, &c.).

(2) A form of quinine poisoning in malarial cachectics (Tomaselli, Bastianelli, Greek physicians, Koch).

(3) A form of poisoning induced by fresh beans.

(4) A specific disease (Manson, Sambon).

Is it an Intense Attack of Summer-Autumn Fever?

The Geographical Distribution of Blackwater Fever does not coincide with that of Summer-autumn Fever.—Our knowledge of the geographical distribution of diseases is most indefinite, because hitherto little attention has been bestowed on this very important branch of medical knowledge. Malaria has been mapped in the most careless way and, necessarily, no distinction has as yet been made of its different varieties, which are distinct diseases, and have each their peculiar distribution. Nevertheless, there are such striking differences in the distribution and prevalence of blackwater fever and summer-autumn fever that they need no minute observation. Summer-autumn fever is very widely distributed and exceedingly prevalent in all tropical and subtropical regions of the earth. Blackwater fever is only met with in some of these regions, and, where both are co-endemic, its numerical, seasonal and local prevalence in no way coincide with that of summer-autumn fever. For example, it is extremely prevalent in intertropical Africa, and so much so in some districts that those who are sent thither fully expect to suffer from the disease. On the other hand, it is almost unknown in Asia and Australia. Several authors have carelessly mentioned Italy as an endemic area of the disease, but though blackwater fever is common enough in Sicily and Sardinia, it is rare in the malarious districts of the peninsula. The same difference in prevalence and distribution has been observed in the Southern States of the American Union, in the West Indies and in other places.

Crosse believes that the difference in distribution and prevalence is only apparent, and that it will be annulled by further enquiry. This is not so. If blackwater fever had prevailed in India and China authors could not have failed to describe it as they did whenever they met it in its endemic areas. Blackwater fever exhibits such striking symptoms that it is absurd to think that it could have escaped the attention of such men as Chevers, Carter, Martin, Fayrer, Morehead and Maclean. But we must not forget that the distribution of diseases is subject to change, just as much as the distribution of other plants and animals that do not become parasites of man. Ross's important discoveries on the transmission of malaria by mosquitoes open up quite a new field of thought, and they suggest, in the instance of blackwater fever, that the disease may have been imported to India from its endemic regions of Africa. In India, as far as I am aware, blackwater fever has been recently located only in Assam and the Darjeeling Terai. Powell states that he has seen the disease in those who returned from the Gold Coast, and he mentions that his Case X. had contracted fever on the Gold Coast. Missionaries and officials returning from West Africa frequently exhibit relapses of blackwater fever in Europe, but there is very little danger of the disease becoming prevalent in England. The case is very different in India, where are to be found all the conditions necessary to the evolution, maintenance and spread of the disease. That blackwater fever can spread has been repeatedly observed, epidemic outbreaks of the disease have been recorded and several authors attest to the marked increase of the disease in some districts.

Several varieties of Hæmatozoa have been found in patients suffering from Blackwater Fever.—In a considerable proportion of cases no parasites were found

ELEPHANTIASIS ARABUM.

Elephantiasis Arabum is a local manifestation of lymphatic obstruction due, in all probability, to the immature ova of filaria nocturna. The disease is characterised by paroxysms of fever, hypertrophy of the skin and subcutaneous tissue, varicosity of the lymphatics, and with these signs and symptoms chyluria is frequently associated. The parts of the body attacked are usually the lower extremities, the scrotum and penis, or the labia of the vulva; but the upper extremities, the mammæ and even the face may become elephantoid.

Geographical Distribution.—Elephantiasis is well known in India, the Malay Peninsula, Siam, Indo-Chiné, Formosa, China, the islands of the Pacific Archipelago, Asia Minor, Egypt and Africa generally, West Indies, and South America. The exact limits of the disease are not definitely known. In Europe and North America it is scarcely known; the endemic cases, which have been reported in these countries, have been cavilled at, and it is doubtful if the disease is really known north of the fortieth parallel of latitude. That obstruction of the lymphatics may occur in any latitude, that hypertrophy of the skin and subcutaneous tissues with lymphorrhœa may occur is well known, but that elephantoid fever caused by a nematode parasite with resulting elephantiasis—in other words, true elephantiasis—has ever been met with in Europe in recent times except perhaps in Spain is doubtful.

Cause.—In the year 1870 Lewis, of Calcutta, found a peculiar microscopic worm in the chylous urine of a patient suffering from elephantoid fever, and in 1872 the same observer found a similar parasite in the human blood. Ever since, the association of this parasitic worm with elephantiasis has fascinated scientific investigators, and the result is an established theory in regard to their connection. The parasite was named the filaria sanguinis hominis, as when first found it was believed to be the only filaria infesting the human blood; but recently these nomads have been still further differentiated, in consequence of the investigations of Manson, who has added many species of blood filaria to our nomenclature, and has proved it is the variety known as filaria sanguinis hominis nocturna which is the cause of elephantiasis.

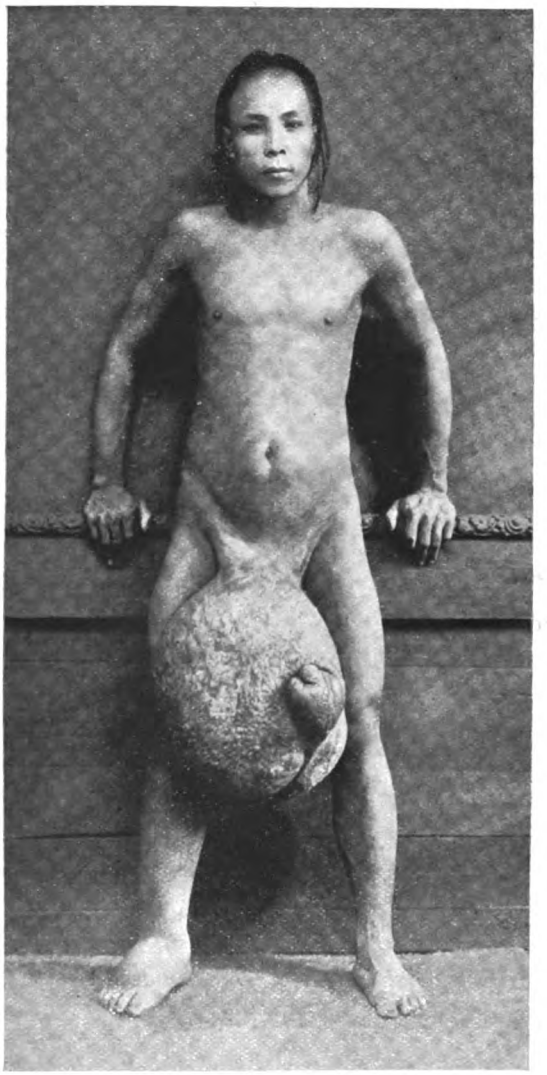
The worm discovered by Lewis is only the miniature young of parent parasites named after the discoverer, Bancroft, filaria Bancrofti. The parent forms have their *habitat* in the lymphatics, and as the ova escape they get into the lymph channels and are carried onwards through the glands and thoracic duct to the blood. Manson contends, and indeed has proved, that it is not the swimming embryos of the parent filaria which serve as the exciting cause of elephantiasis, but the miniature ova due to the parent aborting before the young have attained natatory powers, and that it is due to a crowd of these miniature ova being carried into the lymph channels acting as emboli, which lead to stasis of the lymphatic circulation.

Anatomical Characters.—The skin becomes hypertrophied, rugose and furrowed. The epithelial layer of the skin is greatly thickened, and frequently a clear, lymph-like fluid flows from the surface or remains to form a crust or scab over the integument. In neglected cases cracks and fissures occur yielding a sanio-lymphous discharge. The deeper layers of the skin and the subcutaneous tissues are felt to be hard and indurated; there is no pitting on pressure when the disease is of some standing.

Fever.—The fever known as "elephantoid fever" recurs at irregular intervals. The attacks last for varying times, but they continue generally from one to four days. An attack is ushered in by headache, rigors, nausea and vomiting, accompanied by acute pain in the affected part. During the paroxysm the nearest group of lymphatic glands are felt to be swollen and indurated, ill defined and painful, and the lymphatics communicating with the glands became matted, cordy and tender.

Concomitants and Sequelæ.—The result of lymphatic obstruction, venous congestion, lymphangitis, and deposit of fibrous tissue, &c., is that muscular action is interfered with, the nutrition of the muscle deteriorates, and in consequence the movements of the limbs are at first hampered and then permanently damaged. The veins and even the arteries are either occluded, or their capacity for dilation and contraction diminished, by the dense mass of boardy tissue in which they run; nerves are frequently enlarged and broadened; ulcers appear and refuse to heal. Chyluria is at times a marked accompaniment, giving rise to urinary trouble, owing to the mechanical difficulties the ureter, bladder and urethra have in giving passage to the pultaceous-like mass of coagulated lymph in the urine.

Treatment.—Accepting the theory of the ova of a species of filaria being the cause of elephantiasis, and seeing that parents of these ova gain entrance to the human body by water, it is evident that all water used for culinary purposes, and for drinking, should be boiled. Manson suggests also that all stored water should be protected by gauze netting, to prevent contamination. All other means of treatment, short of removal of the diseased parts, are unsatisfactory. Drugs have been tried, but none have proved curative.



ELEPHANTIASIS ARABUM.

Photograph of a Chinaman aged 22, from the province of Kwang-Si, showing elephantiasis of scrotum, penis, and right leg. The scrotal tumour after removal weighed 52 lbs. Both testicles were preserved, and within 15 months after the operation the patient's wife was delivered of a healthy child.

Bale & Danielsson, Ltd., London.

in the peripheral circulation, though repeatedly searched for by competent observers. But, frequently, hæmatozoa have been found, though never in such numbers as would account for that intense blood destruction which is so characteristic of the disease. Sometimes tertian parasites have been described, but more frequently the small disc-like bodies and the crescents of the æstivo-autumnal group. Once or twice both varieties have been found together. That we should find the tertian, quartan and æstivo-autumnal parasites in blackwater fever is what might be expected, for the ordinary malarial diseases and blackwater fever are in many places co-endemic and, all being debilitating diseases, one predisposes to the active manifestations of a latent infection of the other. But the presence of tertian and summer-autumn parasites is not sufficient in itself to prove that these parasites are the cause of the hæmoglobinuric paroxysm no more than anchylostoma duodenale, tænia saginata, or filaria nocturna would account for it, were they to be found in a blackwater fever patient. The very fact that both tertian and summer-autumn parasites have been found strongly invalidates the theory of regarding either as the causative agent of hæmoglobinuria.

But, possibly, the hæmatozoa described as parasites of the ordinary æstivo-autumnal fevers were really of a distinct variety. Such was the opinion of F. Plehn, who examined many cases of blackwater fever in the Cameroons, and in this opinion several other observers concurred.

Unfortunately the blood has been usually examined by men who had had no special training in the parasitology of tropical diseases and, again, most observers examined it in a late period of the disease, when the paroxysm was almost at an end.

The occurrence of Blackwater Fever in those who have previously suffered from ordinary Malaria does not prove the identity of the two diseases.—That most cases of blackwater fever take place in those who have been previously debilitated by frequent attacks of malaria, is a well-known fact, but it is no proof of identity. We do not believe tuberculosis to be a manifestation of typhoid fever, simply because it often follows in its wake. Moreover we know now of several persons who contracted blackwater fever only a few months after landing in Africa, though they were by no means the subjects of malarial cachexia. Then, again, experience has taught us that if we send to West or East Africa a man who has suffered from malaria in India, China, or Australia, he will contract blackwater fever almost to a certainty, but that he will run no such risk however long he remain in the malarious districts of Asia and Australia.

The Symptoms of Blackwater Fever differ greatly from those of ordinary Malaria.—The difference in symptoms between tertian, quartan, and æstivo-autumnal fever is small compared to that which exists between any of these diseases and blackwater fever.

In blackwater fever we have usually severe and prolonged rigors; they generally occur after the fever has set in, and will probably be repeated several times in the same day, as in cases of septic fever. In malarial diseases there is only an initial rigor. Then again it is important to remark that whereas the chill is severe in tertian and quartan fever, it is far less marked in summer-autumn fever, which is the infection supposed to produce hæmoglobinuria.

Vomiting is a frequent symptom in malarial diseases as in many other infections, but it seldom assumes that persistent and grave character which is so characteristic of blackwater fever. The peculiar olive-green vomit of blackwater fever may, in grave cases, assume a dark brown colour from the admixture of hæmoglobin, and resemble the *vomito negro* of yellow fever with which it has actually been confounded.

In ordinary malarial fevers there is often a slight, sallow colour of the skin, but never that sudden, intense saffron or greenish-yellow discoloration which is so characteristic of blackwater fever, and which is constantly found in all cases, not excluding the mildest relapses.

Another peculiar, constant and most characteristic symptom of blackwater fever is the colour of the urine, which becomes usually as black as stout from the presence of hæmoglobin. This never happens in tertian, quartan, or summer-autumn fever. The urine in these diseases may be highly coloured from an increased quantity of urobilin, as in any other fever; it may even contain blood in the rare cases in which there be a complicating nephritis, but it will never resemble the urine of blackwater fever, which is absolutely pathognomonic.

The temperature in blackwater fever differs also greatly from that of the other malarial diseases. The temperature in uncomplicated blackwater fever may attain 105° or even 106° F., but it is usually low (102° or 103° F.), and several relapses have been recorded in which there was hardly any pyrexia at all. Then again the temperature of blackwater fever never shows that constant, unfailing, periodical return which is the characteristic feature of the ordinary malarial diseases.

Other peculiar features of blackwater fever are the intense blood destruction accompanied by marked leucocytosis; the pain over the loins; the enlargement and tenderness of the liver—and, in bad cases, the suppression of urine.

Thus we see that in all its symptoms blackwater fever differs widely from the malarial diseases as yet differentiated. It might be objected that a certain difference would naturally be expected in a more intense attack of summer-autumn fever, but such a view is not in accordance with fact, because in the most pernicious cases of summer-autumn fever we do not find the symptoms peculiar to blackwater fever and, on the other hand, all cases of blackwater fever, however grave, however mild, always exhibit the very same symptoms, with no difference except that of intensity and duration.

Blackwater Fever begets Blackwater Fever.—Blackwater fever is extremely liable to relapse. Relapses may occur weeks or months after the first attack; they may be very frequent, and they may occur at any season and in any place, far away from the endemic districts of the disease. Each relapse will be usually milder than the previous one, but they will all exhibit the very same symptoms of the original attack, thus proving, in the most evident

manner, that to believe hæmoglobinuria to be the consequence of a more intense attack of æstivo-autumnal infection is an untenable paradox.

(To be continued.)

A PRACTICAL NOTE ON BILIOUS VOMITING.

By St. Geo. Gray, M.B., B.Ch. (Univ. Dubl.)
Colonial Assistant Surgeon, St. Lucia, W.I.

One of the most distressing symptoms met with in many cases of malarial and other fevers in tropical countries, and also one of the most troublesome to treat, is the persistent and almost incontrollable bilious vomiting. Everything that is swallowed comes up again, and with it a varying quantity of mucus and bile—sometimes white, sometimes yellow, and sometimes green. The patient is worn out with the constant vomiting—he complains of pain at the pit of his stomach from the incessant straining, which is often severe enough to cause hæmorrhage, so that the vomited matter is mixed with blood.

If this vomited matter be tested with litmus it will be found to be alkaline or neutral. The alkaline bile entering the stomach neutralises the acid of the gastric secretion, thereby increasing the irritability of the stomach and enhancing the action of the toxin of the fever itself on the cerebral vomiting centres.

Of course it would be useless in such cases to give any medicine by the mouth as a febrifuge or purgative, until the vomiting can be controlled, for nothing will be retained by the stomach and the medicine might just as well have been thrown away.

It has occurred to me that when the contents of the stomach are alkaline the most rational procedure would be to neutralise the alkali and restore the normal acidity of the gastric juice before resorting to other measures to control the vomiting. Acting on this I have employed the diluted hydrochloric acid (being the acid normally present in the stomach) with uniform success. Whenever I find the vomited matter neutral or alkaline to litmus paper, I at once administer diluted hydrochloric acid (♏ x. to ♏ xv. every hour or half hour) at longer or shorter intervals, according to the severity of the symptoms, until the vomiting ceases. Two or three doses are generally sufficient—sometimes the vomiting stops after the first dose—and then the patient will be able to take whatever remedy is required for his particular case.

I have given hydrochloric acid in nearly every case of bilious vomiting that has come under my care during the last three years and have not yet had to substitute anything else for it. Since I began giving hydrochloric acid for bilious vomiting I have learned that the natives of this colony sometimes employ lime-juice in these cases with excellent results, so it can hardly be said that the acid treatment is a new one.

I have occasionally given hydrochloric acid for the vomiting of pregnancy, generally with success, but not always with such marked improvement as when the contents of the stomach are rendered alkaline by the presence of bile in it.

Yellow Fever.

In the Fourteenth Report of State Board of Health of Pennsylvania a circular is quoted giving instruction with reference to yellow fever, about the possible introduction of which into the United States there was some anxiety during the recent war with Spain. Yellow fever is not contagious. The germ, when it leaves the human body, is not poisonous, but requires to pass through certain stages of development outside of the body before it acquires the pernicious activity. There is therefore no danger in nursing a yellow fever patient outside the infected district, as it is not difficult to destroy the infectious material as fast as it is thrown out in the various excretions, and thus prevent the growth of the germ.

It is a disease of low mortality. At least three-fourths of those attacked recover under the most unfavourable conditions of climate and environment, unless they are worried or frightened to death by over-anxious nurses, or drugged to death by over-zealous physicians.

With thoroughly judicious care and management, and under favourable conditions, not more than four out of a hundred should die. It will be seen that it is two and a half times less fatal than typhoid fever, eight times less fatal than diphtheria, ten times less fatal than Asiatic cholera, and twenty times less fatal than the plague. In little children it is especially mild, scarcely ever proving fatal, often escaping observation. There is no instance of its epidemic spread in places north of 35 degrees North Latitude, except, in the language of Professor Hirsch, "when the heat has equalled the mean annual temperature of the tropics; and it has on no occasion become diffused in a temperature below 68 degrees, the winter temperature of the tropics."—*Treatment.*

Protection from Insects.

A memorandum from the office of the Surgeon-General of the Army, issued as far back as June 18, 1890, does not appear to have attracted the attention of medical journalists. We present it here as being of interest to others than those to whom it was first addressed:—"The attention of medical officers of the regular and volunteer armies is invited to the following extracts from a letter addressed to the Surgeon-General by Prof. Stantord E. Chaillé, of the Medical Department, Tulane University of Louisiana, New Orleans:—'Instructions appear to have been issued to our soldiers for the preservation of health in the tropics, and I believe it well worth your consideration whether it would not be well to have disseminated among them information calculated to alleviate greatly their sufferings from mosquitoes, fleas, and other insects, sufferings which often deprive soldiers of the sleep and rest which, with food, are the primary requisites for their efficiency. During the Civil War, many soldiers blessed me for the following information: Mosquitoes, fleas, bedbugs, and, I believe, insects generally, detest and will avoid the essential oils and the plants which, with or without bruising, are strongly impregnated with their odour. In New Orleans, oil of lavender is used by many for relief from mosquitoes; a drop or two smeared on a hand or on any naked surface will protect until it dries up, say half an hour. While a Confederate surgeon I was in a town infested with fleas, and secured great relief by using freely in my boots and clothing and about my bed the bruised pennyroyal which grew abundantly within the town. Smearing exposed parts with grease will protect from mosquitoes until the grease disappears. A small piece, say one or two inches square, of fat middling or bacon to smear on exposed parts is more efficient than essential oils, because the grease does not dry up as soon, and would probably protect during the usual hours of sleep, if used at the beginning and repeated once during the night's rest."—*Journal of A.M.A.*

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THE

Journal of Tropical Medicine

APRIL, 1899.

PLAGUE.

BY the official records it is now known that at least some 200,000 persons have died of plague in India since its first appearance in 1896. The actual number is probably much greater, and has been variously estimated as between 300,000 and 400,000, for except in the largest and most important towns death registration is extremely defective. In country districts it is difficult to ascertain the actual number of deaths, while in the towns, where some more systematic attempt at registration is effected, though the number of deaths may be fairly accurately determined, the cause of death cannot be relied on, as there is no system of medical certificates and as the registration at the burning ghats and burial grounds depends on the statement of relatives or friends of the deceased. It was the recognition of the impossibility, under existing circumstances, of ascertaining the actual number of deaths which occurred from a particular disease, that led the Health Officer of Bombay to estimate the number of weekly and monthly deaths from plague in Bombay, as the figures obtained by deducting from the weekly or monthly total number of deaths the average of the corresponding period for the former five years, and taking the result as deaths from plague. By this calculation the deaths from plague were about double the number registered.

The destruction of life which the plague has caused, even if only the official statistics are considered, is enormous, and the most serious part of the position is, that notwithstanding all the prognostications as to the early disappearance of the disease, the hold which it has acquired in India is gradually slowly extending, and there are no signs of abatement. Bombay, on the contrary, has had its severest visitation this year, and the disease in this city, by all accounts, seems to have increased in virulence. People are dying, not only in greater numbers, but in a shorter time than in the previous years, the system being less able to combat the strength of the virus. It would seem that the epidemic is not to be deprived of the number of its victims, and that what the plague fails to accomplish in a short time it will effect in a series of years.

The comparatively few deaths which occurred in Bombay in 1896 and 1897 were the subject of congratulation, and were very reasonably attributed to the gigantic efforts of the Plague Committee, and it was felt that no such mortality as was recorded in the olden days of plague could happen again. We fear, however, that events are proving this view of the case to be somewhat premature. The plague is the same as ever it was. For reasons that medical science has not yet been able to discover, it attacks one city heavily, another lightly, and leaves a third alone, and however comfortable the doctrine may be, especially when the disease is at a distance, that it has lost all its old characteristics, this will in no way prevent it from insidiously extending first from one place and then to another, and possibly even to those centres of civilisation which are held by some to be invulnerable. Madagascar and Jedda are not within the pale of European civilisation, but they are near enough to the continent of Africa to cause alarm, and far enough from India to demonstrate that distance is not an infallible protection. The continued prevalence of the disease in Bombay has tended to lessen the interest and perhaps unnecessary

alarm which was first aroused, but the swing of the pendulum has probably gone too much in the other direction, for although the danger is counted less now, it is there all the same, and its potentialities are as great as ever, and demand careful watching.

Article for Discussion.

ALCOHOL IN THE DIETARY OF EUROPEANS IN WARM CLIMATES.

We are so accustomed to hear alcohol condemned off-hand as an article of diet in tropical countries, that it would seem as though the subject afforded but little ground for discussion. In spite of this, however, alcohol is consumed by the majority of European residents in every part of the tropics. During a military campaign it is the wise and well-nigh invariable custom to preclude all alcoholic stimulants, but in times of peace neither military, naval, nor civil authorities withhold alcohol from soldiers, sailors or police. The civilian engaged in commercial pursuits, however, is in no way restricted, and but few, if any, mercantile firms stipulate that the men they employ shall be total abstainers. No Insurance Company require their clients to be teetotallers, and the fact that alcohol is or is not indulged in does not affect the amount of premium to be paid. It may be argued, therefore, that statistics do not show that abstainers from alcohol have any reason to be classed as a specially favoured section of the community as regards the maintenance of health in the tropics.

It is a saying that "no man can drink beer or smoke a pipe after a two years' residence in tropical countries north of the equator." Beer, at any rate British brewed beer, is laid aside for claret or whisky, and the pipe is supplanted by the cigar. This is no doubt true in a general way, for beer after a time does not recommend itself, and although an occasional pipe is persisted in, the pipe-smoker is quite the exception.

The drink in India up to thirty years ago was brandy, but it has been supplanted by whisky there, as elsewhere. All kinds of ailments in tropical countries are ascribed to indulgence in spirits, amongst these hypertrophy of the liver, cirrhosis, liver abscess, splenic derangements, dyspepsia, diarrhœa, dysentery and Bright's disease, are the more prominent. It is doubtful, however, whether alcohol plays any more decided a part in these diseases in tropical than in temperate climates. We have recently been made aware of the fact that a microbe is the predominant factor in producing cirrhosis of the liver, and it would seem as though it played its part as effectually, if not more so, in Northern Europe as in equatorial latitudes. Liver abscess is a rare disease amongst drunkards in the tropics, and the teetotallers are as liable to hepatic abscess as those who indulge in alcohol in moderation. It must always be remembered that visceral change is a primary result of malarial infection, and the part that alcohol plays in determining these pathological states is difficult to determine. Many claim that a liberal quantity of alcohol counteracts the toxic influences of malaria, and persons imbued with such a belief use this as a pretext for indulgence.

There can be no doubt, however, that under certain circumstances and conditions, the effects are deleterious in the extreme. One who indulges freely in alcohol whilst exposed to the sun or during a military campaign, will soon succumb to the "exigencies of the climate." Heat stroke and siriasis are ever present dangers to the man who partakes of alcohol freely whilst taking active exercise in the sun, say, during a shooting excursion. On board ship also in the tropics, the heavy drinker is in imminent danger of sudden death from hyperpyrexia. What may be fatal under these circumstances need not, and does not, prove so to the man working in his shirt sleeves in a cool well ventilated office with a punkah waving overhead. It is of course difficult to define what is excess or even what constitutes a drunkard. Every man who "requires" to take alcohol before breakfast may be put down as a drunkard, and every man who drinks a half bottle of whisky a day, say six glasses, is drinking to excess.

J. C.

(To be continued.)

Correspondence on Articles for Discussion.

ARE ASIATICS WHO TAKE TO EUROPEAN FOOD AND DRINK RENDERED THEREBY MORE LIABLE TO DIABETES?

THE facts as regards diabetes in India are very interesting. It is difficult to obtain accurate statistical information regarding the prevalence of the disease, but the pages of the *Indian Medical Gazette*, from the year 1871 onwards, are full of the results of observation and experience recorded mostly by native medical men practising in Calcutta. The best papers are those contributed by Surgeon Kali Pada Goopta, M.B. (vol. xvi. for 1881, p. 119); Assistant-Surgeon Bolye Chunder Sen, L.M.S. (vol. xxviii. for 1893, p. 241); and Rai Kailas Chunder Bose, Bahadur, L.M.S. (vol. xxx. for 1895, p. 135). These were read and discussed at meetings of the Calcutta Medical Society, and they enter fully into questions of prevalence, causation and treatment, preventive and curative. Statistics relating to European and native troops and prisoners located throughout the Indian Empire are recorded in the Reports of the Sanitary Commissioner with the Government, and may be accepted as correct. The admissions per million of strength for each of these communities for the years 1892-96 were: European troops 68, Native troops 72, Prisoners 124. Death rates are valueless for comparison with those of other communities, on account of the elimination of the sick by invaliding and discharge; but the admission rates may be compared with death rates of the general population, because both probably include most of the diabetics whose disease is recognised, and repeated admissions are not likely to take place in the army or in prisons. The death rates per million recorded in the Registrar General's Report for England during the quinquennia 1876-80, 1881-5, 1886-90, and 1891-5 were 40, 51, 62, and 69 per million. The inferences deducible from these figures, taking age into account, are:—

(*a*) As regards Europeans, that they seem to be more prone to acquire diabetes in India than in England, and

(*b*) As regards natives of India, that the disease appears to be more common among them than among Europeans.

I have examined Indian dispensary reports, but the tables of diseases treated do not specify diabetes, and the tables presented in Indian sanitary reports are still more vague. Assistant-Surgeon B. C. Sen gives some statistics relating to the city of Calcutta, which he obtained from the health officer. They show for the quinquennia 1876-80, 1881-5, and 1886-90 death rates per million of 42, 61, and 80; but these are confessedly faulty, and valuable only in indicating a marked progressive increase, the reality of which is more than confirmed by general experience. These statistics also indicate that Hindus suffer from diabetes in much higher proportion than Mahomedans, and males than females. These conclusions are also supported by general experience, which further discloses the great preponderance of diabetes among the educated and wealthy of the Hindu community, and among Bengali as contrasted with up country Hindus.

It has further been remarked that an excess of cases is met with in particular sections of the educated and wealthy Bengali Hindus, notably among judicial officers of the subordinate Civil Service, while men who live by their brains, such as editors, doctors, lawyers, and schoolmasters, are also very subject to the malady, the type of which is of a chronic character, appearing in middle and advanced age, and with care pursuing a slow course, and not seriously impairing capacity, compromising health, or shortening life. Are these facts explicable by an affirmative reply to the question proposed for discussion in the March number of this journal, and which heads these remarks? The causes to which the prevalence and increase of diabetes in India have been attributed may be arranged under three categories, namely—(*a*) nervous strain and exhaustion; (*b*) altered dietetic habits; and (*c*) racial degeneracy. The special causative influences falling under category (*a*) are educational pressure, due to the university system, the harder

struggle for success in official and professional life, and sexual excesses; under (*b*), altered habits of feeding as regards material and times, the absence of rest after feeding, and, in some instances, the consumption of alcohol; and under (*c*), infant marriage, and residence in crowded insanitary cities. The comparative immunity of the country population; of the poorer working classes in towns; of Mahomedans, who have responded less keenly than Hindus to educational advance and social revolution; and of women, who are more uneducated and conservative than men, would indicate that these influences possess the causative powers attributed to them.

The native judicial officer, who seems to be specially subject to diabetes, has gained his position by hard brain work, and is compelled to maintain and improve it by the same means. He leads an inactive life, and spends most of the day in a stuffy court-house, while he probably bolts his food, and being well paid, indulges in rich food—particularly in *ghee* (butter) and sweetmeats. He may or may not consume animal food or alcohol, or adopt in part or whole the clothing and diet of Europeans. There are all kinds of variation in the circumstances and habits of diabetics; but there is, according to my experience, one thing that is common to them, namely, dyspepsia, preceding and accompanying this malady. Whether this has its origin in nervous exhaustion or in dietetic error, or is due to both, it is difficult to say. That the consumption of European food and drink, and assumption of European habits of life, are not alone, or perhaps principally, to blame is proved by the fact that many strictly "orthodox" Hindus suffer. There are races in India which are purely vegetarian, such as the Jains; others which consume as much animal food as they can afford, such as the Mahomedans; others which imbibe alcohol freely, such as the Sikhs; but these sections are comparatively immune. The disease is sometimes observed to be hereditary, descending from parent to child, and in other cases, to break out *de novo* in particular families, attacking one member after another, and in these instances, educational pressure and change of dietetic habits can often be eliminated. Personally, I incline to think that the causation of the disease is complex, and probably due to a combination of many or all of the influences above specified; and I am disposed among these to attach more importance, as a cause of disordered digestion and metabolism, to a departure from dietetic habits, and perhaps habits of life generally, to which races have become hereditarily adapted, than to the consumption of any particular article of food or drink.

KENNETH MACLEOD.

Reprints.

SPLENIC ENLARGEMENTS: SPLENITIS AND PERISPLENITIS.[1]

By JAMES CANTLIE, M.B., F.R.C.S.

Reprinted from *The Clinical Journal.*

(*Continued from page* 223.)

III.—*The anastomoses* by which the blood can pass from the organs of the abdomen to the general circulation when the portal vein is obstructed are as follows:

(1) At the lower end of the rectum a free anastomosis exists between the hæmorrhoidal veins, which reach the portal circulation by the inferior mesenteric vein, and those which convey the blood to the general circulation through the internal pudic vein.

(2) Through the œsophageal orifice in the diaphragm an anastomosis, capable of great development, takes place between the coronary (portal) vein of the stomach and the œsophageal (systemic) veins which ascend along the œsophagus to reach the venæ azygos.

(3) The coronary (portal) vein also anastomoses with the phrenic (systemic) veins, which convey the blood to both the inferior vena cava and, through the internal mammary vein, to the innominate veins at the root of the neck.

The above form the most constant anastomoses, but there are several others.

(4) Between the hæmorrhoidal (portal) and the vesical (systemic) veins a fairly free anastomosis is met with occasionally.

(5) The coronary (portal) vein of the stomach frequently communicates with the left renal (systemic) vein.

(6) The vasa brevia (portal) veins of the stomach,

(7) The gastro-epiploic (portal) veins, and

(8) The superior mesenteric (portal) vein may one and all have anastomoses with the left renal (systemic) vein.

[1] A Clinical Lecture delivered to the London Post-Graduate Class at the Central London Sick Asylum, December 1, 1898.

(9) The vasa brevia (portal) veins of the stomach anastomose with the left inferior phrenic (systemic) vein.

(10) When inflammatory adhesions take place between the liver capsule and the adjacent surfaces of the diaphragm or abdominal wall, vessels pass along the newly formed adhesions from the portal to the systemic veins.

(11) An anastomosis is sometimes found between the splenic (portal) vein and the azygos minor (systemic) vein.

(12) A free anastomosis of great importance takes place along the pathway of the falciform ligament, between the veins upon the inner wall of the epigastrium and the diaphragm (systemic), on the one hand, and the portal vein on the other. The largest branch accompanies the ligamentum teres, and joins the left branch of the portal vein; Sappey found this vein on one occasion as large as the little finger. The systemic veins involved are those in the substance of the rectus muscle, and the blood is conveyed up or down by the internal mammary to the innominate and the deep epigastric to the external iliac respectively.

When the abdominal veins are enlarged the deep veins are primarily affected, the surface veins being involved only at a late stage; therefore distended superficial abdominal veins indicate a long-standing and pronounced obstruction. Œdema of the feet and of the abdominal wall may precede the ascitic swelling, but with the ascitic accumulation the œdema becomes more marked.

IV.—*Perisplenitis—its frequency.*—Considering the anatomical and physiological relations and functions of the spleen, one can readily understand the reason for frequent adhesions resulting when any pathological condition supervenes. With well-nigh every movement of the body the spleen alters its position, and with every meal the spleen enlarges and again subsides when the plethora induced by the meal is over. The peritoneum covering the spleen is constantly moving upon the peritoneal surface of the diaphragm and stomach, and given a splenic congestion, inflammation, or irritation, the proneness to perisplenitic adhesions is readily understood.

The spleen is, moreover, not only the physiological burying-place of the normal corpuscles of the blood and the seat of their regeneration, but, when the corpuscles or blood plasma are charged with septic materials or become germ-laden, the spleen is called upon to deal with the germs freed of their host or carriers. Consequently we would expect, and in fact we find, the spleen becoming affected in almost every infectious or inflammatory fever. In measles, scarlet fever, typhoid, and many other diseases the spleen becomes enlarged, soft, pulpy, and tender. No more common cause of peri-splenitis is known than measles; and in fact when, from whatever cause, the temperature of the body is raised, the spleen shows evidence of being involved. These diseases are frequently associated with basal congestion of the lungs, and a plueritic rub is a common occurrence. This so-called pleuritic rub may be a misnomer; the rub is frequently not pleuritic at all, but is a peritoneal rub, caused by perihepatic or perisplenitic movement and inflammation. In many ailments we find at the base of the left lung a congestion, associated with vomiting, hiccough, and friction, and when the region of the spleen is being examined tenderness is elicited. These symptoms all point to the friction being of peritonitic rather than of pleuritic origin; in fact, that it is a perisplenitis and not a pleuritis that is present. In malarial fever we find the spleen very frequently enlarged, and perisplenitic adhesions common. The plasmodium malariæ is carried to the spleen in quantity,—not in driblets, but in sudden accessions corresponding with the paroxysms of febrile disturbance of quotidian, tertian, or quartan ague. In continued fever such as typhoid, the case is different; the infiltration of the spleen is less sudden, and the organ can accommodate itself to the altered state of the blood. Hence permanent enlargements are less common with continued than with the paroxysmal fevers. It is no doubt for the same reason that the spleen is so frequently of normal size in cirrhosis of the liver. The hepatic disease advances slowly and gradually, giving the spleen time to accommodate itself to the altered condition. By its elasticity, its trabeculæ, and by its muscular contractility the spleen can and does overcome venous engorgement when it is not called upon to do so too suddenly; but paroxysmal and sudden flushings of the spleen are apt to cause distention of such a nature that the elasticity of the organ is destroyed, and the power of resisting blood pressure gone.

V.—*Treatment.*—There are certain conditions of the spleen which can only be dealt with surgically. Tumours of the spleen, whether simple or malignant, may require the removal of the whole viscus. The spleen is seldom attacked primarily by malignant disease, so that excision for such growths is seldom called for. Simple cystic tumours of the spleen, when of great size and the splenic tissue wasting, are the most satisfactory tumours of the spleen to deal with surgically.

Several tumours of this nature have been removed, and the patients made good and lasting recoveries, and the microscopic examination some twelve months afterwards showed that the blood in no way differed from the normal; compensatory splenic action being no doubt performed by some other organ or organs.

When ascites is present the patient must be tapped, not once, but many times if necessary, and this operation is frequently attended by relief and permanent good. The operation is rational, as it is merely assisting nature to accomplish what she is attempting to do rather awkwardly. But it is useless to be content with one or two tappings; one is apt to assume because the fluid recurs after the first or second operation that the process is useless. Not so. I tapped a patient (a Chinaman) for cirrhosed liver and enlarged spleen eight times in all. Between the first two some ten days only intervened, but the intervals gradually lengthened until, between the seventh and eighth, four months intervened. The fluid never returned, and the patient was able to attend to his business and get about as usual.

When the spleen is ruptured, and signs and symptoms of alarming internal hæmorrhage apparent, the surgeon is justified in cutting down upon the spleen and removing it if all other steps prove unsatisfactory.

A wound of the spleen does not bear stitching well, the soft friable tissue breaks away, and futile efforts in that direction may so prolong the treatment that the patient dies from loss of blood. There is no operation much more simple than removing a spleen which is free of adhesions; but if the adhesions are many, and the union between it and the neighbouring parts close, nothing is more difficult. The danger is not peritonitis, but secondary hæmorrhage from the ligatured stump. Provision must be taken to prevent this by applying numerous ligatures to the splenic pedicle during the operation, and securing absolute rest for twenty-four hours after the operation.

Abscess of the spleen is more common than text-books would have us believe. The evidence in support of this is unfortunately generally *post-mortem*, and not clinical. The surgeon finds considerable difficulty in dealing with splenic abscess; the structure of the spleen is such that one cannot freely insert the needle of the aspirator in search of pus, as in the case of the liver. The withdrawal of the needle may be followed by severe hæmorrhage, or the escape of pus into the abdominal cavity. When abscess is suspected the spleen should be exposed, and the abscess dealt with either by evacuation or the removal of the whole organ.

Direct medicinal treatment of enlarged spleen is, to say the least of it, equivocal. When the disease is combined with anæmia and ascites, severe purgation is calculated to tax the patient's strength too severely. Good, however, will result by procuring gentle and regular purgation, and this is best accomplished by salines. Of these sulphate of magnesia is the most reliable and effective, and in the form of magnesia sulphas effervescens, in half-ounce doses, given in a small quantity of water every morning, or more often, is the most agreeable. Should circumstances permit, the determining cause of the splenic enlargement may necessitate constitutional remedies. Syphilitic visceral disease in which the spleen is involved requires appropriate remedies, and in the advanced stage of the disease increasing doses of the iodide of potassium is our sheet anchor. Iodides are recommended for splenic enlargements other than syphilitic, and in passive enlargements of the spleen their use is rational.

When cardiac disease is the origin of the trouble, appropriate remedies must be applied,—now to relieve the effects of visceral congestion, now to strengthen the heart muscle, in order to aid the organ to accommodate itself to the disturbed circulation.

Iron has to be used with judgment in splenic anæmia; mineral acids with chloride of ammonium are effective, more especially when the liver is enlarged at the same time as the spleen.

Quinine is not of much direct value in reducing the enlargement of the spleen in chronic malaria. In this condition change of residence to a non-malarial district is the most effective. A prolonged stay in a temperate or cold climate is the only effectual remedy for splenic enlargements dependent upon malaria, and even in a cool climate the ailment will disappear much more readily when the patient dwells at high altitudes. As in malarial so in all splenic enlargements associated with anæmia, be their causes what they may, it is only at high levels that speedy advantages are to be attained.

Local applications over the splenic regions have some arguments in their favour. Counter-irritation by the liniment of iodine or specific unction by the unguentum hydrargyri iodidi rubri or the oleate of mercury have claims to consideration.

Residence in a city, or at the sea level by the sea-side, will but tend to aggravate the disease, however salubrious the chosen spot may claim to be. At high altitudes alone can the benefits of a cold or temperate climate be fully gained. "The open-air" treatment is an imperative necessity in all splenic troubles. The diminished and altered corpuscles of the blood show, by the anæmic state of the patient, their inability to carry sufficient oxygen to nourish the body satisfactorily, and it is essential that the best and purest supply of fresh air should be provided to compensate for their attenuated condition.

Recent Literature on Tropical Medicine.

OPHTHALMOLOGY IN ITS TROPICAL BEARINGS.

Epidemic Night Blindness. — Dr. Schtschepotzew (*Knapp's Archives*) describes an epidemic form of night-blindness occurring in Russia, which, according to his account, differs in some important respects from the disease as seen in tropical countries. He believes in the malarial nature of this curious affection, as outbreaks occur chiefly in low swampy localities, and quinine has a distinct curative effect. Well-nourished, robust people are often affected, and the disease appears to have no predilection for the anæmic and weak; in the tropics, on the contrary, it is almost invariably associated with general mal-nutrition. Night-blindness is more prevalent in the northern parts of Russia than in the southern, and reaches its greatest intensity in the spring, showing that bright light has little to do with its causation. The author notes mydriasis and cycloplegia as almost constant symptoms, and believes that the disease is due to vaso-motor disturbance.

Extract of Supra-renal Capsule in Operation for Pterygium.—Mullen (*Ophthalmic Record*) has used extract of supra-renal capsule in conjunction with cocaine in obtaining anæsthesia and anæmia of the conjunctiva prior to removing pterygia. He uses Armour's preparation of the capsule, 5 grs. to an ounce of water. A drop is put in ten minutes after the 5 per cent. cocaine solution has been used; the anæmia produced by it is most pronounced; there is no danger and healing is rapid.

Excision of the Conjunctival cul-de-sac in Trachoma. —Galezowski (*Recueil d'Ophthalmologie*) advocates the excision of the conjunctival *cul-de-sac* in cases of granular ophthalmia which resist the ordinary modes of treatment. The operation is a simple one, and in his hands has led to cure in the most obstinate cases. He formulates three conditions as necessary to ensure the success of the excision; (1) conjunctiva only to be removed; (2) it must be excised well into each angle; (3) the tarsus should not be touched.

Argyriasis after Protargol.—Dr. K. Denig, at a recent meeting of the New York Academy of Medicine (*Knapp's Archives*) showed a case of argyriasis in a man who had used a 5 per cent. solution of protargol for four months, instilling one drop three times daily, although he had told patient to return in four days. The case is interesting, as it has been claimed that such a result never follows this remedy.

AN OINTMENT FOR TRACHOMA.—M. Blöbaum, of Cologne (*La Semaine Médicale*, February 15), has found a combination of sulphate of copper and salicylic acid very useful in trachoma. He employs the following ointment: sulphate of copper, salicylic acid, hydrochlorate of cocaine, of each 1 part, vaselin, 10 parts. The presence of corneal ulceration does not contra-indicate the use of the ointment.

M. T. YARR.

FRANCE.

CARDIAC PHENOMENA IN DYSENTERY.

DYSENTERIE A FORMES CARDIAQUES. — Under this title Dr. V. Thébault has recently published an interesting description of an epidemic of dysentery occurring in the garrison of Vincennes during the summer of 1897. So far as the intestinal condition was concerned the disease usually ran a mild and tractable course to complete recovery. The striking feature of the epidemic was the frequency of functional disturbances of the heart. The dysentery itself appears to have been of a highly infectious type, for, of the hospital orderlies in attendance, no fewer than six were attacked.

Of the 159 cases observed, 67 exhibited the cardiac symptoms referred to. In 60 of these the symptoms occurred as follows: cardiac symptoms set in shortly before the dysenteric phenomena declared themselves in 6 cases; simultaneously with the dysenteric symptoms in 6 cases; four to twenty-five days after the onset of dysentery in 39 cases; from four to ten after complete recovery from dysentery in 5 cases; during relapse of dysentery in 4 cases.

The cardiac phenomena consisted of bruits, asthenic conditions, tachycardia, bradycardia, and arythmia of varying degrees of severity and in various combinations. After a time the cardiac symptoms gradually subsided. At the *post-mortem* examination of the two fatal cases no heart lesions which could be held to account for the symptoms during life were discovered. Thébault concludes that the cardiac condition was attributable to poisoning of the vaso-sympathetic by a toxin of microbic origin, and that in this respect *dysenterie à formes cardiaques* is comparable to similar phenomena in erysipelas, scarlatina, septicæmia, &c., and, we may add, beri-beri.

It would be interesting to learn if any of the many forms of tropical dysentery is ever associated with similar cardiac phenomena.

AMERICA.

ARMY RATION IN CAMPAIGNS IN TROPICAL CLIMATES.

In the *New York Medical Journal* of March 18, 1899, there is an article on "The United States Army Ration and its Adaptability for use in Tropical Climates," by Louis L. Seaman, M.D.

"In the Spanish-American war just concluded, the United States soldier who saw service in the Cuban and Porto Rican campaigns passed through several ordeals he should never be called upon to repeat. This paper relates only to one —namely, his experience with tropical diseases without a suitable dietary. The report of the adjutant-general of the army, September 30, 1898, gives the number of those who died from wounds during the few brief weeks of actual hostilities as 345; but the hospital records show appalling lists of sickness and death, the mortality from disease alone reaching 2,485, or about 80 per cent. Had these proportions been reversed, the figures would have been nearer those to be expected in an age so advanced in sanitary science and dietetics. For the time from the landing of the army at Siboney and the fight at San Juan Hill, to the signing of the protocols, a period of less than seven weeks, was an interval too short for disease other than that of an epidemic of preventable character to develop into serious proportions, especially when the class of men exposed were those selected for the special service. It is true the conditions encountered were somewhat formidable, the season of invasion being the worst that could have possibly been selected, the heat and moisture of the summer months combining to produce that extreme relaxation of the system which was a factor in the production of the many forms of tropical diarrhœa among the troops. But we must look beyond climate for the full responsibility.

"From earliest times, history has shown that there has always been a greater mortality from *disease* among armies during a war than from wounds, especially in tropical countries; and Spain, keen in her appreciation of this fact, and knowing also that she *had* to fight, courted a summer encounter, convinced that the enemy which would devastate our ranks was disease, rather than the guns of her Morros. In this she was right: but the aid received by the Spanish from the source mentioned was much greater than it would have been had our troops been appropriately fed. Duncan, the highest recognised authority in military hygiene, states in reference to campaigns in the tropics that, 'although there is an undoubted distribution of disease, yet it is important to remember that so far from being unable to cope with it, we can act in antagonism to it. Take the class of bowel complaints: by insuring wholesome food, solid and liquid, we can remove all causes of irritation from within; by insuring a rational dress, we can remove all irritation from without. Again, in the matter of conservancy, by insuring the absence of putrefaction from the camp, and the burning or disinfection of the excreta, we cut the ground away from the feet of cholera, enteric fever, yellow fever, and bowel complaints. The army sanitary commission, relative to the rate of invaliding in Madras, distinctly states, in its memorandum of March, 1880, that much of the loss from invaliding from Indian climates is due to the continued action of eating, drinking, and clothing directly opposed to the requirements of the climate.' After having considered the factors of temperature and moisture as applied to hot climates, he further says: 'There are two remaining great elements—namely, fatigue and insufficient nourishment—to guard against. Indeed, it has been held that climate *per se* is secondary to these; doubtless, as regards temperature and moisture, this is the case, for if a man be properly fed and not overworked—that is, if his mechanical work be proportioned to his individual powers, and his food in accordance with his proportioned work—the factors of temperature and moisture will have but little power on him, except in extreme instances.'

"For a moment permit me to call your attention to the chart of temperatures as they existed in various parts of our country December, 1898, January and February, 1899.

"*Range of Temperature in United States Territory.*

"From published reports of the signal service bureau, the temperature was as follows:

"*February*, 1899.—Alaska ranged as low as.....		— 50°
"*January* 31, 1899.—White River, Canada (near Fort Brady, United States)		— 40°
,, ,,	Green Bay.....	— 26°
,, ,,	Kansas City.....	— 4°
,, ,,	New York.....	+ 16°
,, ,,	Key West.....	+ 60°
"*December* 24, 1898.—Santiago de Cuba.....		+ 70°
,, ,,	Manila	+ 90°
,, ,,	Havana (in the sun).....	+ 104°

Extreme variation, 154° F.

"Captain C. E. Woodruff, United States army, now stationed with the army in Manila, in an admirable article on Military Food published in the *Journal of the American Medical Association*, in 1892, uses the following simile: 'If two ships were to start from New York, each to be absent several years, one in the arctic regions and the other in the tropics, no one would ever dream of provisioning them alike. Yet, if two armies were similarly to start from New York for long periods, one to the extreme North and the other to the hottest parts of the South, the *law* presumes that both shall carry essentially the same rations. We have not yet

reached the point where it is decidedly recognised that the variety in the ration must be great enough to permit of sufficient flexibility to suit extremes of climate.'

"In a country of such wide ranges of temperatures as the chart indicates, the health of the soldier on active service or in garrison rests primarily upon his being provided with food and clothing suitable to his environment. The government, liberal in all things, freely issues these supplies; but, strange to say, while making certain distinctions in the uniform of the soldier who must endure the rigors of northern winters and the soldier on southern or tropical service, there has never, apparently, been any official consideration given to the diets which climates of such radical differences require. The same ration serves in all sections, and, while it is generous in quantity and usually excellent in quality, it is not such a one as can be universally used in hot and cold climates alike, if due regard is to be paid to the health of the soldier. Rich in nitrogenous, heat-producing elements, it is better adapted for the bodily needs of the soldier stationed in the North; but in extreme southern latitudes and in the tropics it proves an active agent of disease, overheating the system and producing those conditions predisposing to fevers and intestinal and rheumatic diseases, and rendering the labours of the surgeon unavailing. 'The enormous number of cases of rheumatism,' says Woodruff, 'occurring during the Rebellion and since the Rebellion in veterans, may not be entirely due to exposure, as popularly supposed. These men were hardened to exposure and should not have had more rheumatism than hunters, or trappers, or the aboriginal Indians. The limited, often insufficient ration and the absence of fresh articles of diet may have been one of the factors at work.' The intimate relationship between diet and disease is a consideration demanding our closest study.

"At the beginning of the century the ration of our army, established by an Act of Congress, consisted of the following articles: Fresh beef, a pound and a quarter; or salt beef, one pound; or salt pork, three quarters of a pound; bread or flour, eighteen ounces; and for every hundred rations issued, a gallon of whisky, four quarts of vinegar, and two quarts of salt.

"In 1808, Dr. Edward Cutbush, of the United States Navy, commenting upon it said: 'With respect to the component parts of the ration, I think it defective without an allowance of vegetables or pulse occasionally in garrisons.' And again, 'The ration of meat should be diminished, and plenty of vegetables issued in lieu thereof.'

"Whether Dr. Cutbush's article had anything to do with it or not (departmental movements are slow in the army), the ration in 1818 was vastly improved by the addition of dried vegetables. In 1861, at the outbreak of the Civil War, it was still further expanded, but in 1864 it went back again to what it had been prior to 1861. In 1890 it experienced another change, fresh vegetables again being added. I am not aware of any great changes made in it during the past eight years. To-day it is composed as follows: Fresh beef, or fresh mutton, when the cost does not exceed that of beef, twenty ounces; or pork or bacon, twelve ounces; or salt beef, twenty-two ounces; or, when meat cannot be furnished, dried fish, fourteen ounces, or pickled fish or fresh fish, eighteen ounces. Flour or soft bread, eighteen ounces, or hard bread, sixteen ounces; or corn meal, twenty ounces. Baking powder for troops in the field, when necessary for them to bake their own bread, $\frac{16}{25}$ ounce. Beans or peas, 2·4 ounces; or rice or hominy, 1·6 ounce. Potatoes, sixteen ounces; or potatoes, 12·8 ounces, and onions 3·2 ounces; or potatoes, 11·2 ounces, and canned tomatoes, 4·8 ounces, or 4·8 ounces of other fresh vegetables. Coffee, green, 1·6 ounce, or roasted coffee, $1\frac{7}{25}$ ounce; or tea, $\frac{8}{25}$ ounce. Sugar, 2·4 ounces; or molasses or cane syrup, $\frac{16}{25}$ gill; vinegar, $\frac{8}{25}$ gill; salt, $\frac{16}{25}$ ounce; pepper, black, $\frac{1}{25}$ ounce; soap, $\frac{16}{25}$ ounce; candles, $\frac{8}{25}$ ounce.

"This is the ration the soldiers in the Northern States and Alaska were drawing in the winter of 1897-98, and, without any change in a single constituent, it was the food intended, but not provided, for the American troops in Cuba, Puerto Rico, and the Philippines, where the temperature ranged between 85° and 95° F., and frequently reached much higher figures.

"The ingestion of food is to effect two results—the development of body heat and bodily motion or energy. As a general proposition I might state that the latter is stable, and that similar motions will produce similar amounts of heat, the quantity and quality of food necessary to respond to this demand being determined by occupation. On the other hand, the quantity and quality requisite to maintain body heat will vary with exposure and environment. The individual labouring in the open air will require foods of greater heat-producing power than he who is employed within doors; and he who lives in a cold climate must eat more of the starches and fats to produce the body heat necessary to existence than he who lives in the tropics. For with an increase of external temperatures less body heat is required, and the appetite for fat, which was strong in the cold climate, will diminish proportionately. To quote once again from Duncan: 'The mutual relation of income and expenditure has been calculated to a certain degree of exactitude. Taking Ranke's well-known standard diet, it is found that it yields about a million units of force or metre kilogrammes. Now, a good day's work equals 150,000 metre kilogrammes. Subtracting this from the total would give 850,000 metre kilogrammes as expended in heat production; or, in other words, a sixth of the total income of food is expended as mechanical force and five-sixths as heat.'

"Dr. Carpenter on this question (the lesser production of body heat where the external temperature is increased) puts it thus: 'Every change in the organic components of the body in which their elements enter into new combinations with oxygen must be a source of development of heat; and as a considerable portion of the carbon dioxide and water exhaled in respiration is formed within the body by the metamorphosis of its own tissues, and since the metamorphosis is promoted by the active exercise of the nervo-muscular apparatus, it follows that in animals whose habits are peculiarly active, living in climates in which the surrounding temperature is high enough to prevent any cooling influence, the combustive process thus maintained may be adequate for the maintenance of the temperature of the body at its own normal standard. Hence, here it would appear that we do not want to provide for the heat of the body in the tropics, but only for the work done *where there is a peculiarly active life.*'

"Carpenter next says that the general experience of inhabitants of warm climates is in favour of a diet chiefly or entirely vegetable, inasmuch as such a diet affords an adequate supply of the albuminates in combination with the other classes of food, without affording more fuel than the system requires.

"These statements have an especial interest when considered in conjunction with the *adaptability* to the tropics of our own army ration. The life of the soldier was sufficiently active to 'maintain the temperature of the body at its own normal standard,' the surrounding temperature in the West Indies having been 'high enough to prevent any cooling influence'; and without the assistance of a 'highly animalised diet,' such as our ration was and is to-day. My own experience, and that of others, agreed with the 'general experience of inhabitants of warm climates.' We had little craving for meat, our appetites, on the contrary, inclining us toward vegetables and fruits. Left to natural selection, the appetite will always incline to the food supplied by Nature in the particular climate of the individual's environment; and any unusual craving for food primarily belonging to another zone is unnatural and due to an artificial appetite.

"The food products of each zone will be found in every case to be those that are peculiarly adapted to the particular needs and requirements of the zone's inhabitants, and where

the climate creates a demand for an excessive animal diet, Nature furnishes it, or *vice versâ*. A glance over the earth's surface will show this. In the arctic regions there is a great dearth of edible vegetation, but animals and fish abound, whose flesh, fats, and oils furnish the rich heat-producing foods required by the people who live in those lands of almost perpetual snow and ice. In the tropics these conditions are reversed. There is a scarcity of animal food, but an abundance of vegetation that yields nourishment with low heat-producing qualities. Between these extremes, in the temperate zone, we find a mixture of both and each in plenty. And here it might be well to pause and review the procession of the seasons of the temperate zone, observing the effect each has upon the food desire, and the means Nature has taken to meet it. As the spring days lengthen and become warmer, the early garden vegetables make their appearance; summer with its increased heat brings a profusion of vegetables and fruit, the supply of which gradually decreases as autumn approaches; and winter finds us with few fruits and vegetables that can be kept through its icy months, but with butchers' shops well stocked with savoury meats. With these changes our appetites are in perfect sympathy. The early spring vegetables are eaten with a keen relish, our tables in the summer months bear more vegetables and fruits, and in the winter the large roasts and steaks again have the place of honour, and small side dishes are sufficient for all the vegetables our appetites crave. Dr. Koerfer proclaims in the *Deutsche Medizinische Wochenschrift* of last July, that if Europeans would leave their pork fat, their meats and their alcohol at home with their furs and heating stoves when they go to reside in the tropics, they would avoid all disturbances that are erroneously ascribed to the climate, but which are, in fact, only due to the failure to conform to Nature's laws. He considers that Nature makes the food conform to the climatic conditions—from the fish-oil polar zone through to the pork-fat temperate zone to the olive-oil and vegetable tropic zone. He writes from an experience of several years in the tropics, and adds that he felt better subjectively when actively employed than when lounging, no matter how high the temperature.

"I am aware that many of the inhabitants of the temperate zone make but little change in their dietary as the seasons vary; but how many cases of dyspepsia, indigestion, and other ills do we not treat yearly, and how many more ailments and diseases, particularly in middle age or advanced life, could be traced to this very cause if we but carried our investigations back to the root of the disorder!

"Let me invite your attention for the moment to the ration of the British soldier in India for the purpose of contrasting it with our own. It consists of the following articles: Meat, with bone, sixteen ounces; bread, sixteen ounces; potatoes sixteen ounces; rice, four ounces; sugar, 2·5 ounces; tea, 0·71 ounce; salt, 0·66 ounce.

"You will observe that the meat component is less by from four to six ounces than that of the United States ration, although Tommy Atkins is one of the meat eaters of the world. The bread allowance is less by two ounces, but the rice is greater by 2·4 ounces.

"The potential energy of the American ration as compared with the ration of the British soldier is more than a fourth greater, when expressed in caloric units—the American being 3,800, the British 2,800: while the caloric units in the ration of an English prize fighter, as given by Gillespie, of Edinburgh, is but 2,200. In both the English and American army ration the proportions of proteids and carbo-hydrates are equal, the excess in heat equivalent in the United States ration being in its larger proportion of fats.

"Stewart Clark, inspector-general of prisons. N.W. Provinces, India, in his 'Practical Observations on the Hygiene of the Army in India,' says that 'even *this* ration of the British army is, perhaps, more faulty in being *too* liberal than in any other respect; for it is now well known that the quantity of food in a tropical climate is much more frequently to blame than the quality, in causing impaired health, such as disorders of the liver, dysentery, diarrhœa, and other complaints attributed to the climate.' After stating that the most abstemious are the healthiest men in India, he further says that no change contributed more to the health of the European resident than the discontinuance of heavy midday luncheons, and the reduction of meat in the bill of fare of the better classes to one meal a day. He adds that 'the greatest defect in the diet of the European soldier is the want of a due amount of vegetables. If the want of a plentiful supply of this most essential article of diet is the cause of disease in other groups of individuals, why should it not be equally so among soldiers? In fact, they are often so badly supplied with vegetables that there can be no doubt that dysentery, diarrhœa, and other complaints, which often assume a scorbutic character, may in a great measure be attributed to this cause.'

"Even Dr. Benjamin Rush, physician-general to the Military Hospital of the United States, in his 'Direction for preserving the Health of Soldiers,' published in 1808, says: 'The diet of soldiers should consist chiefly of vegetables. The nature of their duty as well as their former habits of life require it. If every tree on the continent of America produced Jesuit's bark, it would not be sufficient to preserve or to restore the health of soldiers who eat one or two pounds of flesh in a day.'

"These few citations which I have made, and I have made them because they especially bear out my own observations in the tropics, for no originality is claimed in this paper, will sufficiently indicate that the results of a too excessive meat diet in tropical climates furnish no new field for investigation. Why, among civilised people, meat should have continued to form a major part of the soldier's ration after its serious effects have been pointed out by the best medical authorities for nearly a century, is past comprehension."

(*To be concluded.*)

Antitoxin—Untoward Effects.

Dr. J. N. Morse (*Boston Medical and Surgical Journal*) reports the following case of antitoxin untoward effects:—

A. B., male, aged 32, and as far as known in good health, developed on April 28, after a week of continuous exposure to diphtheria, a slight sore throat. The throat was generally reddened and rather dry. No culture was taken. As a precautionary measure he was given five cubic centimetres (five hundred units) of the Massachusetts State Board of Health antitoxin. It was injected with aseptic precautions and no local symptoms developed at any time at the seat of injection. The throat was entirely well by May 1.

The patient remained in his usual health until 12.30 p.m., May 3, when a slight urticaria was noticed. By 1.15 p.m., there was marked malaise, chilliness and vertigo. At 2.15 he fainted while telephoning. He was put to bed. The body, face, and extremities were found to be covered with large blotches of urticaria, so profuse as to be almost confluent. The chilliness and vertigo continued and nausea developed. There was no headache, however, and the temperature was normal. There was no diminution of the urticaria until 6 a.m., May 4, and it did not entirely disappear until several days later. The whole surface of the body was covered all the time, but the severity of the process varied in different portions at different times. At one time the face would be puffed up, at another the feet would swell enormously, at another the hands, and so on. The swelling was greater in the feet than elsewhere, however. The thighs were deep purple, and remained so for several days. The nausea continued, and by 6 p.m. vomiting came on, and continued uninterruptedly every fifteen or thirty minutes for twelve hours. The vomiting was watery, acid, and occasionally bile tinged. During the night the

uvula and pharynx became considerably œdematous. This undoubtedly extended down the œsophagus to the stomach, as swallowing was difficult and caused pain throughout the whole length of the œsophagus. This was not relieved before the night of May 4. The respiratory mucous membrane was fortunately not involved. The temperature remained normal, but the pulse was rapid and irregular. There was no diarrhœa. There was, however, almost complete suppression of the urine, only three or four ounces being passed in twenty-four hours. This was very thick and high-coloured, but did not contain albumin. At the end of the first day a general glandular enlargement had developed, which lasted for about ten days. Those in the groins were as large as walnuts. The patient was much prostrated, lost nearly ten pounds in weight, and was unable to resume his work for a week.

J. W. M'Laughlin, M.D., writes to *Medical Brief*:—

Here is a remedy for diphtheria which has never failed in my hands during a practice of more than twenty-five years:—

℞. Zinci Sulph. ... ... 1 drachm.
Aquæ ... ... 4 ounces.
M. Sig.: Apply with swab.

It will remove membrane in two hours. Apply every half hour till throat is clean, then two hours apart. Control fever with veratrum viride, acetanilide or quinine, as case requires. Keep bowels open; secure free discharges two to four times a day. Diet principally milk, or milk and mush, or bread.

GERMANY.

Quinine in Labour.

Schwab says that, while quinine sulphate has little or no power for inducing labour pains, he is positive that after uterine contractions have begun the administration of quinine causes them to become rapid and energetic. While quinine strengthens the labour pains, it does not tend to induce abortion. Unlike ergot, it causes intermittent and not tetanic contractions, and may, therefore, be prescribed without danger during the second stage of labour. Its action begins in about one-half hour: the drug is, therefore, best given in two doses of eight grains each, within a period of ten minutes. He has obtained excellent results from its use in all cases of prolonged labour due to uterine inertia. While quinine has a tendency to produce post-partum hæmorrhages, this is easily controlled by massage of the uterus.—*Int. Med. Mag.*

The Bacillus of Syphilis.

Van Neissen (*Centralblatt für Innere Medicin*, May 7, 1898) has made some interesting experiments on the cultivation of a micro-organism from the tissues and blood of syphilitic patients, and the production of symptoms resembling syphilis in animals after inoculation of the cultivated microbes. He claims to have found a streptobacillus in the secondary stage. For purposes of cultivation he employed bone marrow and the cartilaginous parts of the epiphyses of the long bones and ribs from infants with hereditary syphilis, and also from secondary syphilitic products. The pieces removed were washed in perchloride of mercury, split longitudinally, and scraped with a spoon sterilised by heat; the portions removed by the spoon were cultivated on bouillon, and afterwards on agar, serum and gelatin. A small streptobacillus or streptococcus was found regularly, this being identical with an organism Van Neissen had formerly found in the blood in cases of paralytic dementia and tabes dorsalis. Van Neissen considers this organism specific of syphilis. Inoculations were made subcutaneously and by intravenous injection in pigs and rabbits. In the former there was slight induration at the point of inoculation, followed in eight or ten days by bright red spots on the skin, which disappeared in about a week. In the rabbits there was typical induration at the point of inoculation, after which the animals were paired, and the female gave birth to seven dead fœtuses, two of which had the appearances of syphilitic maceration.

Examination of the blood in the secondary stage of syphilis gave mainly negative results, and from this the author assumes that the contagium is at this time for the most part confined to the skin.—*Treatment.*

Blood Parasites in Beri-Beri.

F. Fajardo describes a small unicellular organism which he has observed in the blood of cases of beri-beri. The organism is found both inside and outside of the red corpuscles. It is smaller than the parasite of malaria, but like it seems to form pigment granules in its interior and to undergo a cycle of development. It is demonstrable not only in the blood of the internal organs, but also in the peripheral circulation.

Dissemination of Leprosy Bacilli from the Upper Air Passages.

Schäffer, in the *Archiv. f. Derm. u. Syph.*, 1898, of Breslau, has made some experiments to determine this question. It was first shown by Neisser's histological preparations that the upper layers of the skin—not only the epidermal structures but the papillary layer of the corium—are free from bacilli in leprosy. It has since been shown that the bacilli are sometimes found in the lumen of the sweat-glands and in the inner root-sheath of the hairs. Hence it is not improbable that the bacilli may gain egress by these channels to the surface of the skin. So far, however, as we have any means of determining, this seldom takes place, although we need far more experimental work before the question is definitely settled. Even if it were found that the bacilli could frequently gain entrance in this way, the danger from this mode would not be very great, as they could be carried to other people only by contact, or by clothing, &c. Schäffer does not believe with Cornil that the leprosy bacilli leave the body only when deprived of life, but thinks it probable that if they remain long on the dry epidermis they gradually die. In any event the danger of an infection in this way may be reduced to a minimum by cleanliness and precautionary measures. In the case of open ulcers the conditions are much more favourable for the life of the numerous bacilli upon the surface. But here, too, by disinfection and antiseptic dressings, the chances of propagation may be made very small.

A method of dissemination of the leprosy bacilli which has not received sufficient attention is that by way of the mucous membranes of the upper air passages, and especially of the organs concerned in speaking. Flügge has shown that in speaking, coughing and sneezing, very minute particles of the secretions of the mouth and nose are disseminated to a considerable distance. Experiments upon these lines were undertaken by Schäffer in cases of leprosy, where lesions occur with great frequency on the mucous membranes of the air passages, and are uncommonly rich in bacilli. The subjects of these experiments were two patients afflicted with tubercular leprosy, in which the nose, mouth and larynx were affected with ulcerated lesions in which numerous bacilli were found. The method used was to cause the patient to speak in his ordinary voice for ten minutes, while a large number of microscopical slides were arranged before him. At the end of this time the slides were found to be covered with very numerous drops of varying size. These preparations, when stained by the Ziehl-Nielsen method, showed large numbers of bacilli that were considered to be the specific micro-organisms of leprosy, besides some other varieties. The number of leprosy bacilli in the different drops varied greatly; in some of the larger

drops but few were found, while oftentimes they were present in great numbers in the smaller ones. It was shown that in ten minutes several thousand leprosy bacilli were thrown out. In some instances they were detected at a distance of one and one-half metres from the patient. Experiments as to the possibility of checking this throwing out of bacilli proved that it could with difficulty be diminished by rinsing the mouth, and touching the ulcerated surfaces with nitrate of silver, but that it could not be wholly checked. It was found that comparatively fewer bacilli were thrown out by coughing than by speaking. In ordinary expiration few bacilli were given out.

Several experiments showed that very great numbers of bacilli were given out in sneezing, in one instance more than 110,000. The conclusion is therefore reached that in lepers in whom there is an affection of the mucous membranes of the air passages, not necessarily of an extreme grade, thousands of bacilli are thrown out to a considerable distance in speaking, coughing and sneezing, and that this dissemination cannot be prevented by therapeutic measures. Schäffer confesses his inability to explain the reason why infection with leprosy is so infrequent, despite the abundant opportunity for receiving the bacilli in the neighbourhood of those suffering from the disease. Even if the bacilli that are thrown off are in part dead, as is maintained by many, the number of those thrown off is so great that the chances are that some at least are living; and if we accept the theory that they have little power of resistance to external conditions of temperature, &c., it would seem that their chance of propagation was particularly good here, when they are thrown off in a moist, warm medium.

At the International Leprosy Conference in Berlin it was shown that in a very large number of cases there was good reason to think that the bacilli gained entrance to the body through the mucous membrane of the nasal passages. This accords well with the theory that the throwing off of the bacilli from the air passages is a frequent mode of infection, as the minute drops containing the bacilli that are thrown off could be easily carried to the mucous membrane of the nose on inspiration.

These experiments seem to teach the importance of prophylaxis in cases where the mucous membranes of the air passages are affected. This can be partly accomplished by interdicting a close relationship with the healthy, and by careful regard to the local treatment of the lesions of the mucous membranes, although the throwing off of the bacilli cannot be wholly prevented in this way.

Schäffer asserts, in closing, that his object is not to create unnecessary alarm, as the danger of infection from leprosy is remarkably small, but to point out the probability that a dissemination of the bacilli from the upper air passages is relatively the most important of the various ways of infection.—*Boston Medical and Surgical Journal*, March 16, 1899.

INDIA.

Geological Strata and Plague.

Mr. G. L. Greisbach, Director of the Geological Survey of India, said, in his evidence before the Plague Commission, that from the list of localities it appeared that by far the largest number of plague cases had occurred on soil covering Deccan trap and crystalline rocks, and only a very small number on the great alluvial belt stretching from the Indian Ocean to the Bay of Bengal. The former formation roughly coincided with the distribution of laterite deposits. The total number of cases was as follows between September, 1896, and November, 1898:—

On trap and crystalline rocks	150,929
On the Indus-Ganges alluvium	12,936
Grand Total	163,865

Of this grand total the cases which had occurred on the Great Indian alluvium formed only 7·88 per cent. At first sight this appeared very striking, and seemed to point to the trap and crystalline area as being specially adapted to the spread of the disease, but in reality there was a more probable explanation for these figures at hand. A look at the geological map of India would show that Bombay was situated near the centre of the western margin of the great Deccan trap plateau. When the exodus of the scared population began after the outbreak of the plague in 1896, the refugees naturally moved in all directions, and spread themselves fanlike over the adjoining country, and this happening to be all composed of Deccan trap, and beyond it of crystalline rocks, the overwhelming majority of cases on these formations seemed easily explained. It had been shown that so far at least it had not been conclusively proved that soils without the intervention of man spontaneously developed diseases. But, on the other hand, there were abundant evidences that the tenacity with which certain epidemics clung to localities were influenced by the geological formation on which they appeared. In Western India the soil was generally very thin, and rested frequently direct on laterite deposits, which were among the most spongy and generally porous rocks known. In such cases it might well be that the direct sources of epidemic might sink some considerable depth into the underlying rock, but this would have to be proved. He accepted the general medical opinion that geological formations had nothing to do with the distribution of epidemics beyond the different physical characteristics attaching to various formations, some being porous and others less so.—*Indian Medical Gazette.*

Anti-Plague Inoculations.

The following table is extracted from Surgeon-Captain Leumann's report on plague inoculation at Hubli, in which the results indicate a protection of 85 per cent. on the whole epidemic.

Dates. 1898.	Number of non-inoculated in Hubli.	Number of once-inoculated in Hubli.	Deaths from Plague amongst		β Percentages in favour of	
			Non-inoculated.	Once-inoculated.	Non-inoculated.	Once-inoculated.
Five weeks (From May 11 to June 14)	44,573	2,323	47	*Nil.*	...	100 %
Week ending June 21	41,494	3,368	22	2	nearly 17 %	—
,, ,, 28	39,042	4,487	29	1	...	70 %
,, July 5	36,020	5,057	55	3	...	64 ,,
,, ,, 12	33,255	5,974	34	4	...	40 ,,
,, ,, 19	29,716	6,565	82	1	...	96 ,,
,, ,, 26	24,122	9,386	100	6	...	84 ,,
,, Aug. 2	21,031	10,016	140	7	...	90 ,,
,, ,, 9	15,584	11,339	272	5	...	95 ,,
,, ,, 16	10,685	10,265	386	30	...	93 ,,
,, ,, 23	6,367	9,671	371	21	...	96 ,,
,, ,, 30	4,094	7,569	328	8	...	86 ,,
,, Sept. 6	2,731	6,798	227	11	...	99 ,,
,, ,, 13	1,116	6,381	143	7	...	99 ,,
,, ,, 20	937	6,567	106	11	...	99 ,,
,, ,, 27	603	6,820	58	4	...	90 ,,

Another important observation of the effects of anti-plague inoculations is given by Dr. Chenai, Medical Officer of the Southern Marátha Railway, and is summarised by Monsieur Haffkine in the following communication to the Plague Commissioner of Bombay:—

(1) In continuation of my No. 1,288 of to-day, accompanying Captain Leumann's Report on the inoculations at Hubli, I have the honour to forward a Report, with supplements, received from Dr. Chenai, Medical Officer of the Southern

Marátha Railway, who was doing inoculations at Hubli on behalf of that Company (*vide* Mr. Cappel's reference in his notes on the inoculations in Hubli).

(2) The report refers to the observations on the incidence of plague among the inoculated and uninoculated employés of the Company who were living in the Railway area, Hubli, and as far as their domicile and general conditions of life went, were equally exposed to the danger of plague infection.

(3) I have ascertained from Mr. Cappel, then Collector of Dhárwár, that the occupants of the Railway chawls and yard were under close daily inspection throughout, and that the accuracy of the record is beyond dispute, as the chawls and other Railway premises are well apart from the town, and the occupants are all under the immediate control of the Railway authorities. They were inspected daily by English officers of the Railway, working as a Committee, with Dr. Chenai as Medical Officer, and subject to the general control of the Chief Superintendent (who represented the Collector) of the plague operations in the town.

(4) On account of these circumstances, and of the fact that the incidence of plague, as well as the *data* about inoculation or non-inoculation, could thus be ascertained with a perfect degree of accuracy, the results observed on this group of people have a peculiar importance. It will be seen that they correspond closely to the observations made on the Southern Marátha Spinning and Weaving Company's employés, and on the Hubli population in general.

(5) As stated in Dr. Chenai's No. 2,633, dated November 21-22, 1898, during the time under observation a few outsiders were admitted to the Railway yard; and of the number of plague attacks which took place, two were among these outsiders (Nos. 45 and 48). As the persons concerned (both uninoculated) may have contracted the disease outside the Railway yard, their cases are to be excluded from the comparison of attacks in inoculated and uninoculated.

(6) The following is the analysis of the *data* embodied in Dr. Chenai's Report:—

After the introduction of inoculation, plague occurred in the Railway yard on 28 different dates, between June 11 and October 6, 1898, viz., on June 11 and 29, July 5, 9, 11, 13, 14, 19, 20, 21, 22 and 23, August 4, 5, 6, 9, 10, 12, 21, 22, 23, 24, 25, 26 and 30, September 21 and October 1 and 6.

On the first of these dates, June 11, there were in the yard:—1,794 uninoculated—41 inoculated once—and no inoculated twice.

On the second date, June 29, there were:—1,248 uninoculated—235 inoculated once—439 inoculated twice.

On the third date, July 5, there were:—1,063 uninoculated—385 inoculated once—471 inoculated twice; and so on.

The number of inoculated twice was gradually increasing, as the people were coming forward for inoculation, and the number of uninoculated was correspondingly decreasing.

(6) *The inoculated once* were therefore present during 28 dates when plague occurred. Their number was the lowest on the first date (June 11, 1898) (41), and reached the highest on the third (July 5, 1898) (385). Their average daily strength throughout the 28 dates of plague was 271.

The daily strength of the uninoculated decreased from 1,794 (on the first date) to 31 (on the last, October 6, 1898), and was, on the average, throughout the 28 dates, 785.

The incidence of plague in these two groups was as follows:—The 785 uninoculated had 35 cases and 21 deaths. The 271 inoculated once had 5 cases and 1 death.

The percentage of protection in the once inoculatcd is obtained from the following considerations:—

If the inoculated had suffered in the same proportion as the uninoculated, they should have had 12 cases and 7 deaths, instead of which they had 5 and 1 respectively.

The number of cases appeared, thus, reduced in the once inoculated by 58·33 per cent., and the number of deaths by 85·71 per cent.

(7) *The inoculated twice* were present only on 27 dates (there having been no inoculated twice on the first date, June 11), and their daily strength gradually increased from 439 (on the second date of plague, June 29, 1898) to 1,713 (the last), and was, on the average, throughout the 27 dates, 972.

The daily strength of the uninoculated present on those 27 dates decreased from 1,243 (on the second date of plague) to 31 (on the last), and was, on the average, 748.

These 748 uninoculated had, during the period in question, 33 cases and 19 deaths, while the 972 inoculated twice had 6 cases and 1 death.

The percentage of protection in the twice inoculated was, therefore, as follows:—

If the inoculated had suffered in the same proportion as the uninoculated, they should have had 43 cases and 25 deaths; instead of which they had 6 and 1 respectively. The number of cases and deaths appeared, therefore, reduced in the twice-inoculated by 86·05 and 96 per cent. respectively.

(8) As regards the proportion of deaths to attacks, this was:—In the uninoculated—60 per cent.; in the inoculated once—20 per cent.: and in the inoculated twice—16·67 per cent.

(9) This result observed in a group of people where, as mentioned above, the information about every individual could be obtained with perfect exactitude; together with the observations made under similar conditions among the employés of the Southern Mahrátta Spinning and Weaving Company, and with the testimony of the house-to-house enquiry embodied in Lieutenant Keelan's investigation sheets, as appended to Captain Leumann's General Report, come as a weighty and extremely important corroboration of the *data* collected with regard to the Hubli population as a whole.

ITALY.

Against Insolation.

The *Riforma Medica* for December 16 recommends a teaspoonful of the following mixture every quarter of an hour until the complete disappearance of symptoms:—

℞ Solution of trinitrine (1 to 1,000) 20 drops
Water ... ... 4,500 minims
M.

When amelioration begins the doses should be progressively diminished. Tepid compresses of arnica may also be applied to the head.

AFRICA.

At a meeting of the Natal Medical Council, held on January 18, 1899, the following resolution was passed that in view of the nearer approach of Bubonic Plague, the Medical Council recommend the Government—

I. To advise Corporations and Local Authorities to enforce the most stringent sanitary precautions; more especially—

(*a*) The free supply of disinfectants for use in drains, closets, &c.;

(*b*) The universal disposal of slops and urine by pails, instead of their being allowed to flow into drains, or be thrown on the ground;

(*c*) The limewashing periodically of the interior of the houses of coolies and other coloured people, and improving their sanitary condition generally;

(*d*) The avoidance of overcrowding, and the instituting of extra special inspection of the premises of coolies, &c.

II. To advise the Natal Government Railway authorities to take similar measures in connection with their coolie employés along the line, &c.

III. To advise all employers of coolie and native labour to take similar measures.

IV. To obtain a supply of Haffkine's Prophylactic Serum at once.

V. To keep outside the port all vessels from India and East African ports; and to take special precautions against rats gaining the shore.

VI. Not to discontinue the immigration of Indians.

Review.

THE CHEMICAL AND BIOLOGICAL ANALYSIS OF WATER. Part II. By T. H. Pearmain and C. G. Moor, M.A. (Cantab). Members of the Society of Public Analysts. London: Baillière, Tindall & Cox. Price 5s.

We have read with much pleasure this second part of Messrs. Pearmain and Moor's book on the analysis of food and drugs. Part II. deals with the chemical and biological analysis of water, and does so in a very lucid and concise manner. The authors divide their subject into chemical analysis, biological examination and physical examination, assigning to the methods the order of importance of the information to be derived from them. The chemical examination permits a larger number of independent factors to be accurately estimated, together with the detection of variation in composition which, as the authors point out, is the most important sign of danger in waters. But while assigning the chemical method the chief place, they are far from over-estimating its value, and emphasise the fact that although the analyst may prove the presence of polluting organic matter, it is impossible in the present state of our knowledge to state positively that such impurity is injurious or innocuous. In doubtful cases a water should never be judged upon one analysis alone, and although the chemical data obtained are to a great extent an index of the amount of pollution, their value can only be properly assessed by a knowledge of the source of the water supply in question.

The following statement is one with which we thoroughly agree, and one also which we are glad to notice is made by two chemists of such practical experience as the authors. "It cannot be too clearly recognised that the 'analysis' of water which aims at discovering that which is not the adulterant or substance to be avoided, but is a probable accompaniment of it, is broadly distinguished by this fact from other chemical analysis, which determines the substance itself. When this fact is mastered, as by the lay public it rarely is, it becomes evident that such process of examination which can at best weave round the offending substance a chain of circumstantial evidence, requires a vastly larger number of observations for the formation of a sound opinion than does ordinary analysis which is capable of directly catching the offender red-handed. This does not, however, alter the fact that such assistance, if rightly used, is the best safeguard at present known to science. Every analyst to whom the opportunity occurs is bound to bring these facts to the knowledge of his clients, and to dissuade them from the babyish pretence that chemical analysis can attack water in the same way as it attacks other substances. The duty of a sanitary authority responsible for a water supply is to keep it under continuous chemical observation."

The bacteriological method is briefly described, and it is to be noted that the anaerobic cultures are not forgotten, including Klein's method of isolating the bacillus enteriditis sporogenes, the detection of which in sewage-polluted water is considered by him even more important than that of the bacillus colis communis.

The chapter on the microscopical examination of suspended matter in water is particularly good. Unlike most text-books, which simply mention an array of names, the more important algæ, fungi, crustaceæ, infusoria, insecta, protozoa, rolifera, ova of parasites affecting man, also animal and vegetable *débris* likely to be found in water, are figured and described, and their significance pointed out. No wearisome description is given of these, but the chief features are briefly noted and the diagrams are expected to do the rest. There is sufficient material for those interested to readily recognise the special classes of suspended matter when found. We should like to see in any future issue of the work a slight addition which we think would make the diagrams still more useful for practical purposes, and this is a statement of the size of magnification under each illustration. We believe this book will be specially useful to medical men abroad, living in countries where rain-water collected in reservoirs, tanks and ponds, forms the chief water supply, and where an acquaintance with the organisms found in such water, and the recognition of the harmless from the injurious will be particularly valuable. There are in these places, when in doubt, no libraries to refer to, and accordingly a book which renders the subject easy, and at the same time is highly practical, is just what is wanted.

Filters and their examination are dealt with. The Pasteur-Chamberland filter is taken as the standard to which other filters should be compared, which is practically to state in other terms that all the old-fashioned filters are useless. In place of filters, appliances for the sterilisation of water by heat are gradually coming into vogue. The important points that are to be kept in view in a successful apparatus for this purpose are stated to be:—

(1) The temperature to which the water is raised and the time that it is kept at that temperature must suffice for absolute sterilisation. This control should be automatically effected.

(2) The apparatus must be so constructed that any deposit of lime salts can be removed readily.

(3) The apparatus should be so constructed that the gases naturally dissolved in water are not eliminated, and that the sterilised water is delivered within 2° or 3° of the original water, or the water will be flat and unpalatable.

It is stated that the Maiche Steriliser was examined by one of the writers, and the requirements satisfactorily fulfilled.

We have no hesitation in recommending the volume on "Chemical and Biological Analysis of Water" to medical officers abroad as a most valuable book, containing much practical information on the subject, written in a clear and interesting style.

News and Notes.

WE are glad to know that Surgeon-Major Ross has come home looking well after his arduous and successful labours in Calcutta.

We wish the Liverpool School of Medicine the success it so well deserves, and hope the inaugural dinner on April 22 will prove propitious. We congratulate the school authorities on their enterprise in offering Surgeon-Major Ross the lectureship (why not professorship?) on Tropical Medicine.

The donations and subscriptions to the School of Tropical Medicine, London, are coming in satisfactorily. Already goodly sums have been promised, the

latest being a donation of £200 from the King of the Belgians, in his capacity as Sovereign of the Congo Free State. We understand that Belgian medical colonial cadets will be passed through the London School.

Correspondence.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—Referring to the statement that appeared in your issue of December 15, 1898, to the effect that after an aboriginal Australian woman has given birth to a child by a white father it is impossible for her to have a child by one of her own race, I beg to state in your columns that the above view is erroneous.

Several cases have come under my notice in which an aboriginal woman has, after giving birth to a half-caste child, given birth to a child by an aboriginal father.

In one especial instance occurring on the Coburg Peninsula, over 500 miles from civilisation, the white father after the birth of the half-caste child left the district, and the woman returned to her tribe, where she gave birth to a full-blooded aboriginal child; the white man subsequently returned, and the woman in due time gave birth to another half-caste child by the same white father.

This case will destroy any theory of throwing back of colour, as the white man left the district before the second conception occurred and there was no white man within 100 miles at the time of conception.

It would appear better to correct these fallacies, otherwise they may be accepted as facts.

I am, dear Sirs,
Yours truly,
F. GOLDSMITH, M.B., Ch.B.
Protector of Aborigines.

Port Darwin,
Northern Territory of S. Australia.
February 17, 1899.

Communications, Letters, &c., have been received from:—

C.—Dr. Chastellux, Mauritius; Mr. Henry Cayley, Weybridge; Mr. P. T. Carpenter, Brit. Honduras.

G.—Dr. F. Goldsmith, Palmerston.

M.—Dr. D. S. Masani, Harda.

S.—Dr. St. Geo. Gray, St. Lucia; Dr. Strachan, Montreux.

T.—Dr. Wm. Turner, London; Dr. P. Turner, Maidstone.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Medicale.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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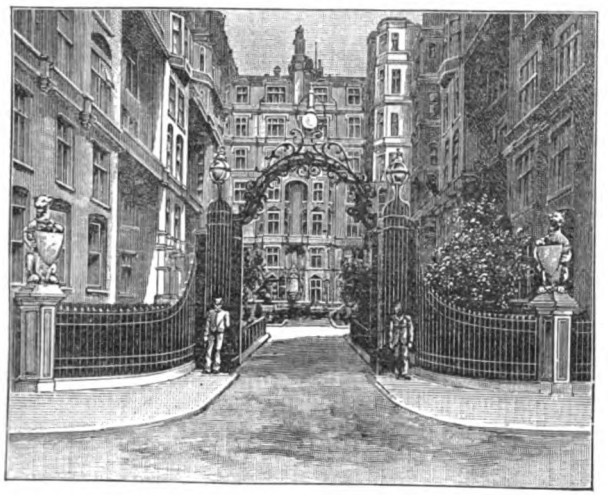

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Original Communications.

A NOTE ON LATHYRISM.

By W. J. Buchanan, B.A., M.B., Dipl. State Med. (Dubl.).
Capt., I.M.S.; Superintendent, Central Jail, Bhagalpur, Bengal.

The accompanying photographs (see plate) illustrate, as far as stance and gait are concerned, the results of poisoning by the prolonged use, as a food, of *Lathyrus sativus*, called in the vernaculars of India *kesàrì* or *teòri*. The patient photographed is one of eleven cases of this disease which I have met with in India during the past year. They all came from areas afflicted by the recent famine (1897). The sleek and healthy condition of the patient here presented does not recall famine conditions, but this is only because he has been over a year well fed in the Central Jail, Bhagalpur.

Lathyrus sativus (nat. ord. *Leguminosæ*) is one of a genus of annual herbs, seven of which are natives of India.[1] It is indigenous all over northern and central India. It is grown wild as a cold-weather crop on land which will raise no other kind of pulse. It is grown largely in the North-west Provinces and Central Provinces of India. It is principally cultivated as a fodder, but being cheap and easily grown, lathyrus is considerably used as a food by the poorer classes and largely so in times of scarcity. The following chemical composition is given by Church ("Food Grains of India"): Water 10·1, albumenoids 31·9, starch and fibre 53·9, oil ·9, and ash 3·2.

This pulse is usually to be met with in bazaars, but the people of the country are well aware of its noxious properties and only use it as a food in times of scarcity, and then only with the hope of being able to soon stop its use. The occasional use of this pulse, or its use along with other food grains, is harmless or attended with symptoms of colic, dyspepsia, &c., only; but if freely employed, especially for long periods, the form of paraplegia known as lathyrism is a very frequent sequel. In olden days, when famines were looked upon as visitations of Providence, to be regretted but scarcely prevented, we heard much more of this disease. Now a-days it is rare, and I have only been able to hear of a few cases during the recent famine in the district of Gaya and Shahabad in Bengal, and in the Central Provinces. Whereas, in 1860, Dr. Irving has recorded that in one Bengal district no less than 4 per cent. of the population suffered from its toxic effects. Colonel Sleeman's account of the disease, as seen by him in Oudh in the famine years 1831 to 1833, is as follows: "In 1833," he writes, "the younger part of the population of the villages of Eastern Oudh, from the age of thirty downwards, began to be deprived of the use of their limbs below the waist by paralytic strokes, in all cases sudden, but in some more severe than in others. About half the youth of these villages of both sexes became affected during 1833 and 1834, and many having lost the use of their limbs entirely are unable to move. . . . The youth of the surrounding villages in which *kesàrì* formed the chief article of food have suffered in an equal degree. . . . no person once attacked has been found to recover the use of the limbs affected. They describe the attack as coming on suddenly, often during sleep and without any warning."

That we have heard so little of this disease of recent years is due to the fact that Government has recognised it as part of "the white man's burden" to "fill full the mouth of famine."

Symptoms of Lathyrism.—The onset of the disease is usually sudden, but it is probable that this is so described because the patients do not recognise that indigestion, colicy pains, and diarrhœa are also due in many cases to the toxic action of this pulse. The muscles of the lower extremities become affected and paraplegia results; the trunk and upper limbs appear to escape entirely. There is said to be a stage of hyperæsthesia. The chronic state is that usually met with. In this the patient presents a characteristic appearance, and in famine-stricken districts they are easily recognised. The gait also is characteristic and is thus described in the article in Allbut's "System of Medicine:" "A peculiar gait, rigidity of the dorsi-lumbar muscles on the side opposite each leg, as it is moved forward, giving a throw of the trunk backwards and sidewards against the weight of the advancing leg. The body is thrown into a series of curves, describing a screw or figure of 8 [*vide* photograph, No. II,] the leg with the toes pointed, and heel drawn up is thrown out in tremulous extension and adduction, the toe reaching the ground before the heel, or the heel does not reach the ground, the gait becoming a tripping on the toes."

An older writer, Dr. Kinlock W. Kirk, of the East India Company's Service (who was killed in the

[1] Watts' "Dictionary of Economic Products," vol. iv., p. 590.

Mutiny in 1857) gives a description to the same effect: "Limbs and joints appear loose, weak, and agitated, and *give* so much at every step that while the person is walking the figure has a constant up-and-down motion . . . the patient walks dragging his toes along the ground."

The two illustrations show this stance and characteristic movement of the limbs.

The skin reflexes are usually lessened, and the knee-jerk and ankle-clonus exaggerated. The patient is usually emaciated, and often takes up the trade of beggar, and hops with the aid of a long stick all over the district. The condition appears to be incurable.

The *Pathology* of this disease has been inferred briefly from the symptoms, which point to implication of the posterior and lateral columns of the cord. Cantarri, of Naples, has described the *post-mortem* appearances in some cases. He appears to have found no affection of the spinal cord, but describes a fatty degeneration of the muscles, especially the adductors of the lower extremities, the transverse striæ being diminished and the ultimate fibres containing little drops of oil.

Ætiology.—That these symptoms of paraplegia will frequently follow the prolonged and excessive use of *Lathyrus sativus* is well known to the people in India, but it is only in times of scarcity that they make much use of this pulse.[2] Its use is forbidden in all Government institutions in India.

Astier is quoted by Watt (*op. cit.*, p. 592) as having found by analysis a volatile liquid alkaloid probably produced by some proteid ferment, which exhibits the toxic effects of the seeds, and which is destroyed by heat. The importance of this discovery lies in the fact that the alkaloid is volatile. It is therefore probably absent when the food is thoroughly cooked.

The toxic effects of this pulse are not confined to the human species. Similar effects have been noted among cattle and horses. In Smith's "Veterinary Hygiene," instances of its acute toxic effects on horses are given when used as food for them even in England.[3] Don also states that swine fattened on this meal lose the use of the hind limbs. It is also probable that many cases of the disease known to Englishmen in India as "*kumree*," or "gone in the loins," a not infrequent disease of horses, are due to feeding on this pulse, which is often used to adulterate gram (*Phaseolus mungo*).

Lieut.-Col. Crombie, I.M.S., has been appointed Lecturer on Tropical Diseases at the Middlesex Hospital.

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[2] In the brief reference to this pulse in Notter and Firth's "Hygiene" (p. 348), *L. sativus* is quaintly called "*dal*," as if this were the English or vernacular name. The word "*dal*" is used by Europeans to denote any pulse used as food, but the term only means "split" (as in the expression "split peas"). *All* the peas and pulses when split are called by natives of India "*dal*," but they more usually call them by the name of the grain, viz., *masur*, *kalai*, *kesdri*, &c.

[3] *Vide* also *Veterinarian*, April, 1885.

THE ETIOLOGY AND TREATMENT OF BLACKWATER FEVER.

By L. W. Sambon, M.D. (Naples).
London.

II.

Is it Quinine Poisoning?

Professor Tomaselli, of Catania, being aware that the clinical features of blackwater fever differed widely from those of the co-endemic malarial diseases, and having observed that the administration of quinine frequently aggravated the blackwater paroxysms or seemed to provoke new relapses, argued that the disease was not malaria *per se*, but a form of quinine poisoning peculiar to malarial cachectics.

Tomaselli's theory was built up in Sicily, where blackwater fever is not very frequent, and was soon widely diffused through Europe and North America, where the disease is still less frequent. Quite recently, Koch gave it very rashly the support of his name, but the English physicians who have had much experience with the disease in inter-tropical Africa are unanimous in protesting against this theory.

Quinine never produces hæmoglobinuria in healthy persons, not even when administered in highly toxic doses. In malaria, it is a specific remedy. Indeed, with the exception of iron in anæmia and of mercury and iodide of potassium in syphilis, we have not amongst the whole ten thousand drugs of the pharmacopœia a more directly beneficial remedy.

Tomaselli and his school first advanced that hæmoglobinuria was produced by quinine in malarial cachectics, but the English physicians in East and West Africa, the French in Madagascar and most American authors in the Southern States, repeatedly observed the disease in people who had suffered only very slightly from malaria and were by no means malarial cachectics. On the other hand, blackwater fever is never seen in the numerous cachectics of most of the intensely malarious regions of the East.

Finding that malarial cachexia could not fully account for the hæmoglobinuric action of quinine, a special congenital idiosyncrasy to the drug was put forward; but then how is it, we would ask, that this peculiar idiosyncrasy is found year after year in almost all those who go to certain districts of inter-tropical Africa yet never in any of those who go to the malarial districts of China, India and Australia? And how is it that those who suffer from hæmoglobinuric fever never show any peculiar idiosyncrasy to quinine either before or after the attack?

The quinine theory of blackwater fever was founded on the assumption that its administration aggravated the hæmoglobinuric paroxysms and provoked new relapses.

In studying the literature of so-called "quinine hæmoglobinuria" we find the following to be the real facts.

In regions in which blackwater fever and malaria are co-endemic, a patient may have suffered repeated attacks of tertian fever or of summer-autumn fever which were satisfactorily treated with quinine. One day, suddenly, instead of an ordinary malarial attack,

blackwater fever manifests itself, and the administration of quinine seems, at times, to aggravate the disease, at other times it appears to have a beneficial influence. The first attack of blackwater fever being well over, the administration of quinine may or may not be followed by a relapse, or the relapse may occur without the administration of quinine.

Later, the hæmoglobinuric infection being apparently spent, the administration of quinine, even in large doses, will never again cause the relapses which it seemed to induce previously.

Thus we see that the connection between quinine and blackwater fever is not one of cause and effect, but merely one of coincidence.

But the fact which cuts this theory at its very root is that blackwater fever breaks out at times in veritable epidemics amongst the natives of inter-tropical Africa, who are supposed to enjoy a certain immunity against malaria, and who obviously do not take quinine.

The Broad-bean Theory.

The theory that blackwater fever is due to a toxic substance contained in broad-beans need hardly be discussed. *Faba vulgaris* is very widely used as an article of food, and it would therefore be difficult to understand how it should give rise to hæmoglobinuric fever only in some few places in Sicily, and, in such places, only to a very small minority of those who feed upon it. Then again, blackwater fever is prevalent in regions in which broad-beans are not cultivated. It is, however, interesting to note that this popular belief arose in districts greatly infected by malarial diseases, because it shows that the natives consider blackwater fever a distinct pathological entity.

Blackwater Fever a Specific Disease.

The peculiar and characteristic symptoms of blackwater fever, its morbid anatomy, geographical distribution and epidemiology are so strikingly marked that it is almost surprising to find that it has not been extricated sooner from the malarial "hold-all."

I believe that F. Plehn, Fisch, Woldert and others did see the parasite of blackwater fever, and justly observed that it differed somewhat from those of ordinary malarial fevers. Their observations concur in describing a small unpigmented hæmatozoon showing active amœboid movements, and assuming mostly an almond or pear shape. Woldert found about one corpuscle in every fifty affected. He noticed that in some cases two hæmatozoa having the same shape occurred within the same red corpuscle, and found that the larger sizes sometimes contained one small dotlet of pigment.[1]

This description corresponds exactly to that of *Pyrosoma bigeminum*, found by Smith and Kilborne in *Texas fever*, which is the hæmoglobinuric fever of cattle.

It is important to note that *Texas fever*, or better, *redwater fever*, is found not only in oxen, but also in horses, sheep, and probably in other species. It is not to be wondered, therefore, that like anthrax, tuberculosis and other diseases that have a wide zoological distribution, it should be found also in man. The duration of the disease, its clinical features and its morbid anatomy in the lower animals, are identical with the hæmoglobinuric fever of man.

The redwater fever of cattle is conveyed by a tick (*Boophilus bovis*), which is not found only on oxen, but infests also other animals (sheep, goats, horses, mules) and man. I have been attacked by ticks myself more than once in Southern Italy while botanising.

Another fact which concurs to prove the identity of the hæmoglobinuric fever of man with that of cattle is that in Sardinia the disease has been observed frequently amongst cow-boys (Silvestrini, Conti and others).

In the Southern United States, in Sardinia, in East Africa and in many tropical and sub-tropical regions, we find hæmoglobinuric fever at the same season both in man and cattle, and we know that in either it may relapse out of season and far from the locality of infection.

But what appears at first inexplicable is that the geographical distribution of the blackwater fever of man, and of the redwater fever of cattle, which is the same in tropical and sub-tropical regions, seems to differ in the higher latitudes. I believe this difference to be only apparent and due to the fact that in Northern Europe and in Northern America the hæmoglobinuric fever of man changes name, and is called *paroxysmal hæmoglobinuria* instead of blackwater fever, and *epidemic* or *infantile hæmoglobinuria*, when it breaks out amongst the infants of a lying-in hospital, as in the cases described by Winckleman.

I know full well that so-called paroxysmal hæmoglobinuria in northern latitudes is far from being as severe and deadly as the hæmoglobinuric fever of Africa, but then we must remember that in Africa the disease usually attacks those who have been already seriously affected by tropical malaria, and again, that the death rate of blackwater fever varies greatly in different regions; thus it is 60 per cent. in British Central Africa (Scott), 49 per cent. on the Gold Coast (Papafio), and 22·4 per cent. in Greece (Spiridon Kanellis). It seems to me that paroxysmal hæmoglobinuria stands to blackwater fever in the same relation as our dysentery, "entero-colitis" of some authors, to tropical dysentery.

Dr. Wheaton proved, some years ago, that the morbid anatomy of paroxysmal hæmoglobinuria was identical with that of blackwater fever. The symptoms also are the same in both diseases; and again, paroxysmal hæmoglobinuria, like blackwater fever, seems to attack chiefly those who have been debili-

[1] In all probability, the parasites of blackwater fever, like those of æstivo-autumnal fevers, are not ordinarily found in the circulating blood, but chiefly in the spleen, liver, and kidneys. In the hæmoglobinuric fever of cattle, large quantities of the specific parasite are found within the capillaries of the internal organs (80 per cent. of red corpuscles infected), while only a few (1, 2 or at most 10 per cent.) can be detected in the peripheral circulation. Then, again, it is almost useless to look for parasites in the peripheral circulation except in the initial stage of each paroxysm, when the temperature rises and the patient experiences feelings of chilliness.

The parasites of blackwater fever, like those of Texas fever, differ from the ordinary malarial parasites in their action on the red corpuscles of the blood, and by the fact that instead of presenting a multiple, rosette-like segmentation they apparently multiply by simple binary fission. In Texas fever no other form of multiplication has been observed.

tated by previous diseases. We usually find it in malarial, syphilitic and tubercular patients.

I do not wish to infer that all cases of hæmoglobinuria are parasitic, because we know that it may be induced by some mineral poisons (chlorate of potash, &c.), by vegetable poisons (some mushrooms), and by animal poisons (snake venom); but I certainly believe that most of the cases called paroxysmal hæmoglobinuria, and vaguely attributed to cold or to nervous and circulatory disorders, are really cases of blackwater fever, and indeed it seems to me that the otherwise inexplicable difference in the latitudinal range of blackwater fever and cattle hæmoglobinuria suggests this to be the fact.

I dare say that, in a disease with so wide a geographical and zoological distribution as hæmoglobinuric fever, we may find several varieties of parasites; but, however this may be, I believe I have brought forward sufficient evidence to prove that blackwater fever is a distinct morbid entity; that it is caused by a specific parasite; that this parasite is an hæmatozoon belonging to the class of protozoa; that clinical, pathological, geographical, and epidemiological reasons, together with somewhat uncertain microscopical data, prove that this protozoon, if not identical with *Pyrosoma bigeminum*, is certainly very closely allied to it.

(*To be continued.*)

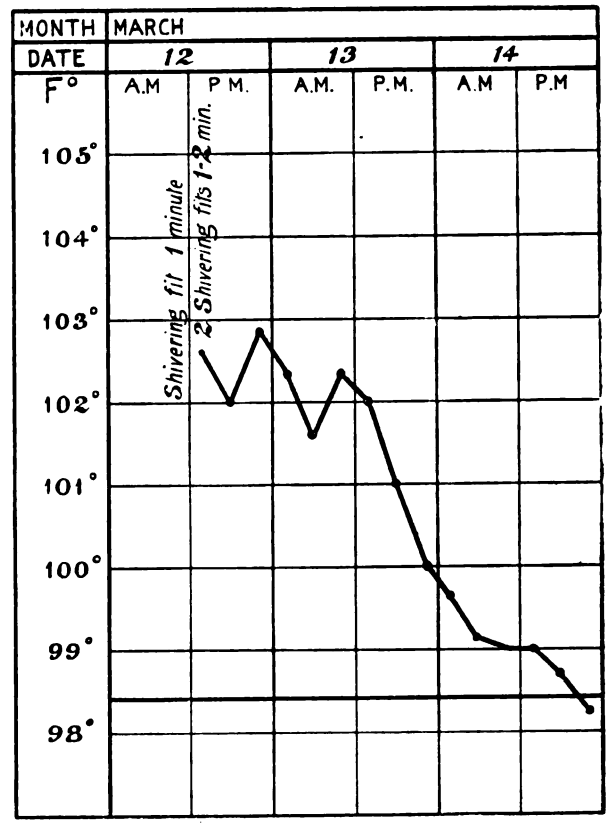
A CASE OF SUEZ CANAL FEVER.

By J. Preston Maxwell, M.B. Lond., F.R.C.S.

F. M. P., aged 33, a shipping clerk, was on his first trip out to the East. He was a strong, healthy young man, with a good family history and no past history of disease of any importance. At Port Said he went on shore and indulged too freely in brandy and soda. He did not drink anything that was not bottled, and returned on board at 6 p.m. As a consequence of his indulgence was very sick during the night. On the following day he was apparently well, ate and slept well. The steamer on which he was travelling passed through the first part of the canal (March 10) and anchored in the morning of March 11 at the lower end of the Bitter Lakes, there being a block in the canal. The patient did not leave the vessel, but was apparently in perfect health.

On March 12 the steamer was still anchored in the Bitter Lakes. He rose in the morning as usual and felt well. At about 12.30 a.m. was suddenly seized with a slight shivering fit lasting about a minute, ate but little lunch, and at 2 p.m. had another shivering fit. By 4 p.m. he felt thoroughly ill, and sent for me. He had not been exposed to the sun during these days, neither had he eaten any bad food.

On arriving at his cabin, the first thing one noticed was that his face was puffy, giving him a bloated and quite unnatural look. The swelling was most marked about the lips and under the eyes. The skin was pungently hot and dry and the conjunctivæ were injected, but there was no nasal or pulmonary catarrh, and no pain anywhere. There was no rash or œdema about the body or extremities, and although his face was flushed, there was nothing like an erysipelatous rash, and the glands were not enlarged. He complained of headache, but no nausea. T. 102·6. P. 112 soft. R. normal. 5 gr. of quinine bisulph. and 5 gr. Dover's powder were given at once, followed in an hour by another 5 gr. of quinine, but the only effect noticed was that sweating began and continued till the end of the illness.



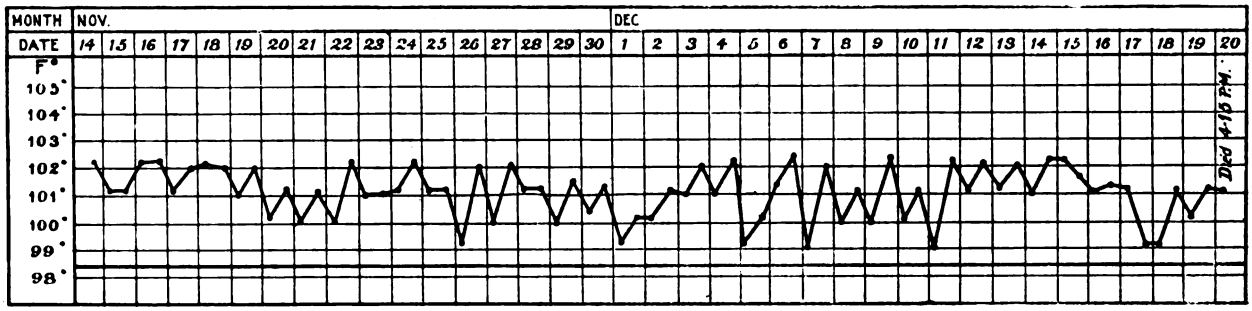
During the night his temperature kept up about the same height, and he slept but little; however, there was no delirium. In the morning of March 13, 5 gr. of antifebrin and 5 gr. quinine bisulph. with a sharp purge were given, but apparently, beyond the effect of the latter, the medicine had no effect. After midday the temperature began to fall and the swelling of the face to subside; but neither did the temperature become subnormal or the face natural till about 12 midnight (March 14, 15.) The following morning (March 15) the patient was apparently quite well, but a little weak. There were no sequelæ.

Comment.—As soon as I was called to this case, I recognised that I had something to deal with that I had never seen before. The swelling and puffiness of the face was more like that of a sharp attack of facial erysipelas, but it certainly was not that disease, and there was no glandular enlargement. Quinine had no effect on it, and I am inclined to think it ran its natural course. I immediately began making inquiries, and learnt that there was an affection called canal fever, known by two on board. The chief officer of the steamer (*Kawachi Maru*), who had made twenty voyages through the canal, had seen it twice, and the pilot had had it once himself. The former, on coming

to see the patient, at once said that it was characteristic, and would run a sixty hours' course. The other officers on board, some of whom had been through the canal many times, had never seen the affection, so that it cannot be a common one.

As to its *ætiology*. In my case it occurred in one who had lowered his resisting power by the abuse of alcohol at Port Said. In both the cases of which the chief officer told me, it occurred in debilitated men who were returning home from the East. And the pilot informed me that it was only acquired by those who were run down, and that he, whenever he felt run down, used to take a dose of quinine as a tonic before going on board a steamer to save himself from getting it.

As to *pathology*. I am absolutely ignorant on this point. While the steamer passed through the canal, a strong wind was blowing from the east bank, carrying sand and in places unpleasant odours from the material which the dredgers, at work in certain situations, were pouring on to the bank, and the patient was constantly on deck.



The *incubation period* appears to be about twenty-four hours. In none of the cases I have quoted did the affection begin till eighteen to twenty-four hours after passing through the canal, and in none did it occur later than thirty-six hours after leaving.

To sum up. The affection appears to be a definite one, having an incubation period of about twenty-four and a duration of sixty hours, and followed by no sequelæ. Its diagnostic points are:—

(*a*) Its onset directly connected with passing through the Suez Canal.

(*b*) The high fever, which starts suddenly with one or two shivering fits and its gradual decline after twenty-four hours, reaching the normal about the sixtieth hour.

(*c*) The extreme puffiness of the face and dryness of the skin.

(*d*) The absence of pulmonary and nasal catarrh, delirium at night, and nausea.

An American Commission for the Study of Tropical Diseases.—The Johns Hopkins University has just sent off a scientific expedition to study tropical diseases. The places visited will include Yokohama, Kobe, Shanghai, Hong-Kong, Singapore, and Columbo, but most of the actual work will be done at Manila.

TWO CASES OF ULCERATION OF SMALL INTESTINE RESULTING IN DEATH FROM PERFORATION.

By W. F. Law, M.D.
Govt. Medical Officer, British Guiana Medical Service.

I. Batusi, a female East Indian coolie, aged 8 years, was admitted into hospital on November 5, 1898, suffering from "fever," which the parents stated had begun about three or four days before.

On admission the child, who was fairly well nourished, was found to be markedly anæmic; temperature was 102·2°, spleen much enlarged, and could be felt fully 2½ inches beyond the costal arch; bowels costive. All the other organs apparently normal; tongue furred. Ordered a pure milk diet. The case was diagnosed as malarial fever. A mild mercurial purge was given, followed by castor oil, and afterwards 3½ grains quinia sulph. was ordered to be given every four hours. In spite of this treatment the pyrexia continued, dropping only 1° or so in the morning and rising again in the evening.

On November 28 there was found to be some cough, and examination of the chest showed a slight congestion of bases of both lungs. The quinine was then discontinued, and a mild expectorant mixture was ordered. The pyrexia continued, and was of an irregular type. On December 12 slight vomiting occurred, and a mixture of soda and bismuth was ordered. At the same time it was noted that some œdema of the feet and ankles had appeared: the urine was tested, but found free from albumen. No change occurred until the afternoon of December 18, when the nurse found the patient complaining of pain in the abdomen. When seen by me on the following morning there was marked distension of the abdomen, and all the signs of general peritonitis. It was evident that from some cause or other perforation had taken place. Opium was given, but the child rapidly sank, and died at 4·15 p.m. the following day. There had been no diarrhœa all through.

Post-mortem examination.—Lungs and heart normal, liver slightly soft but otherwise normal. Spleen enlarged, soft and dark in colour. Kidneys normal. *Abdominal cavity:* A considerable amount of semi-purulent fluid present, and a quantity of recent lymph on intestines, and all the signs of acute peritonitis. On opening the intestines a number of small ulcers were found in jejunum and ileum. These ulcers were small, situated opposite the attachment of the mesentery, and were transverse; they were mostly smooth floored, and their edges showed no raggedness or

hardness, but were in some cases slightly inverted; generally they were shallow, reaching only to circular muscular coat, but in two or three instances they had penetrated to the peritoneal coat, and in one instance, at lower end of ileum, perforation had taken place, through which intestinal contents had escaped. No evidence of tubercular disease could be found.

II. Govocharan, a male East Indian child, aged 6 years, and brother of Batusi, was admitted into hospital on January 23, 1899, said to have been ill with fever for two days.

On admission, temperature 102·2°, decidedly anæmic, liver and spleen both somewhat enlarged, spleen reaching just beyond border of costal arch; other organs normal. Ordered hyd. cum cret. gr. ii., followed by castor oil, and then 1½ gr. sulphate of quinine every four hours. *Diet*, milk.

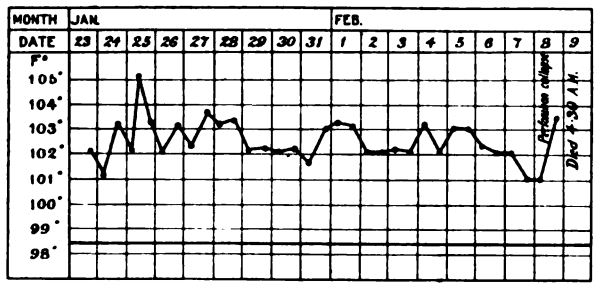
January 25.—Pyrexia has been continuous and severe, and patient complains of pain in abdomen. No diarrhœa. Bowels moved moderately after medicine.

January 27.—Temperature since admission has run mostly from 102° to 103°, but at midday on 25th there was a sudden and sharp rise to 105°. Patient still complains of pain in abdomen, and with the experience of his sister's case, ulceration of intestines is suspected. Ordered to have liq. morph. ♏v. and quin. sulph. gr. 2½ every four hours, and hot fomentations with belladonna and glycerine over abdomen.

January 29.—No change, except that pain seems less, and there has been a slight looseness of bowels since yesterday. No rose spots on abdomen.

February 1.—Temperature continues high, and as patient is drowsy, with a tendency to delirium, morphia and quinine stopped, and a simple diaphoretic mixture substituted.

February 6.—Patient continues the same, delirious at times, and always very irritable. Diarrhœa slightly increased last two days, motions greenish-yellow, and contain a large quantity of undigested milk. Ordered to be fed on peptonised milk only. Spleen and liver are the same as on admission. There has been some cough last two days, but beyond a few bronchial râles nothing abnormal can be discovered in lungs.



February 8, 8 *a.m.*—Temperature a little lower last night and this morning, and spleen is decidedly smaller, but liver is larger, being now fully three inches below border of ribs. Skin is now acting freely, and the motions are fewer and more healthy. At 10 *a.m.* patient suddenly began to groan very much, became very restless, and vomiting set in. Morphia was then given freely, as it was evident that perforation had occurred, but patient sank rapidly, and died at 4.30 on the following morning.

Post-mortem examination.—Heart normal. In right lung a small patch of pneumonia was found at the base in the stage of red hepatisation. The liver was enlarged and softened, but otherwise normal. The spleen was a good deal enlarged, and rather soft and dark in colour. Kidneys were normal. The abdominal cavity contained a large quantity of purulent fluid and some fæcal matter; the intestines were matted together with recent lymph, and the peritoneum deeply congested. On opening the intestines the jejunum and upper part of ileum were found healthy, but in the lower half of ileum numerous ulcers were discovered. Most of these were shallow, and their direction was generally transverse, but some smaller ones were circular. They had mostly penetrated only the mucous and sub-mucous coats, but one low down close to the ileo-cæcal valve had a sloughy appearance, and had penetrated almost to the peritoneum, whilst a little above it another ulcer, which was circular, and had clean-cut edges, had perforated the peritoneum. None of these ulcers had at all the appearance of tubercular ulcers, and their generally transverse direction seems to indicate that they were not those of enteric fever. There was no evidence whatever of tubercle in other parts. The mesenteric glands were slightly enlarged, but no other change could be detected in them.

Remarks.—On admission, the clinical aspect of these cases was entirely that of malarial fever, and they were diagnosed as such. Their subsequent course, however, and their termination led me to suspect that they were really cases of enteric fever, and though the character of the ulceration cannot be considered as typical of that disease, yet to my mind it does not at all exclude it. On the other hand, enteric fever is a rare disease in this country (though a few cases have been recorded), and in this particular district it seems never to have been met with.

THE OCULAR MANIFESTATIONS OF LEPROSY.

By MM. E. Jeanselme and V. Morax.

Translated and abridged, with the kind permission of the authors, from the original paper in the "Annales d'Oculistique,"

By M. T. Yarr, F.R.C.S.I.

The study of the ocular manifestations of leprosy is interesting, not only from the point of view of the pathology of the eye, but also as showing the widely different reactions provoked by the same pathogenic agent. We hope, also, in the course of this paper to exhibit in a clear light the clinical and anatomical analogies between the ocular manifestations of leprosy, syphilis and tubercle: useful deductions may be drawn from a comparison of these diseases as affecting the eye, and in a special degree the study of leprous lesions throws light on the pathology of certain syphilitic lesions and *vice versâ*.

We have had the opportunity of studying ocular leprosy in a certain number of patients (fifteen) at the St. Louis Hospital, and in one case were able to supplement clinical observation by a *post-mortem*

examination. This being evidently too small a number of cases to generalise from, we have supplemented our personal observations by a careful analysis of the literature of the subject.

Eye lesions are extremely frequent in leprosy—a fact noted by every writer on the subject. Boinet[1] found eye troubles in 24 out of 80 lepers examined by him at Hanoi. Lyder Borthen[2] examined 456 lepers in Norway and found the eyes affected in 80 per cent. Tubercular leprosy attacks the eyes much more frequently than the anæsthetic form; it has been estimated that no less than 98 per cent. of tubercular lepers are thus attacked.

For the sake of convenience we have classified the ocular manifestations of leprosy according to the part of the eye attacked—lids, conjunctiva, sclerotic, &c.; at the same time it must be clearly understood that this division is a purely artificial one, as the bacillus never localises itself in one structure of the eye to the exclusion of the others. Of the 15 patients examined by us, 4 presented no lesions of the globe: in these 4, however, the infection dated only from one to two and a-half years before. *Per contra*, all 15 had palpebral lesions, notably nodules in the region of the eye-brow or at the free border of the upper lid.

The Lids.

As regards the palpebral manifestations of leprosy, a very few words will suffice, as they differ little, if at all, from cutaneous manifestations in other parts of the body. In the lids, as elsewhere, we find maculæ and tubercles, which either disappear after a variable period without leaving traces, or terminate in atrophic lesions, fall of hair, &c. The sites of election are the eyebrow and the free border of the lid; it is by no means unusual to find patients suffering from extensive leprosy of the facial skin still preserving a perfectly sound zone between the free border of the lid and the eyebrow in the upper lid, the lower margin of the orbit in the lower.

We have not found ulcers of the lids in any of our cases, and judging from the observations of others they are of rare occurrence. An example of an ulcerative lesion will be found figured in "Boeck and Danielssen's Atlas" [plate x., fig. 4 (a)].

In certain cases little lepromas simulating chalazia make their appearance on the free border. In anæsthetic leprosy atrophy of the orbicularis with lagophthalmos is occasionally seen.

All the palpebral lesions are indolent.

The Conjunctiva.

According to the descriptions of writers on leprosy it would appear that the conjunctiva is as frequently invaded by the bacillus of leprosy as the nasal mucous membrane. As a matter of fact, however, localisation in the conjunctiva is rare, and always secondary to subjacent scleral lesions—a fact conclusively demonstrated by Hansen and Bull. In our own cases we searched in vain for *primary* leprous localisations in the conjunctiva, and were able to convince ourselves that the so-called conjunctival tubercles are nearly always tubercles of the *episclera* elevating the conjunctiva. In one of our patients who presented little peri-corneal tubercles we excised portions of the superjacent conjunctiva, which we submitted to careful histological examination without discovering any leprous lesions in the conjunctival tissue. It is only in the immediate neighbourhood of the limbus that one occasionally finds infiltration of the conjunctiva proper.

Boinet (*op. cit.*) noted in two patients a thickening of the conjunctiva resembling pterygium. In four other cases the same observer found that the conjunctiva was the seat of a vascular injection having the form of a triangle, with the apex on the cornea; probably, however, in these cases the conjunctival lesion was secondary to a corneal or ciliary infiltration.

Babes[3] writes as follows in this connection:—"Amongst the mucous membranes attacked by leprosy the conjunctiva occupies a prominent place. From it the infection spreads towards the sclerotic, the limbus, and the deeper tissues of the eye. . . . More often, however, conjunctival leprosy is secondary; it is seldom absent in pronounced facial leprosy, and the mucus nearly always contains bacilli. . . . In nearly every case of well-marked leprosy, often from the very beginning, the conjunctival sac contains a large number of bacilli. Bacilli are often found here though absent from the nasal mucus, so that one may well believe that the conjunctiva, like the nasal mucous membrane, is one of the ports of entry for the bacillus into the body. . . . I wish to lay stress on the fact that the conjunctiva is, in any case, a centre for the dispersion of the bacilli of leprosy."

The occasional presence of Hansen's bacillus in the conjunctival secretion has long been demonstrated, but there does not seen to have been a methodical search for it in a large number of cases. We have repeatedly sought for it in lepers presenting ocular lesions, but only found it in one case, so find ourselves unable to accept Babes' views on the subject.*

The Sclerotic.

The anterior portion of the sclerotic, extending from the insertion of the recti to the cornea, with the corresponding episclera, appears to be the seat of predilection for the primary ocular localisation of leprosy. In every case where the eye is invaded this zone is affected either by circumscribed infiltrations, tubercles, and little tumours, or by diffuse infiltrations showing clinically as stainings, vascularisations, or modifications of colour. The subjective symptoms accompanying uncomplicated scleral manifestations are slight, and confined mainly to a little photophobia and lachrymation. Pain, spontaneous or induced by pressure, indicates iritis or cyclitis.

In all the published descriptions of cases of ocular leprosy, lesions of this "zone of election" are described. The leprous infiltration is especially marked at the point of penetration of the anterior ciliary vessels: in the case in which we made a *post-mortem* examination, as also in Doutrelepont's and Wolter's case,[4] the vascular lesions were very pronounced at

* *Translator's Note.*—A recent writer, M. Auché ("*La Lèpre en Nouvelle Caledonie*," *Arch. de Med. Navale*, April, 1899), furnishes incidentally an interesting confirmation of this experience. He states:—"I have only found bacilli in the conjunctival sac *twice* in twenty-five cases of ocular leprosy examined, and in both these cases there were ulcerative lesions."—M. T. Y.

this point, numerous bacilli being present both in the tunica media and endothelial cells.

The infiltration in this zone manifests itself mainly in the episclera on the one hand, and in the deeper layers of the sclerotic on the other: save where the vessels pass through the superficial infiltration is separated from the deep by a layer of almost unaffected sclerotic.

The Cornea.

The corneal manifestations of leprosy are of extreme interest on account of their frequency and the deductions as regards general pathology which may be drawn from them. Before proceeding with the subject of leprous keratitis we may note in passing the frequent occurrence of corneal anæsthesia in anæsthetic leprosy. In one case of ours both corneæ were completely anæsthetic, while the cutaneous sensibility of the lids was not in the least impaired.

Leprous corneal lesions may be grouped under two classes, basing the division partly on objective signs, partly on the alterations of the structure of the cornea.

In the first class of cases the infiltration presents the characteristics of a tumour. A swelling appears, either limited to a part of the cornea, or affecting its entire area and conveying at first sight the impression of a neoplasm: in a case described by Meyer and Berger[5] a leprous tumour of this kind was at first diagnosed as a leuco-sarcoma. In another published report the right cornea was covered throughout by a rosy, irregular mass traversed by vessels, encroaching by 1 to 2 mm. on the conjunctiva: the left presented an analagous but more limited lesion. Such tumours on examination are found to be composed of an accumulation of bacilli-containing leper-cells with here and there hæmorrhages.

The second type of corneal manifestation is more common; we met with it in six of our patients and were able to follow its evolution step by step. The pathological process closely resembles that of interstitial keratitis and tubercular keratitis. In a certain area of the cornea, close to the margin and often at a point corresponding to a pre-existing lesion of the sclerotic or limbus, a slight clouding appears beneath the epithelium. Examined with a lens this clouding is seen to be composed of a number of tiny opacities disposed at varying depths in the corneal substance, the superficial and deepest layers being chiefly affected: occasionally more extensive infiltrations in the immediate neighbourhood of the limbus accompany these punctiform opacities. These marginal infiltrations present a superficial resemblance to arcus senilis but differ in being less extensive and in the absence of the transparent corneal zone which separates the opacity from the limbus in the latter. When limited in extent, the disease may subside almost completely, but it is very apt to recur, and invade the entire cornea bit by bit. When the entire cornea has been thus affected, complete return to normal transparency cannot be hoped for and vision is left markedly lessened; blindness even may supervene in course of time from secondary lesions of a glaucomatous nature or the invasion of the fundus by the infection. In two of our cases the opaque cornea bulged out as a staphyloma owing to increased tension.

Lopez[7] in his description of leprous lesions of the cornea lays stress on the resemblance between this leprous keratitis and Hutchinson's keratitis, at the same time shewing the difference in the ultimate result in the two diseases, the former being usually progressive, the latter tending to disappear. The infiltration of the cornea, as in interstitial keratitis, is often accompanied by vascularisation. Sometimes epithelial erosions precede or accompany the keratitis, producing the appearance of little facets on the surface. Such facets are figured in "Boeck and Danielssen's Atlas" (plate xviii., figs. 1, 2 and 3), and we observed them in one of our cases. These losses of substance are not extensive and usually end in cicatrisation.

We have had the opportunity of making a complete microscopical examination of both eyes affected by diffuse keratitis in one of our cases, a leper who died of erysipelas. The globes were removed twenty-four hours after death, fixed with formol and hardened in alcohol; sections coloured by hæmatoxylin were treated with Ziehl's fuschin, 2 p.c. chlorhydrate of anilin and alcohol, dehydrated, washed with xylol and mounted in Canada balsam; others were treated with carmin and Gram's method.

Under a low power the situation of the active foci of the disease could be easily made out owing to the accumulation there of cellular elements and the red points indicating masses of coloured bacilli. The limitation of the leprous infection to the anterior segment of the globe was remarked at once, the ciliary muscle, base of the iris, the corresponding sclera, and the superficial layers of the corneal stroma being mainly affected. Apart from one tiny spot in the retina, no cells or bacilli were found in the posterior segment of the globe.

In the cornea, as we have said, the leprous lesions were chiefly in the superficial layers. Here were found both tiny nodules and more diffuse infiltrations. The nodules were situated immediately beneath Bowman's membrane which was slightly displaced up, the corneal epithelium, however, being quite unaltered. Each nodule was composed of leucocytes of which the central ones contained masses of bacilli, while those at the periphery enclosed only a few or none. A very few similar nodular infiltrations appeared in the deeper layers of the corneal stroma. Here and there bacilli appeared free amongst the layers of the cornea. Approaching the limbus the cellular infiltration became thicker and more confluent, and at the limbus formed a mass separating the epithelium from the cornea proper; here the bacilli were present in enormous numbers. This zone invading the limbus was continued partly as a well-marked episcleral infiltration, partly as a sub-conjunctival infiltration with fewer foci of bacilli; there was no solution of continuity in the conjunctiva. The episcleral infiltration, which increased in volume from behind forwards, began a little behind the insertion of the unaltered recti tendons; the tissue proper of the sclerotic was almost normal save at the points of penetration of the anterior ciliary vessels where infiltrations surrounded the vessels passing through; here baccilli and bacilliferous cells were very numerous.

In the ciliary region the alterations were most pro-

nounced, the deeper layers of the sclerotic, the ciliary muscle, and the base of the iris being completely disorganised by diffuse leprous infiltration, composed of mono- and poly-nucleated leper-cells in every stage of degeneration and stuffed with bacilli. At certain points bacilli appeared to be free amongst the fibres of the ciliary muscle.

In the iris the lesions were relatively discrete and mainly confined to the base; the sphincter iridis was markedly atrophied. One pupil was closed by a fine membrane, attached to the free border of the iris but not adhering to the lens.

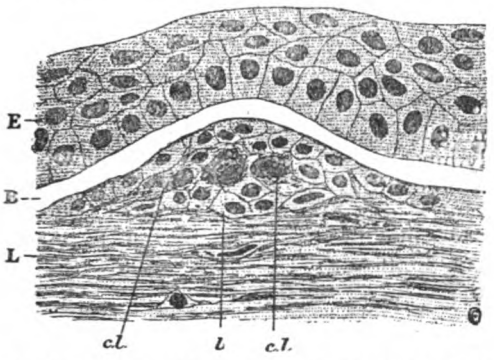


FIG. 1.—SUPERFICIAL NODULAR INFILTRATION OF CORNEA UNDER HIGH POWER ($\frac{1}{12}$ Immers.).
E, Epithelium; B, Bowman's membrane; L, Layers of cornea; *c.l.*, Leper cells filled with bacilli; *l*, Leucocytes. A few free bacilli are also seen between the leucocytes and amongst the layers of the cornea.

We were unable to discover any alterations in the choroid or retina save at one spot, although we examined a large number of sections. Near the end of one of the branches of the arteria centralis retinæ was a tiny nodule intimately connected with the wall of the artery; a few bacilli could at this point be seen in the endothelium of the vessel. The optic nerves were unaltered.

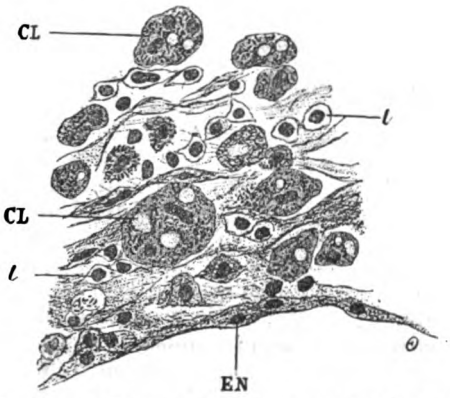


FIG. 2.—LEPER CELLS AT THE ROOT OF THE IRIS, UNDER HIGH POWER ($\frac{1}{12}$ Immers.).
Mono- and poly-nucleated cells, with and without vacuoles, stuffed with bacilli. CL, Leper cells; EN, Endothelium; a leucocytes.

In fine, the histological examination of this case indicated the existence of a chronic inflammatory process, characterised by cellular infiltration without necrosis or manifest hypertrophy practically confined to the *anterior segment* of the globe, and affecting more especially the episclera, cornea, and ciliary body. During life the only evidences of disease were opalescence of the cornea, with episcleral injection.

(*To be concluded.*)

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TRACHOMA AND RACE: A PROBLEM FOR SOLUTION.

By M. T. YARR, F.R.C.S.I.
Major, R.A.M.C.; Member Ophthalmological Society, &c.

OF the many problems connected with granular ophthalmia, the most interesting perhaps is the influence of race in its etiology. Some branches of the human family suffer severely from this terrible disease; others are but slightly affected; one race seems to enjoy almost complete immunity.

Upwards of twenty years ago, Swan Burnett, of Washington, first drew attention to the extraordinary fact that the negro race in the United States is not susceptible to the trachomatous virus,[1] and quite recently the same observer has reiterated and emphasised his belief in this immunity[2]—a belief now shared by most, if not all, ophthalmic surgeons in the United States.

Some of the facts illustrating this immunity are very curious. A large number of men, whites and negroes, were employed in constructing railways in the State of Tennessee; trachoma broke out amongst this body of men, and while the whites (mainly Irish) suffered severely, not a single case occurred amongst the negroes. Again, in the State of Kentucky, more especially in the south and west, trachoma of a virulent type is very common—so much so, that the disease is known as "Kentucky trachoma"; yet the negro population of the State is never attacked. Dr. Ray, of Louisville, in a careful statistical study of the 175 blind pupils of the Kentucky Institute for the Education of the Blind, notes that trachoma is responsible for the blindness of 12 per cent. of the whites, while there is not one case of trachomatous origin amongst the negroes.[3] Instances of this immunity might be multiplied *ad infinitum*. The fact that the aboriginal Indians suffer greatly from granular lids stands out in marked contrast to the foregoing.

Van Millingen, of Constantinople, starting with the theory that "all races are equally susceptible to the trachomatous virus," endeavoured to show that the admitted immunity of the United States negro is not a racial characteristic, but solely the result of higher civilisation and improved sanitary surroundings. He

attributes the susceptibility of the Italians, Poles and Irish in American cities to an imperfect acquaintance with modern sanitary requirements, and the immunity of negroes to the fact that they are "old inhabitants of the country, and enjoy the benefits of better hygiene."[4] The reasoning is a little difficult to follow, and I cannot help thinking this usually acute observer has permitted himself to be carried away by his *parti pris*. His contention, as I understand it, is effectually disproved by an impartial consideration of the facts. It is undoubtedly true that since the abolition of slavery, an insignificant minority of the negro race has made much sanitary progress; but the exact contrary is the case with the great majority, who still crowd together, and live in contravention of almost every law of hygiene. Granting his assumption for the sake of argument, it is difficult to understand how he can reconcile his axiom that "trachoma is an infectious and contagious malady," with the well-ascertained fact that in mixed communities of negroes and whites in the United States the latter may, and do, suffer severely from trachoma, while the former are immune.

I have had the opportunity of discussing this subject with several American *confrères* practising in places where the negro population is large, and have found all unanimous in the belief that—at all events in America—the pure-blooded negro is not subject to trachoma; most of them believed that even the rare cases observed were more probably severe cases of follicular conjunctivitis than cases of trachoma.

It may then safely be accepted as an established fact that the United States negro is not subject to trachoma. That this immunity is a racial characteristic, and not due to any local influences or surroundings, seems also practically certain. One would then naturally assume that the African tribe or tribes, from which the American negroes derive their origin, would display the same characteristic. Here, unfortunately, matters stand on a different basis; reliable evidence from Africa is lacking, and that from other countries (excluding the United States) is scanty and unconvincing. Van Millingen[5] brings forward the following instances in support of his conviction that negroes are only immune *in certain countries*. 1. There are some 4,000 negroes in Constantinople; of these the majority are eunuchs in the harems, and live a lazy and comfortable life; in this class trachoma is unknown. On the other hand, the minority, composed of poor workmen living huddled together, and suffering the extremes of misery and privation, suffer severely, trachoma accounting for no less than 24 per cent. of their ocular affections. 2. Professor Gama Pinto, of Lisbon, states that he has seen "some negroes" in that city suffering from the disease. 3. Dr. Moura, of Rio de Janeiro, informed Professor Gama Pinto that he had seen "many cases."

Of these statements the two latter are so vague as to be practically valueless. The first is definite enough so far as it goes, but it does not go very far. Africa is an immense continent, inhabited by many different races, some of which are intensely susceptible to trachoma. The Constantinople negroes are probably derived from a *couche* in Africa differing widely from that of the United States negroes, and until this and many other points are cleared up it cannot be seriously contended that race has no influence in the etiology of granular ophthalmia.

Such then is the present position of this problem. I am, unfortunately, not competent to say from what race or races in Africa the American negroes are descended, but there must be many who are. Many of our *confrères* in East, West, and Central Africa enjoy unrivalled opportunities of collecting ethnological and statistical facts bearing on this branch of the great problem of Trachoma and Race. May I venture to appeal to them for their collaboration?

Trachoma is still one of the scourges of the human race, and the cause of enormous direct loss to individual States—to none more than to England, with her scattered colonies and dependencies. Anything, therefore, tending to the elucidation of the laws governing the development and spread of this terrible malady must be of importance; each new fact added to our knowledge of disease, however trivial and irrelevant it may appear at the time, eventually and inevitably fits into its place in the mosaic of the complete picture.

In the space at my disposal I have only been able to glance at one aspect of the problem—that of the immunity of once race. The question of the greater or lesser susceptibility of other races is a vast and intricate one, consideration of which must be deferred to a future occasion.

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LEPROSY IN INDIA.[1]

By H. A. Acworth, C.I.E.

(*Continued from page* 238.)

In 1873, Dr. Vandyke Carter wrote a report on "Leprosy, and Leper Asylums in Norway," in which he says:—"In my opinion the decline of leprosy in Europe was mainly due to segregation, harshly and badly carried out as it was, and there is no essential connection between leprosy and bad sanitation. In Norway there has been no such general improvement in the condition of the people as would account for the decline in leprosy. It is due to asylums and segregation." He continues (and the sentiments expressed are so just and noble that the quotation must be allowed), "It is most desirable to remove from the sight of and contact with healthy men, women, and children, the diseased and repulsive leper. . . . Familiar contact with a loathsome malady can have but one effect on young and old, when not accompanied, as in an asylum, by constant efforts to palliate or cure. . . . Passive tolerance of frightful disease is surely the work of blunted sympathies, or worse, of reprehensible indifference, failings in the long run which cannot but re-act on the community. . . .

[1] Read at the Indian Section of the Society of Arts.

Much of the indifference to suffering, which we often wonder at among the Hindus, is due to persistence in the conditions now hinted at. On the broad grounds of social policy they should be dealt with."

The curious point is that when Dr. Vandyke Carter expressed these strong opinions as to the value of segregation he was not a believer in the diffusion of leprosy by contagion. The contradiction between theory and practice in his mind is very striking. As a medical man, he would not go an inch beyond where the evidence led him; but as a practical adviser of Government, he was able to free himself from the trammels of mathematical demonstration. He was the master of logic, not its slave.

At a later date, viz., in 1876, in reporting on "Leprosy in Kattiawar," he wrote as follows:—"Leprosy flourishes, but does not arise among bad hygienic conditions. I am indisposed to admit that leprosy ever arises spontaneously; it can, therefore, only be propagated by contagion, or by hereditary transmission. There is a good deal of negative evidence against contagion. I am disposed to attach more weight to positive evidence . . . . to act upon the presumption most favourable to the interests of society in general . . . ." (He speaks with great caution and reserve.) . . . "Heredity, as the exclusive agent in the propagation of leprosy does not seem to be entitled to the position once allotted to it." (Yet he had been once a powerful advocate of the theory of heredity; his judicial wisdom is exceedingly striking). . . . "In Kattiawar the disease persists in the midst of a general prosperity, and in spite of the improvement of the people. . . . My plan is the complete isolation of the leper, and, if possible, his removal to an asylum. . . . I suggest (*a*) convenient refuges for vagrant lepers who are to be detained in them; (*b*) similar asylums open to the poor who are not vagrants, and which they should be compelled to enter; (*c*) that persons who are willing to provide separate maintenance for their lepers should only be allowed to do so on condition that isolation be efficient."

This is almost the same system as has been adopted in Norway, partly before and partly since Dr. Vandyke Carter made these recommendations. It will be convenient now to turn to Norway, which furnishes the best modern evidence as to the value or otherwise of isolation.

The publications of Dr. G. A. Hansen, the Inspector-General of Leper Asylums (or of Leprosy—the writer is not sure what the exact title is) have already been more than once referred to. He published in October, 1893, a short article in the *Lancet* commenting on the report of the Leprosy Commissioners. In that article he makes nearly the same remark as Vandyke Carter makes when considering the question of contagion, that one bit of positive evidence is worth a thousand negatives. But without dwelling at this point on Dr. Hansen's opinions, it will be better to look for a moment at his facts and figures. The general truth of the observation that figures may be made to prove anything may be admitted, but facts in the shape of figures nevertheless furnish the best quality of evidence when they are studied with the desire, not to manipulate them in favour of a preconceived theory, but to obtain light from them upon the path of truth.

In a monograph on leprosy, published in 1895 (which has been already referred to) by Drs. Hansen and Looft (the latter being assistant physician at a great leper hospital) a tabular statement is included, which shows year by year from 1856 to 1890 the number of lepers in Norway, the number of new cases, the deductions by death, cure, and emigration, the admissions to asylums, the number remaining at home, and finally the total of lepers in Norway at the end of the year. It is believed that the statistics are absolutely accurate. As Dr. Hansen says: "Every leper in Norway is known by name." There were 2,833 lepers in Norway at the end of the year 1856, when asylums were first established, and the system of isolation entered on tentatively and imperfectly. At the end of the year 1890, the lepers of Norway were 954; in the year 1894 Dr. Hansen says they were 700.

Year	Total lepers at beginning of year	New cases	Result					Total at end of year	Proportion in Asylums to total at beginning of year
			Died	Cured	Emigrated	At home	In Asylums		
1856	—	238	—	—	—	2,598	235	2,833	—
1857	2,833	242	293	3	15	2,339	427	2,766	15·07
1858	2,766	210	224	3	3	2,294	475	2,769	17·17
1859	2,769	239	213	8	7	2,267	523	2,790	18·88
1860	2,790	219	251	1	6	2,218	539	2,757	19·31
1861	3,757	219	239	6	14	2,028	711	2,739	25·77
1862	2,739	211	215	5	11	2,009	698	2,707	25·55
1863	2,707	196	192	5	4	1,947	749	2,696	27·66
1864	2,696	201	202	—	8	1,914	781	2,695	28·98
1865	2,695	201	205	5	8	1,910	772	2,682	28·64
1866	2,682	203	214	3	10	1,879	795	2,674	29·64
1867	2,674	200	191	8	4	1,876	787	2,663	29·43
1868	2,663	206	210	6	7	1,865	788	2,653	29·59
1869	2,653	183	199	10	13	1,820	787	2,607	29·62
1870	2,607	187	203	3	13	1,762	764	2,526	29·30
1871	2,526	170	238	2	16	1,681	747	2,428	29·57
1872	2,428	131	205	5	10	1,627	708	2,335	29·16
1873	2,335	129	177	9	17	1,592	672	2,264	28·77
1874	2,264	137	183	6	9	1,566	642	2,209	28·35
1875	2,209	134	203	5	14	1,499	623	2,122	28·20
1876	2,122	115	187	3	6	1,440	613	2,053	28·88
1877	2,053	110	163	3	7	1,372	629	2,001	30·63
1878	2,001	105	149	10	8	1,341	618	1,959	30·88
1879	1,959	88	162	5	10	1,277	602	1,879	30·73
1880	1,879	72	150	7	7	1,178	617	1,795	32·83
1881	1,795	60	164	5	8	1,092	608	1,692	33·87
1882	1,692	66	137	11	7	1,061	553	1,614	32·68
1883	1,614	87	127	9	5	1,022	535	1,557	33·14
1884	1,557	55	140	10	2	944	519	1,463	33·71
1885	1,463	71	146	9	12	855	522	1,377	35·68
1886	1,377	48	135	16	9	748	522	1,270	37·90
1887	1,270	47	111	2	3	704	514	1,218	40·47
1888	1,218	27	99	8	1	631	524	1,156	42·94
1889	1,156	27	86	9	12	551	530	1,081	45·04
1890	1,081	10	122	6	2	447	507	954	45·97

A study of the Table will show that from the time that the system of isolation was resorted to, partial and incomplete as it was, at all events at first, there has been a steady decrease in the number of lepers in Norway, not absolutely year by year, but practically so. There were only two years, 1858 and 1859, in which the number of lepers exceeded—very slightly—the number of the previous year. In 1858 there were three more than in 1857; in 1859, there were 21 more

than in 1858. With these exceptions every year shows a steady decline.

The writer has added a column to this Table, in which he shows for each year the proportion out of the total number of lepers in the country at the beginning of each year, who had been isolated in asylums. It seems to him that this is an useful addition to make. Looking at the absolute figures alone, without taking out their relations to each other, might give an erroneous impression as to the extent to which asylums have been made use of. Thus in

1857	there were	427	lepers	in asylums.
1867	,,	787	,,	,,
1877	,,	629	,,	,,
1887	,,	514	,,	,,
1890	,,	507	,,	,,

One might conclude from this that the value of isolation was less and less felt by the people, and less and less insisted on by the authorities as time went on, but such a view would be quite erroneous, inasmuch as it would leave out of account the diminishing number of lepers in the country. Thus the proportion of lepers isolated in asylums, to the total number in the country at the beginning of each of the above years, was:—

1857	15·07
1867	29·43
1877	30·63
1887	40·47
1890	45·97

A study of the Table will show that from 1857 to 1866 the proportion of lepers in asylums rose rapidly; there was then for some years a pause, and even a slight decline; in 1876, for instance, the proportion was 28·88, against 29·64 in 1866; but there was then a fresh start, and after 1876 the proportion never went back, except very slightly in 1882; the progress was steady and continuous, till, at the end of 1890, it had risen to over 50 per cent. The total lepers in the country were 954, of which 507 were in asylums.

The writer was hardly prepared for such a result when he began to extract the proportions. Among people like Norwegians, camparatively highly educated and civilised, it was to be expected that the value of isolation would be so fully appreciated that it would be largely enforced at home, without any necessity for resort to an asylum. This had evidently been the case to some extent, but not so much so as to check the growing belief in the value of asylums, not only as places of isolation, but, no doubt, also as places of medical relief. Examining the statistics district by district, Drs. Hansen and Looft point out that the decrease in the disease in each district depended on and succeeded to the numbers isolated in asylums. "Where isolation was insufficient or absent, there was little or no decrease, where it was thorough a decrease was invariable." The same thing went on through each quinquennium (they consider the figures for quinquennial periods, which is more satisfactory than taking them year by year, as it reduces the effect of casual causes); as the numbers of lepers remaining in their homes were reduced, so were the "centres of infection" diminished, and the numbers of new cases were continually lessened. To take the same years as have been already quoted, the numbers of new cases were:—

In 1857	245
1867	200
1877	110
1887	47
1897	10

If isolation is of no value what do these facts mean? Dr. Hansen is, of course, a strong contagionist; but the writer has nothing to say on his own account on that point. He will once more, however, quote the views of a great authority against contagion, Dr. Vandyke Carter. He says in his report on Leprosy in Norway:—"There has been no such general improvement in the condition of the people of Norway as would account for the decrease of leprosy. It is due to asylums and segregation . . . . The detention of lepers in asylums leads to the diminution of the disease outside asylums. This can only arise in one manner, by the abstraction along with the leper of some injurious influence appertaining to himself . . Asylums in Norway were first established under the belief that leprosy was hereditary, but whether transmissible by heredity or contagion, asylums are equally useful." This was written in 1873, when the number of lepers in Norway was still over 2,300. Had Dr. Vandyke Carter seen the figures now before us, he would have been even more strongly convinced. During the first 17 years, from 1857-1873, the number of lepers in Norway was reduced by about 500; during the next 17 years it was reduced by more than 1,200.

The Leprosy Commissioners, quoting Dr. W. J. Collins (*Lancet*, May 17, 1890), appear to agree with him that "to attribute the decline of leprosy in Norway to compulsory isolation is entirely erroneous . . . . I met many lepers . . going about their usual vocations." This seems to the writer to be mere trifling. It is not contended by any one that a complete system ef compulsory isolation exists. Had that been the case, probably Dr. Hansen would say that leprosy would have been stamped out years ago, and that it is being eradicated only slowly and gradually, because isolation is only partial. Dr. Collins attributes the decline to the material improvement of the people, which Dr. Vandyke Carter denied. The writer is unable to comprehend how Dr. Collins's theory can be made to square with figures and facts. They seem to him to point conclusively in the other direction. If in 1890 he had had the facts of Norway, and the experience gained in that country before him, instead of knowing nothing about them, the writer would have had a lighter heart in undertaking the responsibility of compulsory segregation in the Matoonga Asylum.

Dr. Hansen says, "Our statistics clearly demonstrate that this result" (decrease in number of lepers) "has been brought about by isolation." The writer is unable to see what other deduction from them is possible.

It appears that in Norway it was vagrant lepers only that in former days were compelled to enter an asylum. But in 1885 a law was passed at the instance of Dr. Hansen, providing that any leper who chose to

live at home should have his own room, bedding, and eating apparatus, and have his clothes separately washed. If he was unable to ensure these precautions (and every leper was known by name, and was under the eye of the physician of the district), he was to be compelled to enter an asylum. The proportion of lepers in asylums rose very rapidly for some years after 1885, no doubt in consequence of this law.

There is one column in the tabular statement relating to Norway which will cause most laymen a good deal of surprise; that is the column headed "cured," which shows that 208 persons have been cured of leprosy between 1856 and 1890. The writer had always supposed the disease to be incurable. Drs. Hansen and Looft say, however, "Patients usually die before the disease has run its course. But in the maculo-anæsthetic form the cure of the leprosy is almost invariably the result. What remains, however, after the cure of leprosy is very different. We have . . . usually only a miserable rudiment of a human being, with more or less paralysed and deformed hands and feet, with unclosable eyes, of which the lower part of the cornea is opaque, and from which the tears run down over his cheeks, and with paralysed facial muscles, unable to close the mouth, so that the saliva constantly dribbles from it. Such cases may, however, live long and reach great ages, if such is an advantage." The leprosy is cured, but it leaves an animated, partially animated, corpse behind it. This awful picture, which is one only of many others equally awful which might be drawn of the results of leprosy, indicates where the justification really lies for speaking of the disease as an "Imperial danger." The phrase was much condemned when first used, as an unjustifiable exaggeration, and in so far as it was meant to convey that leprosy was dangerously on the increase, it was no doubt rightly condemned. But in its intensity and its fearful effects upon the human frame, though not in its wide diffusion, the disease may justly be spoken of as an Imperial danger.

The Governments of Bengal and of India, in legislating on this subject, made no reference to Norway, but appealed to the advice of the Leprosy Commissioners and the example of Bombay in justification of their action. But Norway seems to furnish as strong a case as a legislator need ask for of the value of isolation. And the Commissioners, after accumulating reasons to prove that isolation is of no use, and quoting authorities in support of that view, end by recommending it. It is true they do so in a half-hearted way. They deprecate compulsory, they advise voluntary isolation. But isolation is isolation, whether compulsory or voluntary, and seems to be justifiable only on the ground that the leper, to use Vandyke Carter's words, carries about with him some noxious influence "appertaining to himself." If this is so, the isolation ought to be as complete as it can be made, having regard to all the circumstances which should be considered in fixing a limit to it.

Voluntary isolation in India may be pronounced to be impracticable and useless. Neither intelligent appreciation of its advantages, nor a sense of obligation to society can be expected where education and anything resembling a high standard of civilisation are so meagrely diffused. Nor would it be possible, the area and population of the country being so vast as they are, that the watchful scrutiny of the medical man, which has been found so valuable in Norway, should be generally effective. The only resource, therefore, which is open to Government in India is to make isolation compulsory, if it is to be resorted to at all. This is what has now been done, the application of compulsion being confined to vagrant, or as they are called in the India Act of 1898, as well as the Bengal Act V. of 1895, "pauper lepers."

Both Acts contain definitions of the terms "leper" and "pauper leper." A leper is defined to be a person suffering "from any variety of leprosy, in whom the process of ulceration has commenced." In the draft Bill circulated by the Government of India in 1889, a leper was defined to be a "person certified . . . to be suffering from leprosy." The writer has already remarked that he prefers the latter definition. When the Bengal Bill was sent to him for opinion, he submitted his reasons for preferring it; but his views were not in accordance with those of the medical advisers of the Government of Bengal, and afterwards of the Government of India, and they did not prevail. But he thought then, and continues to think now, that practical inconvenience may be caused by the narrower definition, which confines the term leper to persons in whom the process of ulceration has commenced. It may perhaps be true that the leper does not carry about "any noxious influence appertaining to himself," except when ulcers exist. But the writer's experience in Matoonga teaches him that on admission to an asylum where the patient is properly housed, clothed, fed, and medically treated, ulcers, unless very far advanced, generally close up. They reappear, no doubt, but often not for months. If the leper is discharged as no longer belonging to the statutory class of lepers, as soon as his ulcers are closed up, he will go out to beg again, the ulcers will speedily reopen, he will again become the medium of the "noxious influence" spoken of by Vandyke Carter, and all the trouble and expense of re-arresting him and re-conveying him to the asylum will have to be gone through for a second time—or, indeed, for all the writer knows to the contrary, it may have to be gone through a dozen times. As soon as he is exposed to the unhealthy conditions of a vagrant life the ulcers will reappear. As soon as he comes once again under the palliative influence of the asylum, they will close, and thus he will continually be oscillating between the outside world and the asylum, until the disease is so far advanced as to resist all palliative treatment.

It is true that the definition objected to classes every man as a leper in whom ulceration has "commenced;" and it may be replied that once ulceration has been discovered, a patient is to be regarded as a leper thenceforward for ever; that a subsequent disappearance of the ulcers does not dispose of the fact that they once existed, and that a claim to discharge on that ground could not be entertained.

It is to be observed, however, that the Board of Inspection provided for by the Act possesses the power, and no doubt is expected to exercise the power, of discharging from an asylum every leper who "can be released without hazard or inconvenience to the community." (Section 14, and Form E, Schedule to

Act III. of 1898.) Ex-hypothesi, a leper is not a source of hazard or inconvenience to the community unless the process of ulceration has commenced. How, then, can he be detained if it has ceased?

But supposing the above objection to be invalid on the ground suggested, the writer would still venture to think that to restrict leprosy within the limits expressed by ulceration is inexpedient, for a leper may, and probably will, go free long after the process of ulceration has commenced, because, though known to be a leper, it may not be discovered or known that he is an ulcerated leper. Thus the very danger against which the Act is intended to guard is allowed to have a much wider operation than was at all necessary.

It must be here added that to remove a vagrant leper, in whatever stage of the disease, into a properly constituted asylum such as that at Matoonga, is an act of kindness to him as well as to the community; at least, such is the writer's firm opinion. If the inevitable "interference with liberty" (Army Sanitary Commissioners) amounted to a hardship, when duly weighed against the immense advantages that accompany it, it would be another thing altogether. But the writer is convinced, and he speaks from an intimate experience, that such is not the case. If the anxious and careful regard to the comfort and happiness, material and mental, of the patients, which ought to be paid, and no doubt in all asylums under the Act, will be paid, is maintained, the objects of it will almost without exception in a very few days after admission prefer the life within to the life without. If this be so, the moral objection to making prisoners for life of innocent men (and the case cannot be more strongly stated than in these words) disappears entirely.

The writer may of course be wrong in the view he takes of the possible consequences of the defining section of the Act. Time will show; and he can only regret that he has so few opportunities or possibilities of watching its operation.

Another point in which the Act III. of 1898 (which is practically the same as the Bengal Act V. of 1895) differs from the draft Bill of 1889, is that it contains no provision for the segregation of the sexes.

In 1889, laymen at large no doubt believed in the heredity of leprosy. The writer most certainly did when, in the ensuing year, he segregated the sexes at Matoonga. But he must admit that if he were establishing an asylum now, he would be in a difficulty on this point. He does not in the least believe now in the heredity of leprosy as a "working hypothesis," that is to say, in the hereditary transmission of the disease. But this belief or disbelief does not exhaust the question of sexual segregation, or the reverse. There can be no doubt that if married lepers are allowed to live together, and lepers in an asylum allowed to marry, the amenities of such an institution will be greatly increased. All observation, it is true, seems to show that lepers are not prolific; still some children will be born to them; and if contagion be under any circumstances possible, the close intercourse between parents and children, particularly mothers and infants, will vastly increase the danger of it. It is true that orphanages or children's homes can be instituted, to which the offspring of leper parents interned in asylums may be ultimately conveyed, but it will not be possible to separate the mother and the nursing infant. For some years, probably, the child and the mother must continue to live together in the most intimate association, and the chances are perhaps at least considerable that under these circumstances the child will become a leper, particularly when its other surroundings in a leper asylum are considered. This consideration furnishes an argument, though by no means a conclusive one, in favour of segregating the sexes. The advantages and disadvantages on both sides have to be carefully weighed, and, of course, were carefully weighed by the Government of India before it determined to exclude from the Act of 1898 the provision for sexual isolation which the earlier Bill had contained. All that the writer wishes to point out is that the question is not disposed of by a repudiation of the theory of the hereditary transmission of leprosy.

The Act of 1898 contains a certain number of restrictive provisions which debar a leper from preparing or selling food, drink, or clothing, from bathing or washing in the public wells or tanks, and from using public conveyances. Under the City of Bombay Municipal Act of 1888 a considerable advance had already been made in this direction. Persons suffering from contagious diseases are debarred (Section 38 of that Act) from using public wells, tanks, standpipes, &c.; by Section 428 from using public conveyances, and the Municipal Commissioner, under Section 410, and others, possesses considerable power of control over the sale of articles of food. But the Muncipality will, no doubt, find it to their interest, if they have not already done so, to move the Local Government to supplement the provisions of the existing law by notifications imposing further and more precise restrictions, such as it is empowered to issue under Section 9 of the Lepers' Act.

There is one other point to which the writer would wish to refer. Dr. Hansen's law of 1883, under which lepers not wishing to enter an asylum, are compelled to give guarantees for efficient isolation at home, has been already referred to. This provision makes the law in Norway complete by embracing every class of lepers, the rich and poor alike. Nothing of the sort has yet been attempted in India. It would be very desirable to do it if possible; but is it possible? Independently of the extent of the country and population, the extreme difficulty and delicacy of any inquisitorial law which invades domestic privacy, are very serious obstacles. It is true that the public opinion of the country would be on the side of Government if any such attempt were made. The people of India do believe in the segregation of lepers. Possibly, as time goes on, it may be practicable to make, with extreme care and caution, some advance in this direction, to collect valid statistics as to the existence of leprosy among the rich and influential, and by the aid, more of the doctor than the magistrate, to inculcate, if not enforce, that degree of isolation which the great authorities of Norway deem to be sufficient to combat and resist the danger of contagion. When this has been done, the work of the statesman and legislator will be as complete, very nearly, as they can make it.

LATHYRISM.

Paralysis caused by the consumption of the pulse obtained from *Lathyrus Sativus.*

Some 60 years ago Dr. Buchanan Hamilton drew attention to the injurious consequences resulting from the consumption, for any considerable period, of that form of dâl derived from the Lathyrus sativus. Since then the symptoms described by this astute observer have been frequently confirmed and on all occasions famine and destitution have preceded and accompanied the ailment.

The herb Lathyrus sativus belongs to the natural order Leguminosæ and to the great Vetch family.

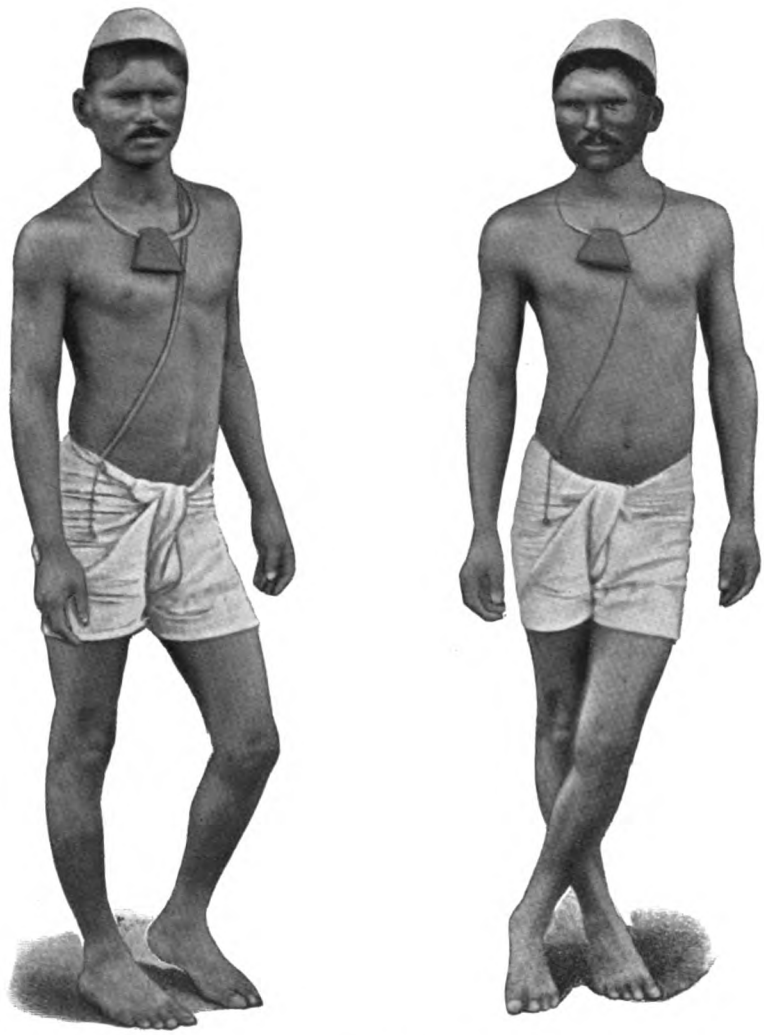
It is one of the plants which contribute to form dâl, a generic term bestowed on all leguminous seeds met with, in some form, in every household and bazaar in India. Of the various forms of dâl the following are those met with in India :—

1. Urhur or Toar (Cytisus cajan) is considered the best of the pulses, but owing to its cost is consumed only by the richer classes. It is pleasant eating, and the natives, even although aware of its bad effects, are unwilling to discontinue its use. The symptoms consequent on its consumption are those of intestinal catarrh and cutaneous affections such as urticaria, bronzing of the surface, dry skin and burning of hands and feet. In course of time the bones of the legs become painful, the periosteum thickens, the moral sense and physical powers deteriorate, and women become barren. The pernicious effect of the consumption of Urhur is most marked when it is consumed without the separation of the skins.
2. Oordh or Marsh Dâl (Dolichos pilosus) is more largely used than any other legume in India, it is the staple article of diet among all classes except the richest and the very poorest. Without the skins it is a wholesome food but when consumed with the skins it induces colic, indigestion, and a dry harsh skin.
3. Moong (Phaseolus Mungo) is the variety of dâl which usually finds its way to the table of Europeans in India. It is a rather tasteless food but is by far the safest form, being light and wholesome.
4. Motth (Phaseolus aconitifolius) is grown in India for the sake of its straw, which is a valuable food for cattle; and its fruit is eaten by only the very poorest of the poor.
5. Chunna or gram (Cicer (or Ervum) Arietinum), and
6. Mussoor (Ervum lens), are not in frequent use as a food.
7. Khesaree, Kesari or Teori (Lathyrus sativus) is the least wholesome of all the pulses met with in the bazaars of India. An excellent description of the paralysis induced by its continued consumption is given at page 261, No. x., vol. i., of this Journal by Capt W. J. Buchanan, I.M.S. It is said that the extent of poverty in a village may be estimated by the amount of the Lathyrus sativus dâl met with in the bazaar. It is only during periods of famine or utter destitution that people take to its use. The natives are quite aware of the evils likely to follow its consumption, but they partake of it rather than starve, either mixing it with more wholesome food, or if such is unattainable it is used as the sole article of diet, in the hope that a short period of its use may not be followed by evil consequences. An interesting reason, as showing the low estimation in which the dâl from Lathyrus sativus is held, is given by Dr. K. W. Kirk: "The villagers said that if they sowed a better kind of grain, it would be plundered by the Beloochees from the hills, but that they would not take this." The plant is not sown, but is left to grow among the wheat and other grains. When the grain crop fails the Lathyrus sativus flourishes and a rich crop of the latter may be gathered with the blighted grain. On this the natives subsist with the result that many become paralysed. At the present day the effects of famine are happily less evident in India than formerly; communication is more rapid, and consequently the paralysis resulting from the consumption of Lathyrus sativus is more rarely seen. General Sleeman, writing in the " thirties," states: "The deleterious effects of eating Lathyrus sativus come on insidiously, gastric irritation, and intestinal catarrh preceding the paralytic symptoms." The poor people, condemned to this form of subsistence, believe that by ceasing to consume the pulse when these symptoms obtain, further effects will not ensue. In this they are sometimes correct, but frequently paresis supervenes, and if the food is persisted in complete paralysis of the lower extremities results. The signs which betray the onset of the paralytic seizure are a difficulty in walking and balancing the body. During progression, the body weight is thrown perpendicularly first on one lower extremity and then on the other causing a waddling, rolling gait; and when the patient attempts to stand still, one foot is planted below the centre of the axis of the trunk, whilst the other is either advanced, retracted, abducted, or crossed over the supporting limb so as to maintain the balance. This condition is admirably exemplified in the accompanying photographs. When the paralysis is more complete the patient can progress only on crutches, the lower extremities being dragged forwards with the toes scraping the ground. In the most advanced stage of the disease the patient can only move about in a sitting posture, the upper extremities being used as a pair of crutches or supports on which the body is swung forward. The paralysis affects the lower extremities only, the trunk and upper limbs remaining unaffected. The signs and symptoms point to an affection of the posterior and lateral columns of the cord, but no anatomical changes in these tracts have been seen. Cantarri has had the opportunity of studying the disease in Italy and states that *post-mortem* appearances were confined to a fatty degeneration of groups of muscles in the lower extremities.

So decided is the evidence of Lathyrus sativus being the cause of this peculiar form of paralysis that it is prohibited as an article of diet in Indian Jails and institutions over which the Government have direct control.

Thorough cooking lessens the pernicious effects of this form of food and it would seem, as stated by Astier, that the toxin contained in Lathyrus sativus is weakened if not destroyed by heat.

No cure is known, and although the general health of victims of poisoning by Lathyrus sativus may be re-established, the paralysis remains.



LATHYRISM.

Photographs illustrating the paralysis resulting from the prolonged use of Lathyrus Sativus as a food in India.
By Capt. W. J. BUCHANAN, I.M.S.

Bale & Danielsson, Ltd., London.

Some years must pass before the candid observer in search of truth will be in any position to judge of the value of the coercive action which has now been applied to Her Majesty's Indian Empire, and it will be for a future generation to discover and appraise its full benefit. The writer has no more doubt, not only that the measures which have been taken are wise and beneficent in themselves, but that they are the sure and certain prelude to the final extirpation of leprosy, than he has of the movements of the planets round the sun. He has already had occasion to remark—and he hopes he may be excused for repeating it, and with some pain—that his personal services in the cause have been as completely forgotten as if they had never been; but he believes that the future historian of India will, in the category of India's debt to England, place these measures for dealing with leprosy in the same rank as those by which Suttee and Thuggee were suppressed. But that historian will never know, for those who could have told him will have passed away, that the wise and humane policy by which the most awful of human diseases was conquered and swept away, was born, its feasibility proved, and its efficacy established, in a little institution known as the Matoonga Asylum.

DISCUSSION.

The CHAIRMAN said they must all have been deeply interested in this paper. It was quite evident that, after mature consideration, the Government of Bengal and the Supreme Government of India had adopted the recommendations which Mr. Acworth was the first to bring into practice, viz., the segregation of lepers. He had thus very powerfully contributed to the solution of a very difficult problem. On the other hand, all who had heard the paper would recognise that there was a very strong opinion in the opposite direction, and he hoped they would have the advantage of hearing from some of the medical men present an expression of their opinion.

Dr. PHINEAS S. ABRAHAM, M.D. (Hon. Sec., Special Committee, National Leprosy Fund), said he had listened with great interest to this paper. He could testify to the fact that when Mr. Acworth started the Matoonga Asylum his work was not ignored, because a notice upon the subject appeared in the *Journal of the Leprosy Committee*, which publication was still looked upon as one of the literary authorities on the subject. It was very curious to notice the apathy in this country, and amongst medical men and government of the British colonies, on the subject. This was illustrated at the Berlin International Conference some time ago, which was initiated by some scientific men in Berlin, who sent invitations to the governments of all the British colonies. The conference was attended by 250 delegates from various parts of the world, but he himself was the only representative of England, India, or the colonies. Very important investigations were then brought forward, and Mr. Acworth's conclusions were in the main borne out by the medical authorities present. It was very satisfactory to have for the first time from Mr. Acworth the full history of the Matoonga institution, about which there had been some confusion, but he could not understand how it was that the authorities had not up to the present time given Mr. Acworth the full credit he deserved. With regard to the etiology of leprosy and the question of contagion, that was hardly the time or place for a discussion. It was a very difficult question, and any one who thought the question of leprosy lay in a nutshell and was easily settled, simply showed that he had not worked much at the subject. He regretted to say they did not yet know how the poison of leprosy entered the system, but the general impression amongst medical men now was that there was a microbe, whose presence could be demonstrated in well-developed leprous tissues. How that microbe gained entrance was not yet settled. Mr. Jonathan Hutchinson would, no doubt, explain his view, which was that it entered the body with food; many believed that that was possible, but his own belief was that the poison might enter in as many ways as the poison of tubercle. They knew by observation and by experiment on animals that the poison of tubercle might gain entrance through the skin, by inoculation through the lungs, and through the stomach, and he could see no *à priori* reason why the leprosy poison might not do the same. But there was this difference between the two, that you could prove these facts with regard to tubercle, but it had not yet been done in the case of leprosy, and in spite of Mr. Acworth's observations they could not say that infection had been proved. Although he (Dr. Abraham) believed in the bacillary theory he was met with certain facts which he could not get over. First of all, experiments had failed to inoculate leprosy, either on animals or on human beings. Professor Profeta inoculated fifteen or twenty medical students and himself with leprosy, and not one of them developed the disease. Similar experiments had been made in Norway by Danielssen, and in some of the islands of the Grecian Archipelago, with the same results. Wherever the attempt at inoculation had been made it had failed, except in one possible instance in Honolulu. There there was a convict going to be hung, who was pardoned on condition that he submitted to inoculation with leprosy, and a year afterwards he developed the disease, which, at first sight, seemed conclusive; but, unfortunately for the theory, it transpired that this man's son, his brother, and other members of his family were lepers, and he came from a district where leprosy was prevalent. Knowing, as they did, how long the incubation period was, the germs of the disease might have been in his system before he was inoculated. Another fact which Mr. Hutchinson laid great stress on was this. There were always a certain number of lepers in England, and before the Berlin Conference he (Dr. Abraham) took a great deal of trouble to get up the facts, and with great difficulty he was able to trace between fifty and sixty cases within the last ten years. Of course, there must have been some others who were not traced, and, altogether, there might probably have been about 100 cases; but there was no reason to suppose that there had been any increase of late years. Ever since there had been so much communication with India and the colonies there had been

cases of leprosy in England, but with one exception —which might be doubted, though he had no doubt about it himself—every case in England had acquired the disease in India, or in one of the colonies, and it had never appeared on any inhabitant of the United Kingdom who had not been abroad and lived in a leprous district. In this one exceptional case, a man came from India with leprosy, and his brother took it, but that was the only case he knew of. If leprosy were as infectious as some people imagined, how was it that it never spread in this country, where no precautions were taken against it? Cases were taken to hospitals and treated and attended by the same doctors and nurses, placed in the same beds, and so on, as the other patients, there being no attempt at isolation. There were cases now in London which he knew personally, many of whom went about just like other people. The public did not know it, or there might be another scare, such as there was some years ago with regard to a case which was mentioned by H.R.H. the Prince of Wales at Marlborough House, in which a leper was seen buying and selling meat in one of the public markets. Mr. Acworth admitted that absolute segregation was impossible, as the slighter cases were difficult of detection, in fact, quite impossible, except by skilled observers. A child might have a spot on the arm, and the leprosy might not become obvious to an ordinary observer for many years, but, nevertheless, that child was a leper. In all leprous countries there were numbers of these cases which were not known; the patients were healthy in other respects, and able to work, and an attempt at compulsory segregation of such persons would not be tolerated. It could be done in a partial way, as was done in Norway, but it was only partial. He was there in 1888, three years after the compulsory clauses were introduced, and saw cases of leprosy walking about the streets. The doors of the asylum were not locked, the inmates could go out, and the doctor in charge of one institution told him they allowed the inmates to go out and walk about, but did not like them to go to churches or crowded places, otherwise they were practically at liberty. With regard to the isolation in their own homes which Dr. Hansen laid stress on, they knew it was impossible to insist on people, who were perfectly free, in such a country as Norway, being strictly isolated in every way. At the same time he admitted that this partial isolation had had a very good effect in calling attention to the subject, and also directly in diminishing the disease; and Mr. Acworth had done great service in showing the possibility of doing this in India. But when they saw the riots which ensued when the Government enforced certain sanitary measures in connection with such a malignant disease as plague, it struck him at once how much more trouble there would be if any stringent measures were attempted with regard to leprosy. At the Cape an attempt was made to send all lepers to Robben Island, and the result was that the people hid their lepers, great difficulties arose, there was a danger of riot, and a few years ago it became quite a political question. The compulsory clauses had now been modified, and lepers were allowed to remain in their own homes, with a certain amount of isolation and notification. It seemed to him that was the proper way of dealing with the question. Of course, those who went about begging and showing their sores should be compulsorily isolated, and the others should be isolated as much as possible. In his opinion the Government of India had been wise in passing their Act of 1896, and, he believed, that this could not have been done had not the possibility of carrying out such measures in India been demonstrated by Mr. Acworth.

Sir STEUART COLVIN BAYLEY, K.C.S.I., C.I.E., here took the chair in place of Lord ONSLOW, who was obliged to leave.

Mr. JONATHAN HUTCHINSON, F.R.C.S., F.R.S., said: —None of us can, I feel sure, fail to give to the author of the important paper which we have just heard, our hearty applause for the humanity of motive and energy of action which it displays. Without doubt, he has set an excellent example and accomplished a very beneficent work. To admit this, and to admit it thankfully and without stint, is still very far from signifying acceptance of his conclusions. Seldom, indeed, has, I think, a better example of counting chickens before they are hatched been afforded, than that given in some of his latter paragraphs. Mr. Acworth feels confident that he has commenced the extermination of leprosy from India, but he has not offered us the slightest proof that he has done anything towards diminishing it. What he aimed at doing, was to remove conspicuous lepers from the streets of Bombay, and this he had accomplished partly by straining a legal enactment beyond its scope, but chiefly by bringing into play the living law of kindness. He built a leper house, and made its inmates comfortable and happy, and thus, as he has repeatedly told us, he attracted to it even more than it could accommodate. If, in addition to this, he fenced it with barbed wire, and employed the police to apprehend vagrant lepers found in the streets, it is yet absurd to call his place compulsory isolation in the sense meant by the sanitarians. His measures were, indeed, directed to the concealment of the leper from sight, rather than extirpation of the disease. For the latter object, the leper who lives at home must, according to the theories of the contagionist, be sought out and forcibly removed from those whom his presence endangers. To take into custody those only who obtrude themselves in the public streets, is to crop off a few of the shoots of a shrub, whilst leaving its roots undisturbed. The "segregation" which Mr. Acworth accomplished was kindly and judicious, the measures at which I have hinted are, if not necessary for the public, cruel in the extreme to the individual. I do not believe that they are necessary, nor that however rigidly carried out, they would exercise the slightest influence upon the prevalence of leprosy. They would leave its cause wholly untouched. In saying this, let me say again that what Mr. Acworth did has my approval; I dissent only from his assertions as to what he thinks he was doing. There is no inconsistency in being an advocate for leper-asylums, and, at the same time, a disbeliever in contagion. I am a Christian Socialist enough to hold that it is the duty of the State to provide for the maintenance and comfort of all who,

by misfortune, are disabled from earning their own living, and I see nothing at all unjust in a community declaring that it will not permit loathsome exhibitions of disease in its streets. In these respects the leper, and the victim of any other disabling malady, should be treated alike. In most parts of Mr. Acworth's paper there is a tone of most judicious moderation, and a very evident desire to avoid exaggerations. This is shown in his statements of the number of lepers in India and in Bombay, and in some of his references to the fallacies of statistics. We may thank him for not having attempted to construct any statistics of his own; and if he has been —as I think he has—misled by those sent us from Norway, it is not a matter for surprise. Permit me to say a few words about these, with the object of showing that they do not prove what they are supposed to do. Leprosy at one time prevailed extensively all over Europe, including the British Isles. In the latter it lingered last in the Shetlands. It disappeared from amongst us by slow degrees, *pari passu* with the advance of agriculture, and it is not more than a century since the last case occurred in the Shetlands. During the period of its decline it attracted but little attention, and all measures of isolation ceased to be observed. Now, when it died out in our northern islands, it still lingered on the opposite coast of Norway, and in Iceland. The parts of Norway in which it persisted were chiefly those on the west coast, with Bergen and Molde for their centres—the homes of the fishing industry, and the homes, in another sense, of a population steeped in poverty, and almost wholly without agricultural land. They had participated but little in the general advance in social comfort made in more favoured regions, and they were still largely dependent upon fish as food. Yet there is reason to believe that for long leprosy has been slowly on the decline in Norway. That it has declined more rapidly in the last fifty years we may thankfully admit. Norway has become a pleasure-ground for tourists, and wealth and comfort have increased, and precisely those conditions have prevailed before which in all other temperate countries professing the Protestant religion, leprosy has always yielded ground. To claim the recent diminution of the disease as being attributable to segregation is to ignore the experience of other countries. Leprosy was common in New Zealand during the early period of our colonisation, but with the advance of agriculture and the diminution of fishing it has disappeared, without any aid whatever from attempts at segregation. As to segregation in Norway, the truth is that it has been of very recent introduction, and only to a very partial extent. The disease was rapidly diminishing before the new law was enacted, and at a time when lepers were frequently to be seen in the streets of Bergen. When the new law was passed, it was not necessary to provide any additional accommodation, for what existed was quite enough. It may be plausibly held that the new law has been mainly coincident with the decline of the malady from other causes, and that to boast of its results is to imitate the fly on the coach-wheel. Mr. Acworth has told us that to him it appears the "merest trifling" to argue that the 300,000,000 of India's population must wait for the extirpation of leprosy, until it is brought about by "the general improvement of physical conditions and sanitary surroundings." Surely it is not necessary to remind him that if such improvement be the one means to that end, he will have to wait. The forces of nature are inexorable, and we shall do well to study them patiently, and not to rush in ignorant haste upon useless expedients. Leprosy has disappeared from a thousand of its old-world haunts, and it has done so under the influence of precisely those changes which we are now told it is the "merest trifling" to wait for. I hope soon to show that even in reference to them it is not necessary to wait inactively, and that there are measures which may be adopted promptly and with great hope of success. Cruel and barbarous in the extreme have been the measures which in the past have been resorted to for the extermination of leprosy. Permit me to record my conviction—a conviction resulting from a lifelong study of the subject—that those measures have never contributed in the least to the end desired, and that that end has meanwhile been slowly brought about by the very influences which are now spoken of so disparagingly.

(To be continued.)

THERAPEUTIC NOTES.

A Mixture for Anorexia.—We find the following in the *Progrès Médical* for March 4:—

℞ Tincture of gentian, Tincture of calumba, Tincture of star-anise, Tincture of nux vomica, } equal parts.

M. Ten drops to be taken before each meal, in a little water.

Treatment of Brittle Nails.—Dr. N. S. Teft (*Medical Brief*, April), in answer to a correspondent's query, says that for many years he has recommended the use of lemon juice. He recommends the keeping of half a lemon on the washstand and directs that every time the person washes he should put each finger into the lemon and use as little soap as possible. The lemon, he says, neutralises the alkali and will restore the nails in a week.

An Application for Excessive Sweating of the Hands. —The *Riforma medica* gives this formula:—

℞ Boric acid		5 parts;
Borax, Salicylic acid,	each	15 ,,
Glycerin, Dilute alcohol,	each	60 ,,

M. To be rubbed on three times a day.

Yellow Fever in Mexico.—According to the report on the last little epidemic at Tampico, it appears that the disease was not imported, because no vessel had arrived from infected ports, and, moreover, the quarantine had been much too strict to allow any sick person coming from elsewhere, to enter the city. It must be concluded, therefore, that the germs of the disease always rest in the ports of Mexico, to break out suddenly at the propitious hour.

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THE

Journal of Tropical Medicine

MAY, 1899.

PROTECTIVE INOCULATION AGAINST TICK FEVER IN NEW SOUTH WALES.

DR. ASHBURTON THOMPSON has, in his capacity as Chief Medical Officer of the Government and President of the Board of Health, presented to the New South Wales Government a report by Dr. Frank Tidswell on protective inoculation against tick fever. The subject is of peculiar interest to New South Wales, owing to the calamitous consequences of tick infestation in Northern Queensland, and its steady southward progress.

The tick fever of Australia is identical with the American Texas fever, which has also been found prevailing in Jamaica, the Argentine Republic, South Africa, Roumania, and Java. The masterly researches of Smith and Kilbourne in 1893 made it clear that the tick is not the essential cause of the fever, but that a micro-organism, the Pyrosoma bigeminum, which lives in and upon the blood of the infected animal, is the real agent. The tick is the means by which the real causal micro-organism gets into the blood of cattle, and also the means by which it eventually gets out again. As is pointed out by Dr. Tidswell, the intervention of two parasites in the causation of tick fever is not without parallel in the realm of disease. Similar symbiotic *rôles* are played by the Tsetze fly and the micro-organism of the African cattle disease, "nagana"; by the mosquito and the micro-organism of malaria; and it is said by the common flea and the micro-organism of plague. In each of these the production of the disease is not an essential attribute of the insect concerned, but a chance circumstance which renders it the conveyer of the pathogenic microbe. Reference is made to the American researches, which demonstrated that cattle can acquire immunity against tick fever, but that this immunity becomes perfect only after repeated attacks, and that the immunity conferred by a single attack, although not always preventing fever, may prevent death from a second attack. The observations showed that it was possible to devise protective measures by exposing healthy animals to a mild attack of the disease, or by inoculating them with the blood of infected animals. The American authorities are inclined to favour the former process as being simpler, and not involving operation, while the Queensland authorities prefer inoculation. The practice of protective inoculation as carried out in Queensland consists of injecting blood from animals who have recovered from the disease, in the hope of obtaining immunity at the expense of a mild attack of tick fever; and it appears that 17,960 cattle were inoculated with a fatality of about 3·6 per cent.; 95 of the inoculated cattle remained unaffected on exposure to ticks, whilst 30 uninoculated cattle all suffered from tick fever. As a matter of practice it has been found that a certain mortality occurs among inoculated animals when they become tick infested. Most of them remain well, but some fall sick, and a few die. This is in accord with the American opinion, that complete immunity is not conferred by a single attack of the disease.

The effects of inoculation on cattle are the same as those of natural tick fever, but very much milder as regards both fatality and illness. The fatality from natural tick fever is between 60 and 70 per cent. The fatality after inocula-

tion is usually between 3 and 5 per cent. The three principal features of the inoculation illness are: fever and its accompaniments, bloodlessness or anæmia, and changes in the urine ("red water"); the illness usually does not last longer than a month, although occasionally an individual beast may remain an invalid for two or even three months. The observations on milk and meat show that the effect on these products is slight. The possibility of introducing by the inoculation other diseases than tick fever is considered, and Dr. Tidswell remarks that although the risk is probably not great, its existence must be admitted if the operation be performed by unlearned persons. The only safeguard is careful preliminary examination by a skilled individual. It is not to be denied that inoculation has its defects. The existing method is crude and uncontrollable in its results, but it is the only one which is known at present. It remains for the future to decide whether or not it can be improved upon. Dr. Tidswell is of opinion that in any case the defects are not such as to contra-indicate its practice; in the mass the balance turns in its favour. Its advantages are a clear gain; its defects such as would be many times magnified by the incidence of tick fever upon uninoculated animals. But the fact that inoculation is not an unmixed blessing must be recognised. The method must not be relied upon for more protection than it can possibly furnish. It is on account of this that Dr. Tidswell has given prominence to these defects, though they are not to be regarded as prohibitive of the method, but indicative of the cost; and he concludes that in view of all the evidence and in the face of threatening tick invasion, protective inoculation is a perfectly justifiable procedure.

Beri-beri in the Brazilian Navy.—The Brazilian cruiser, "Benjamin Constant," arrived lately at Bahia de Rio. During the voyage, twenty sailors died of beri-beri, and this fact gave rise to more rigorous measures in order to combat the disease. Henceforth, vessels will no longer start on a long voyage during the summer season, and ventilation on board will be improved.—*Gaceta Med. de Bahai*, 1898.

THE LONDON SCHOOL OF TROPICAL MEDICINE.

The dinner for the establishment of the London School of Tropical Medicine and the extension of the branch hospital in the Royal Victoria and Albert Docks, took place under most auspicious circumstances under the Presidentship of Mr. Chamberlain. Much of the success which has attended the efforts of the promoters of this scheme has been due to the support of Mr. Chamberlain. It is a most encouraging sign of the times that a statesman of his position should direct a movement of this kind, which has for its objects the prevention and treatment of disease in our great tropical dependencies, and should view it as an obligation laid upon the country to see that every precaution is taken to prevent our splendid administrators being lost, owing to the neglect of precautions which might save their lives or extend their usefulness in the great work of civilisation and government. As pointed out by Mr. Chamberlain, "the greatest enemy was not hostility of savage chiefs, nor the influence of barbarous customs, nor even the physical difficulties of countries in which primeval nature still held sway, but it was rather the insidious attacks of disease, which weakened where it did not kill, and shortened the lives or spoiled the careers of many of the ablest and most energetic of those who represented the empire in these dependencies."

That the scheme has the support of prominent and influential men interested in Greater Britain was evidenced by their presence in large numbers at the dinner, and by the large sums subscribed. The total reached was £16,000 which is an excellent beginning, ensuring success at the early stage of the movement; and we have little doubt that in a little time even larger subscriptions will be forthcoming, which will place the school on a thoroughly sound financial basis, and enable it to fulfil its purposes under the best conditions. Investigation must go hand in hand with teaching if progress is to be made, and both require appliances and money. It is surely not a dream to suppose that the London School of Tropical

Medicine, which will concern itself not only with the teaching of the diseases of the tropics, but with the investigation into their causes and treatment, shall be endowed with at least £100,000, the sum so easily raised for the College at Khartoum.

Article for Discussion.

ALCOHOL IN THE DIETARY OF EUROPEANS IN WARM CLIMATES.

II.

(*Continued.*)

No more common statement is heard than that drink is the chief cause of illness amongst Europeans in a tropical climate. From the West Coast of Africa we are familiar with the report, time after time, that it is drink that causes the high mortality and gives this region so bad a name. No doubt there is both truth and misstatement in these sweeping assertions. On the one hand we have people claiming that alcohol keeps off fever, stimulates the appetite, helps the digestion, neutralises deleterious substances in drinking water, and counteracts the lowering influences of exposure and fatigue. On the other hand we have those who not only detract from its beneficial effects, but declare that the use of alcohol lowers the stamina, renders recovery from illness more precarious, upsets the digestion, and leads to hepatic, renal and gastric derangement.

One must not trust altogether to the reports of medical men in Britain. The returned colonists who seek their advice consist of persons who have suffered more or less from the "climate;" and amongst them, as amongst the ailing in every community, alcohol will be found to have played a prominent part in producing disease. The proportion, however, is by no means higher—if in fact it is so high—amongst tropical residents than amongst the home dwellers. On the other hand, one cannot receive without reservation the reports of rabid teetotalers, who see nothing but evil in alcohol, and ascribe many illnesses to its use which belong either to its abuse, or may have nothing to do with it.

That the daily use of alcohol is a necessity is not to be believed, that its regular use is essential is not true, but that its benefits on occasions are decided there is no gainsaying. Here is a picture of the condition of things in West African mines. "It is almost impossible for people at home to realise the conditions under which men work here, often wet through from standing and working in water, breathing hot, stifling air, and doing the hardest manual labour. When they come up from the mines they have a goodly uphill tramp to reach their quarters, and it is needless to say the men come in greatly exhausted."

The report goes on to say, "a small quantity of whisky is a most useful stimulant at this stage, and there is no doubt it saves many a man from fever." The whisky is usually administered in the form of a "cocktail," containing about half-an ounce of whisky, and the writer (the late Dr. Reynalds, whose obituary is published in this number) regards with favour this ration of alcohol, given when the men are wet and exhausted, and before they sit down to their meals. Dr. Reynalds regarded this imbibition of alcohol as "medicinal" and necessary. From this opinion and practice surely few will be found to dissent. No doubt this is an extreme case, for Europeans are not often found working in mines in the tropics. Unfortunately, men more favourably situated point to these and similar experiences as justifying the use of stimulants in the tropics, but although there is no justification for such conclusions, it is evident that occasions arise when exhausted nature calls for alcohol in a physiological sense.

The sportsman who knows the "tropics" not only shuns alcohol whilst the sun is up, but even refuses to drink fluids of any description. Many, tormented with thirst, put a bullet or pebble in the mouth, or attempt other devices of assuaging their craving for water, rather than indulge their appetite. The same man, however, has a glass or two of champagne if he can afford it, or whisky and soda if he cannot, immediately he reaches home and before having dinner. There is no doubt champagne is the most "reviving" and

least harmful under the circumstances, and after a hard day's walking its "gouty" effects are negligible quantities.

To those living in the tropics a wise rule is to drink little fluid of any kind, and no stimulants, until the setting of the sun. Were this rule followed there is no doubt there would be fewer invalids; but that our brethren in the tropics are to be off-hand denounced as a class who, by drinking to excess, induce most of their ailments, is a perversion of the truth so gross that it will not even admit of discussion.

J. C.

Clinical.

WANTED, A DIAGNOSIS.

THE following is a very good type of a class of case which is frequently seen in Indian hill stations. My reason for publishing it is that it appears to open up some very interesting points for discussion and speculation.

B., a "conductor" in the Commissariat Department, has been twenty-seven consecutive years in India; he has had no serious illness, and is at present a spare, healthy-looking man, organically sound.

He landed in India in 1872, and went to Rawal Pindi. In 1873 he got his first attack of ague at Fort Lahore; during this year he had a good deal of ague, diarrhœa, liver and spleen—to use his own expression.

He had ague again in 1877 at Amritsar, and again in 1889 at Allahabad.

He was sent to Dum Dum Oct. 16, 1896, and, with the exception of six months' field service, remained there till January, 1899.

In September, 1898, he got ague at Dum Dum, which stuck to him till he left for Darjeeling, January 30, 1899.

The attacks were sharp but irregular, never daily, sometimes every other day—more often at irregular intervals of a few days, a week, a fortnight, or longer. He never knew when the attack would come on; the temperature frequently rose to 105° F. He took large quantities of quinine, but never more than twenty grains in one day. Between the attacks he felt in his usual health.

On January 30, the day he left Dum Dum for Darjeeling, he had a severe attack of ague, followed by profuse sweating; took quinine.

He arrived at Darjeeling January 31, and remained free from any sign of ague until February 7. On February 5, he left Darjeeling for Siliguri.

Darjeeling is a Himalayan hill station, at an elevation of 7,700 feet. The climate in January and February is very severe, with frosts, cold winds and damp mists. Siliguri, on the other hand, is at the foot of the mountains, with a mean temperature between 70° and 80° F., and an elevation of about 200 ft. The difference in climate is very great, though only fifty miles separate the two places.

On February 7, B. was about all day in the sun superintending the entraining of mules; at about 3 p.m. he felt a sharp attack of ague coming on; he began to shiver and his hands turned blue. He started to walk six miles to shake it off, and then went to bed about 5 p.m. He took no quinine; that night he passed two loose, very dark-coloured motions, described as nearly black. The next morning at 9.30 a.m. he had another slight shivering fit, and said the fever was on him all day. He returned by train to Darjeeling, and went to bed at 2 p.m.; during the day he vomited occasionally.

I saw him on the morning of the 9th; he had no fever, his tongue was slightly dirty, no appetite, troublesome vomiting; had five motions the previous night, getting lighter in colour; liver and spleen apparently normal; no albumen. I ordered him calomel grains three. After this he had two pale-yellow stools.

On the 10th he had no fever; the motions, two during the night, were nearly white. He felt better, but the vomiting was still troublesome. Ordered a sinapism to the epigastrium, and powders, containing hyd. c. creta gr. i., pulv. rhei grs. ij., sodii bicarb. grs. ii., twice a day.

His condition gradually improved, and the bile returned to the motions. I had him under

observation till February 16, and he had no return of ague. He took no quinine.

Now, was this illness due to malarial poisoning? Let us assume that it was.

According to our present knowledge, ague can only be caused by the presence of a specific organism in the blood, and the attack can only be caused by this organism when it is at a certain stage of development (the sporulating stage). As far as we know, quinine is the only drug which can remove this organism from the blood. It therefore follows that this man must have had sporulating hæmatozoa in his blood on February 7, at Siliguri. As he had constantly suffered from ague ever since the preceding September, and as Dum Dum is a very malarious station, there is every reason for supposing that he had the malarial parasite in his blood for a long time. But how are we to account for the fact that he had no other attack of ague? He remained free, as far as I know, to February 16, and presumably much longer, for I left the station. The man took no quinine. What, then, became of the organism?

There are two curious facts about malaria and hill stations in India, which are well known, but which are not easily explained.

(1) People suffering from persistent and intractable malarial fever in the plains are sent up to the hills, and immediately—sometimes in a day—the fever disappears and does not return.

(2) People who have been free from malarial fever for years go up to the hills and often get an attack there, though endemic malaria may be quite unknown.

Instances will occur to most medical men who have experience of Indian hill stations. They are, however, difficult to explain unless we suppose that the malarial parasite has at the same time the power to clear out at a moment's notice and to remain latent for years.

In the case at present under consideration, I have no evidence beyond the man's own statement that he had an attack of ague on the 7th. He was, however, absolutely positive on the point; and when a man has suffered frequently from ague he is a pretty good judge of its symptoms.

When I saw him on the 9th there was no sign of ague about him; he appeared to be suffering from a simple functional disturbance of the liver. To obviate this, and not a hypothetical malarial poisoning, my treatment was directed, and with good result.

Yet this illness, which so readily yielded to this course, began with a serious explosion, the result of malarial intoxication.

Are we justified in assuming, from the subsequent course of the illness, that the attack this man had at Siliguri was not really ague? Then we must admit that a disordered liver can produce symptoms so closely simulating ague that the patient himself—no mean judge—is quite incapable of discriminating between them; and in the same category we must be prepared to place all those apparently well authenticated cases of ague occurring in healthy localities, after long intervals, and usually attributed to chill, &c.

Perhaps we may also assume that those old Anglo-Indian warriors who frequent Cheltenham and condole with one another over their old fevers, to which they give fanciful names, are suffering from nothing more serious than a disordered digestion.

I regret that I am unable to throw any light upon these questions by blood examinations. Perhaps others who read these lines may be more fortunately situated.

R. H. Moore, M.D., T.C.D.
Darjeeling. *Major R.A.M.C.*

To the Editors of "The Journal of Tropical Medicine."

Sirs,—A few weeks ago I saw a curious specimen of the Guinea worm—*Filaria medinensis*; at least, it was curious to me, as I have never before encountered a similar specimen among the hundreds of cases of this parasite which I have had under observation. The peculiar feature in the worm was that, about two inches from the head, what appeared to be a second body joined the main body at right angles, was nine inches long and was unprovided at its free extremity with a caudal hook.

The patient left the district before the worm was entirely expelled, so that I am unable to say whether the main body of the worm, which was still embedded in the sub-cutaneous tissue, presented any further peculiarity. Perhaps some of your readers could enlighten me as to the significance of the double body.

I am, &c.,

W. M. ELLIOTT,
M.D., F.R.C.S.E.

Kintampo, Northern Territory,
Gold Coast, Feb. 8, 1899.

Recent Literature on Tropical Medicine.

TROPICAL OPHTHALMOLOGY.

MALARIAL EYE AFFECTIONS.—The *Transactions of the British Guiana Branch of the British Medical Association* for 1898, just received, includes a valuable and interesting paper on "Malarial Amaurosis." The author, Dr. Gervaud, of Georgetown, Demerara, states that his attention was first drawn to the visual defects associated with malaria in 1891, when he was in charge of the Nickerie Medical District in Dutch Guiana. He then observed that a certain number of his malarial patients—approximately 4 per cent.—complained of dimness of vision in various degrees; all these cases recovered under anti-malarial treatment, and as he was unable to find any objective signs he did not attach much importance to the symptom, especially as the patients—prisoners and East India immigrants—belonged to a class prone to malingering. Later, however, in Demerara, he saw many cases of defect of vision, varying from transient dimness to almost complete blindness, in gold diggers, farmers, and country people suffering from malaria, and became convinced that the phenomenon was a true symptom of malarial infection. On examining the cases more carefully a "slight opacity of the transparent media" was found in many, but no other objective sign; Dr. Gervaud, however, expresses his belief that fine lesions of the fundus or media might have been discovered by a specialist. Details of the exact amount of defect of vision are wanting; the pupils re-acted normally and there was no conjunctivitis. The author concludes by discussing the differential diagnosis of malarial amaurosis with special reference to hysteria, quinine amaurosis, and night-blindness, incidentally alluding to the prevalence of the last-named disease in British Guiana.

From Dr. Gervaud's description it seems probable that a large proportion of these cases belonged to the class of congestion of the ocular membranes which I ventured to style "malarial retino-choroiditis" (*vide* paper on "Malarial Eye Affections" read at the last annual meeting of the British Medical Association). A small percentage of such cases, forming however an appreciable aggregate, end in capillary atrophy of the choroid and serious defect of vision. It has been shown by Poncet, and quite recently by Guarnieri, that this condition is brought about directly by the circulation of the plasmodia in the choroidal and retinal vessels.

The thanks of all interested in tropical ophthalmology are due to Dr. Gervaud for his thoughtful and suggestive paper, and further contributions from his pen on this little-worked subject will be eagerly looked for.

EPITHELIAL XEROSIS. — Dr. Alessandro (*Archivio d'Ottalmologia*) publishes details of a case of epithelial xerosis; the patient, as is almost invariably the case, presented all the signs of extreme malnutrition. The bacillus was present in abundance, but the author's endeavours to inoculate animals with pure cultures invariably yielded negative results; nevertheless he still believes this micro-organism to be the *causa causans* of the disease, and attributes his inoculation failures to absence of necessary conditions of receptivity in the animals experimented on. Indican was found in the urine, and this, in the author's opinion, furnishes a strong argument in favour of the disease being due to general rather than local causes, indicating as it does the putrefaction of albuminoid substances in the intestine.

M. T. YARR.

ITALY.

AN ANGLO-AMERICAN NURSING HOME FOR ROME.

No one who has stayed in Rome for any length of time can require to be assured of the urgent need of a nursing home for English-speaking persons in that city. In the event of visitors falling ill at their hotel apartments it is difficult to obtain, under present conditions, the attendance of a skilled nurse, and accommodation at a suitable home is out of the question. The efforts which are now being made to supply a much recognised want therefore merit the warmest support. An influential committee, with the British Ambassador as President, Sir George Bonham as Vice-President, including among the members the Bishops of Gibraltar and Southwark, Dr. Wilkinson, Lady Vivian, Miss Brabazon, and Mrs. R. Crawshay, has been formed; and it is proposed to hire a detached house, which will be comfortably furnished. Patients of every denomination will be received, and attended by their own doctors and spiritual advisers; a staff of trained nurses will be maintained, and the latter will be available, if needed, to attend patients outside. We are glad to notice that there will be several free beds in the home, for patients who may be unable to contribute anything to their own maintenance. The charges for attendance to others will be on a sliding scale. There is no reason why the institution should not become self-supporting in a short time; but at the outset some financial assistance is needed, and contributions for the Treasurer, Mr. E. F. Ede, can be paid in London through Messrs. Coutts and Co., 59, Strand.—*The Hospital*, April 22.

AMERICA.

DISINFECTION IN CUBA.

Circular No. 12, dated Havana, February 20, 1899, embodies the recommendations of a Board of medical officers convened for the purpose of devising "some uniform system of disinfection of grounds, buildings and appurtenances of all buildings and sites formerly occupied by Spanish troops before their being assigned to United States troops for habitation."

The recommendations of this Board were that walls and ceilings, when painted, should be washed with bichloride solution and repainted; when kalsomined, to be washed thoroughly with milk of lime and rekalsomined. Window-frames, doors and other woodwork are to be carefully examined and any rotten material removed and burned. The sound parts are to be scrubbed with lye and hot water, washed with bichloride solution and repainted. All floors are to be taken up, including joists on lower storey, and replaced by tiles or tongued and grooved lumber, all decayed wood to be removed and all joists to be washed with milk of lime and repainted.

If on the ground the earth should be removed for a depth of about twelve inches, and the space filled with concrete and paved with tiles, cement or asphalt. The new floor is to be above the ground level. Tile or cement floors, if in

good condition, are to be thoroughly scrubbed with soap, lye and hot water, and washed with bichloride solution. Earth floors are to be excavated one foot, filled with concrete, and paved with tiles, cement or asphalt.

Additional doors and windows are to be cut wherever necessary.

Cesspools, if external, and their use be necessary, are to be emptied with the odourless excavator, thoroughly cleaned, have their walls cemented and be thoroughly sprayed with milk of lime. Such as are to be abandoned, if full, are to be emptied, filled with earth and paved over with cement or asphalt. If the contents are more than four feet from the surface and are sufficiently solid, they may remain, provided they are covered with a layer of lime six inches in depth, filled with dry earth and paved with cement and asphalt. Internal cesspools are to be emptied, sprayed with milk of lime, filled and sealed in the above manner.

All weeds, high grass, dead vegetation, thick shrubbery or banana plants are to be removed and burned. All marshy spots are to be thoroughly drained and the surface of the ground thoroughly policed. No flower or vegetable beds are to be permitted near the barracks. All wooden out-houses, palm huts, &c., are to be burned. Cisterns are to be pumped out, cleaned by scrubbing and cemented where necessary. All articles of furniture are to be destroyed or submitted to steam disinfection. It was recommended by the chief surgeon, in an endorsement in this report, that no building be occupied by troops until the above plan of disinfection had been thoroughly carried out.—*Boston Medical and Surgical Journal*, April 6, 1899.

AFRICA.

Medical Reminiscences of South-East Africa.

Dr. Helkenberg gives an interesting account of his medical and social experience as surgeon to the cosmopolitan gangs employed on the Transvaal-Delagoa railway (*Munch. Med. Wochenschrift*, Nos. 2-4). Space only permits an abstract of the former, which consisted chiefly in the prevention and treatment of malaria. The author considers quinine a specific for both these purposes, if taken in large enough doses. He himself took 1 gram in tabloid form every evening for four seasons, and remained healthy in spite of his exhausting duties. In treating severe cases he commenced with a dose of antifebryn or injected the dihydrobromide of quinine hypodermically. The tabloids were at first despised by patients unless dissolved so as to form a "strong medicine." At last, however, they conceived the happy idea of explaining their undoubted effect by supposing them to be "patent," like the innumerable drugs sent out from England and America warranted to cure anything, and when in 1893 Messrs. Burroughs and Wellcome's tabloids were to be obtained at all "stores" along the line the case was clear, tabloids must be "genuine patent." With regard to alcohol, only heavy British ales and stout were obtainable, but Dr. Helkenberg considers this rather an advantage, for the men will drink, and they get less alcohol on the whole from a moderate amount of the stronger ales than they would by swilling lager-beer all day.

Janus.

INDIA.

The Saline Treatment of Dysentery.

W. J. Buchanan, B.A., M.B., Captain I.M.S., gives in the *Indian Medical Gazette*, December, 1898, a paper on this subject.

The use of salines in dysentery is common in India, though little has been written on the subject; and outside India their value has only recently been recognised. They have been used by French colonial surgeons ever since the days of Bretonneau. The writer gives notes of a great number of cases showing the rapid and beneficial action of magnesium sulphate in acute dysentery. In mild cases many other drugs act admirably, for example, castor oil emulsion, but it does not cure so rapidly. Cinnamon acts fairly well in mild, but slowly in acute cases. A mixture of cannabus indica and perchloride of mercury is useless in acute attacks or in acute exacerbations of chronic attacks, but of value in the frothing fermenting stools of chronic cases, though magnesium sulphate is preferable. Ipecacuanha in mild cases is not necessary, in chronic cases it is of doubtful value and safety, but in sthenic cases it acts exceptionally well. In fact, it is the only drug beside magnesium sulphate which will produce a satisfactory change in the stools in twenty-four hours. A patient will be passing dozens of "meat-washing" stools with pain, griping, and tenesmus, yet on giving magnesium sulphate in twenty-four or thirty-six hours the stools will be free from inflammatory products and be passed with ease and comfort.

The following mixture is very suitable for hospital cases:—

℞ Magnesii sulph....	...	...	...	℥ ii.
Acid. sulph. dil.	...	...	...	♏ iii.
Tr. Zingib.	...	...	...	♏ iii.
Aquæ ad	...	...	...	℥ viii.

For private practice a more palatable mixture may be made by using aromatic sulphuric acid and aqua cinnamoni. In acute cases the best method is to give one or two drachms of this mixture every hour or every two hours, but if this is inconvenient, an ounce may be given twice a day or half an ounce four times a day. Free gentle purgation must be assured. The drug should be continued for one or two days after mucus and blood have disappeared from the stools. It may then be reduced. When the stools become thin and watery it should be stopped. The effect of the drug must be watched by examining the stools daily or oftener. In chronic relapsing cases the action is not so prompt. But if all cases were thus treated from the first the chronic form would be less frequent. Diet is of the greatest importance. The writer gives boiled milk (1 pint), sago (8 ozs.), and soup made from goat's flesh or mutton broth. This low diet is rigorously enforced until the stools become solid.—*The Medical and Surgical Review of Reviews*, April, 1899.

FRANCE.

Dangerous Medicines.

P. Lucas-Championnière (*Jour. de Méd. et Chirug.*, March 10, p. 161) notices some important points in the chapter "Médicaments Dangereux," in Prof. Brouardel's remarkable work, *L'Exercice de la Médicine et le Charlatanisme*. Idiosyncrasy to ordinary drugs is a source of catastrophe.

Mercury.—A physician treated a syphilitic woman with calomel hypodermically, who had undergone the same treatment previously at the hands of a colleague. Finding the dose too feeble he gradually increased it. After an injection of a grain and a-half the patient died. Neither of the practitioners were aware that the woman had albuminuria and marked renal disease.

See also "Death from Mercurial Injection," p. 284.

Aconite is, perhaps, the most dangerous of all drugs. M. Brouardel quotes several cases where its use has been fatal and concludes with these words: "General rule, never use aconitine; if perchance you are obliged to do so, use it in solution and prescribe at most a milligramme ($\frac{1}{65}$ grain) daily in doses of quarter of a milligramme ($\frac{1}{1617}$ grain) every three or four hours. Further, watch the patient carefully so as to be able to interfere on the least sign of intolerance. With a solution one knows what one orders; not so with granules. Analysis of granules obtained from a reputable house showed that some contained a quarter of a milligramme, others none, others half or one milligramme. Hypodermic injections should never be given."

Atropine subcutaneously has frequently proved fatal, but several of the patients had advanced visceral disease from

alcohol. It is best to abandon this method. As to injections of morphine and atropine combined, under the pretext that the morphine is an andidote to the atropine, M. Brouardel believes, on the contrary, that there is a double source of danger.

Morphine subcutaneously is dangerous. Accidents are rare in hospitals, M. Brouardel thinks, because the injections are given with the patients in bed; they are more common in private practice where the patients are often about. In a case of intense neuralgia a man was given a hypodermic injection of morphine whilst sitting in the consulting room of a physician. Syncope occurred and he was unable to leave for two or three hours. A woman died immediately after an injection given similarly in a consulting room. "A short time ago two patients—a man and a woman—on the same day, in the same locality, near Paris, died suddenly after the injection of $\frac{1}{100}$ gramme ($\frac{2}{3}$ grain) of morphine, ordered by different physicians. The solutions used were absolutely normal, containing $\frac{1}{100}$ gramme of morphine per cc., and had been made by different chemists. Moreover, these physicians had used the same solution previously without accident. What is the explanation? It has been supposed that the needle pierced a vein and that the morphine entered the circulation directly. Never give injections in your consulting room. Let the patient be lying down. If there is albuminuria or cardiac disease abstain from injection."

As to danger of injecting morphine into a vein, we may refer to a case published by J. C. Balfour (*Lancet*, November 11, 1888). A lady received for eight months injections of pretty large doses of tartrate of morphia, which had little effect. One day a smaller dose than usual ($\frac{1}{3}$ grain) was injected into the forearm. Immediately she called out that there was something wrong, as she felt a burning pricking sensation all over. She became unconscious. The lips were blue, the face was swollen, and the skin was grey. The pulse was weak and fluttering, and the breathing stertorous. A convulsion occurred. She regained consciousness, but was much prostrated and suffered from headache for several days.—*The Medical and Surgical Review of Reviews*, April, 1899.

Malaria and Mosquitoes. By A. Laveran.

Member of the Academy of Medicine of Paris.

A great many observations testify in favour of the part that mosquitoes play in malaria fever. I have previously referred to this. It remains for me to examine the results of experiments.

Major Ross, of the I.M.S., made the first experimental researches on the *rôle* that mosquitoes play in malarious infection. Major Ross at first studied the evolution of the hæmatozoon of malaria in mosquitoes by causing the mosquitoes to bite the patients in whose blood he had found the malarial microbe, and by observing the modifications which took place in those microbes in the body of the mosquitoes. He also studied the evolution in the mosquito of the endoglobular hæmatozoa of birds which have been described by Da'nilewsky, and which are known under the name of proteosoma. The conditions of the experiment are easier here than with the hæmatozoon of malaria, and the analogies are so great between the proteosoma and the malarial microbe that one can admit *a priori* that the evolution of the two parasites is almost the same.

Reversing the chronological order of these researches, I shall study at first the results to which Ross has arrived in experimenting upon birds. These experiments have indeed given some results more complete and more certain than those made in patients attacked with malaria.

The plan employed by Ross for the study of the development of the hæmatozoa of birds in the mosquito is very simple. The cage in which are put the birds infected with proteosoma is placed under a mosquito net with a vase of water containing the larva of the mosquitoes. The mosquitoes develop, and bite the birds during the night and morning, and it is easy to see by their distended stomachs that the mosquitoes are gorged with blood. They are then collected and placed in a tube, which is immediately closed with cotton wool.

In proceeding Ross has observed that the mosquitoes nourished with the blood of infected birds of proteosoma have, in the lining of the stomach, the pigmented elements which are always absent in the mosquito nourished by healthy birds. These pigmented elements, which are at first very small, increase rapidly; their diameter, which is only 8 μ thirty hours after the absorption of blood, attains a greater size on the third day, 16 to 30 μ the fourth day, 40 μ the sixteenth day, 60 μ more or longer. At the same time as their bodies increase in size they lose their pigment; the fifth day the pigmented grains have in general disappeared completely. It is very important to note that the pigment has all the character of the malaria pigment and that of the proteosoma.

The rapidity of the evolution of these elements varies with the temperature, from six days in the best conditions of temperature. The duration of this evolution can be continued by immediately cooling the atmosphere to two weeks and more, which is important knowledge for those who wish to verify the results obtained by Ross.

The spherical bodies which existed often in great numbers in the lining of the stomach of the mosquitoes nourished upon the birds infected with proteosoma are coloured well with most of the colour reagents; in the young elements, the central portion does not colour so well as the peripheral portion where the pigment is found; in the central part the existence of chromatine granulations is observed. Arrived at their complete development the elements project from the external surface of the stomach of the mosquito, and finally fall into the general cavity of the insect.

These spherical bodies give birth, according to Ross, to two kinds of germs:

(1) Small filiform elements of 12 to 16 μ in length, or germinal threads.

(2) Larger elements than the preceding of a dark brown colour, cylindrical, with rounded extremities and a variable curve closely enveloped. Ross gives these last elements, which are much less numerous than the first, the name of black spores.

The germinal threads at liberty in the abdominal cavity scatter themselves about in the infected bodies of the mosquitoes. They are found notably in great numbers in the salivary glands, which explains the process by which healthy birds can be inoculated by the mosquitoes, and also the manner of the propagation of the disease. The experiments made by Ross leave us no doubt as to the *rôle* of the germinal threads. At the end of the month of June, 1898, four sparrows and a weaver, the blood of which had been found to be free of parasites, were submitted to a number of mosquito bites, nourished more than eight days previously upon a sparrow infected by proteosoma. The five birds, examined on July 9, had hæmatozoa in great numbers. They died soon after, and the liver and spleen were found over-charged with pigment. This remarkable experiment was repeated several times with success by Ross.

Some 28 healthy larks were found infected five and eight days after having been exposed to the bites of mosquitoes nourished on unhealthy birds.

Ross succeeded in infecting some larks, a crow, and several weavers with the nourished mosquitoes, but with other kinds of birds the experiment has given negative results.

The blood of healthy birds submitted to the bites of mosquitoes remains completely free of parasites up to the fifth day or less; then the proteosoma, at first in small numbers, multiply so rapidly that often from 20 to 30 and more are found in the *field* of an objective. Several birds thus infected died, and at the autopsy were observed lesions

similar to those found in pernicious fever, the blood was of a brown colour, and the liver and spleen were over-charged with black pigment.

In the bird that recovered, the parasites diminished rapidly. A great number of healthy birds have been used for these experiments; those birds which were confined in the laboratory, and protected against the bites of the mosquitoes, were never infected.

The nature and the *rôle* of the black spores are more obscure than those of the germinal threads. The black spores remained unaltered in the mosquitoes and in the water; Ross kept some elements in water during seven months without observing any modifications; administered *per os* to some sparrows, the black spores never gave birth to the infection of birds.

Manson and Ross suppose that the black spores are destined to infect the larva of the mosquitoes after staying a certain time in the water, and after exposure to solar light these spores ripen in the perfect insect and reproduce the germinal threads and black spores of such a kind that the evolution cycle of the parasite is able to complete itself without necessarily passing through the bird, which is only an accidental host for the mosquito sporozoa.

Dr. Ross very kindly sent me from India some preparations showing the different phases of the transformation of proteosoma in the mosquito. I saw very clearly in these preparations the spherical elements, pigmented at the beginning, which existed often in great numbers in the lining of the stomach of the infected mosquitoes, and which arrived at their complete development—which broke off into the abdominal cavity in giving birth to germinal threads—I saw very clearly thus the accumulations of germinal threads in the salivary glands. The existence of the elements described by Ross under the name of black spores is certain, but the nature of these elements is yet hypothetical. For my part, I have been struck by the fact that the black spores are of irregular form and of very variable dimensions, while spores in general possess great uniformity of aspect. It strikes me that the black spores result from a degenerescence of germinal threads, but this idea is merely an hypothesis, and new researches are necessary to decide this question.

Be this as it may, Ross has very well described the transformation of the proteosoma of the birds in the digestive tube and in the general cavity of the mosquitoes, and he has succeeded in infecting healthy mosquitoes, causing them to be bitten by the mosquitoes nourished upon unhealthy birds. One can only point out a gap in the work of this excellent observer.

Ross has not yet seen how the proteosoma introduced into the stomach of the mosquito transforms itself in giving birth to the spherical pigmented bodies that are found at the end of thirty hours in the thickness of the coat of the stomach. Manson and Ross suppose that the same changes occur as in the observation made *in vito* by Mac Callum; the male flagella elements fertilise the female elements; the fertile elements give birth to some vermicules which are introduced into the lining of the stomach. The experiment of MacCallum's was carried out with the hæmatozoon in birds which is known under the name of halteridium and not the proteosoma, but Koch has observed that the transformations of this last parasite in the stomach of the mosquitoes were the same as those of the hateridiums.—*Janus*, April 1899.

(To be continued.)

LIEUT.-COL. CROMBIE, I.M.S., has been appointed by the India Office to attend the Congress on Tuberculosis at Berlin.

News and Notes.

THE LONDON SCHOOL OF TROPICAL MEDICINE—THE FESTIVAL DINNER.—Under the auspices of the Seamen's Hospital Society a festival dinner was held on May 10, at the Hotel Cecil, in order to raise funds to promote the formation of the London School of Tropical Medicine, and to aid in the construction of an enlargement of the branch hospital of the Society at the Royal Victoria and Albert Docks. Mr. Chamberlain presided and some 350 sat down to dinner. Amongst those present were the Belgian Minister, the Marquess of Lansdowne, the Marquess of Lorne, the Lord Chamberlain, Lord Lister, Lord Strathcona, Mr. Justice Grantham, Lord Ampthill, Lord Loch, Admiral Sir Anthony Hoskins, Admiral Keppel, Sir Wm. Priestley, Mr. H. M. Stanley, Sir Frederick Young, Sir Wm. Robinson, Sir W. R. Kynsey, Sir Gage Brown, Major the Hon. W. Rowley, Inspector General Turnbull, Dr. Stephen Mackenzie, Dr. Patrick Manson, Surgeon-Major Ross, Professor Crookshank, Dr. Dawson Williams, Mr. Malcolm Morris, Dr. Irvine Rowell, C.M.G., Colonel Andrew Duncan, I.M.S., Colonel Crombie, I.M.S., Colonel Oswald Baker, I.M.S., &c. Mr. Chamberlain, in proposing the toast of the "London School of Tropical Medicine," advocated in an able, eloquent, and telling speech the claims of the school to public support. After pointing out the responsibility the British Empire had incurred by undertaking the government of something like 300,000,000 of the coloured people of the world, Mr. Chamberlain stated: "The man who shall successfully grapple with this foe of humanity and find the cure for malaria, for the fevers desolating our colonies and dependencies in many tropical countries, and shall make the tropics livable for white men, who shall reduce the risk of disease to something like an ordinary average, will do more for the world, more for the British Empire, than the man who adds a new province to the wide dominion of the Queen. All those who co-operate in securing this result, whether by their personal service or by some pecuniary sacrifice, will be entitled to share the honour, and to add their names to the golden record of the benefactors of mankind."

Donations amounting in all to £15,800, included £3,550 from the Colonial Office, £1,000 from the India Office and £200 from the King of the Belgians. Lord Strathcona and Sir Donald Currie each gave £500 to endow a bed, and Sir H. Burdett £300 for three years for a travelling scholarship.

THE LIVERPOOL SCHOOL OF TROPICAL MEDICINE—A SPECIAL WARD—THE INAUGURAL DINNER.—On Saturday, April 22, Lord Lister opened a special ward, for the reception of patients suffering from tropical diseases, in the Royal Southern Hospital, Liverpool. The ward is to be named the Samuel Henry Thompson Ward, after the munificent donor. Mr. W. Adamson, who received the guests invited to take part in the ceremony, stated that "in the year 1877, nineteen cases of malaria were brought into the wards of the hospital in one day," and also "that during the last

few years 460 cases had been under treatment in the hospital wards." It is evident, therefore, that there is no want of clinical material, and the situation of the hospital, in the proximity of the great docks, is calculated to see that supply continued.

The Inaugural Dinner, held on the evening of the same day as the hospital ward was opened, was presided over by Mr. A. L. Jones, the chairman of the Liverpool School of Tropical Medicine. There were present the Right Hon. the Lord Mayor of Liverpool, Lord Lister (President of the Royal Society), Dr. Church (President of the Royal College of Physicians), Professor Michael Forster (President-elect of the British Association), Dr. J. W. Moore (President of the Royal College of Physicians, Ireland), Sir R. Thorne-Thorne, K.C.B. (principal medical officer to the Local Government Board), Sir William Broadbent, Bart., His Excellency F. M. Hodgson, C.M.G. (Governor of the Gold Coast), Sir C. Nixon, Sir J. Crichton Browne, Sir C. Cameron, Sir Bosdin Leech, Sir Raylton Dixon, Sir J. A. Willox, M.P., Sir Edward Russell, Mr. W. F. Lawrence, M.P., Mr. A. F. Warr, M.P., Mr. C. M'Arthur, M.P., the President of the Manchester Chamber of Commerce, Professor Clifford Allbutt (Regius Professor of Physic, Cambridge), Dr. Patrick Manson, Dr. Waldemar Hoffkin, C.I.E., Mr. W. Adamson (Vice-chairman of the Liverpool School of Tropical Diseases), Principal Glazebrook, Professors Boyce, Paterson, and Carter, Dr. Alexander, Dr. Adam, Major Ronald Ross, I.M.S. (Lecturer on Tropical Diseases to the Liverpool School), the President of the Liverpool Consular Association (the Consul-General for Brazil), the Secretary (the Consul for Uruguay), and the Treasurer (the Belgian Consul) of the same, the U.S.A. Consul, the Consuls-General for Chili, Belgium, France, Mexico, and Italy, Consuls for Costa Rica, Denmark, Norway, Sweden, Switzerland, and Venezuela; Professor Sims Woodhead, Professor S. Martin, S. Pye-Smith, Dr. Leech, Dr. R. Bradford.

In replying to the toast "The Study of Tropical Diseases," Lord Lister said "he confessed he sympathised deeply with them as Liverpool men, in feeling that it was somewhat of a hardship, or would be a hardship, if men who had studied in Liverpool under the best conceivable conditions that could exist in the British Isles should nevertheless have to go to London afterwards. Such an arrangement would act very prejudicially on the Liverpool School, because it would naturally occur to most students that he might as well go to London at once. He felt the matter so strongly that he took the opportunity of seeking an interview with Mr. Chamberlain on the subject. Mr. Chamberlain most cordially sympathised with Liverpool in its efforts, and the speaker learned from him that the arrangement proposed was only made for a single year, and Mr. Chamberlain had commissioned him (Lord Lister) to say to those interested in the Liverpool scheme that when the year had expired he would reconsider the question." Mr. Chamberlain added significantly, "We shall then see what Liverpool can do." Now having seen what Liverpool could assuredly do, he felt confident that at the expiration of the year this requirement of Mr. Chamberlain's would no longer exist.

A Tropical Medical Society.—At a meeting held in London, on May 1, Sir Wm. Kynsey, K.C.M.G., in the chair, the formation of a Society devoted to the reading of papers upon, and the discussion of, tropical diseases was considered. The idea was favourably entertained, and strongly advocated by the chairman, Dr. Patrick Manson, Lieut.-Colonel Crombie, Major Ross, I.M.S., Major Giles, I.M.S., and Mr. James Cantlie.

The editors would be pleased to receive expressions of opinion from medical men resident in the tropics upon this subject. The promoters of the scheme would thereby be enabled to gather information as to whether the establishment of such a society would be likely to receive encouragement and support.

Obituary.

GEORGE FREDERICK REYNALDS, M.R.C.S., L.R.C.P.

The blackwater fever of West Africa has claimed another member of our profession as a victim. On February 15, Mr. Reynalds, aged 31, died at Taqual, Gold Coast, where he was acting as medical officer to the Taqual and Abosso Gold Mining Co., Limited, after a residence of some fifteen months in the country. Up to the time of his illness he had enjoyed remarkably good health, but whilst returning to Taqual from seeing a patient at the neighbouring village of Tawsoo, he caught a chill and speedily developed blackwater fever. As is so frequently the case, the fever had subsided some days before death took place, and Dr. Gardiner, who attended him, was satisfied with his progress. Although weak a fatal issue was not apprehended, but he died almost suddenly, having been ill only about one week. Dr. Reynalds was greatly respected alike by Europeans and natives for his devotion to the needs of the sick under his charge, for his indefatigable labours in their behalf, for his forgetfulness of self and for his eminent professional qualities. He was a distinguished student of St. Bartholomew's Hospital, and after qualifying settled in Bermondsey, London, for a time. In 1897 he went out to the Gold Coast.

The January number of the Journal of Tropical Medicine for 1899 contains an able article on blackwater fever by Dr. Reynalds. In fact, it is the most authoritative statement in medical literature on the relation subsisting between the administration of quinine and blackwater fever. The care in compilation and the scientific acumen displayed in the article testify to the loss tropical medicine has sustained by the early death of this most promising man. Dr. Reynalds did not live to see his observations in print, for the disease he knew so well had carried him off before the Journal containing the article could reach the West Coast.

He leaves a widow and two young children to mourn his loss.

Correspondence.

ALBUMINURIA IN MALARIAL FEVER.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—Professor Thayer's observations on the frequency with which he found albumen in the urine of malarial fever cases (viz., 46·4 per cent., or, in nearly every other case) alluded to in your February number (p. 196), has attracted considerable attention in the medical press, but no one seems to have pointed out that this high proportion of albuminuria cases is by no means of universal application. It certainly does not apply to the malarial fevers found in the hospitals of ordinary Bengal districts. Dr. Thayer, I know, only refers to the cases under his own observation, but writers unacquainted with the malarial fevers might suppose that all malarial fevers had this high proportion of albuminuria cases. I have paid some attention to this point during the past autumn fever season and I am inclined to believe that 3 or 4 per cent. of cases only have albumen in the urine. It is probably different in Baltimore and Demerara, where Dr. Daniels found kidney disease in 228 out of 926 *post mortems*, an observation which agrees with that of Martin Solin, quoted by Laveran (*Traité du Paludisme*, p. 162), who found albuminuria in one-fourth of his cases. But, as Laveran says, this must be "quite exceptional."

Even in advanced malarial cachectic cases I have never found anything like this percentage. Mere intensity of the malaria will not account for this difference, for in that form of fever known in Assam as *Kala azar* (which, worm or no worm, is partly, anyhow, very bad malaria), Capt. Rogers, I.M.S., specially noted that naked-eye kidney disease was very rare, and in most of the clinical cases he quotes, "No albumen" is noted. One could not well get worse cases of malarial fever than those in the Assam Valley.

I call attention to this merely to point out that what is true as regards albuminuria in the fevers of Baltimore is certainly not true, in my experience of the ordinary malarial fevers of the plains of India. What may be the case in the *terai* fevers I know not; *e.g.*, hæmoglobinuria is occasionally found in the *terai*, but it is undoubtedly very, very rare in ordinary malarial fevers of ordinary Indian districts. Laveran's experience of albuminuria seems to agree with our Indian experience.

Yours, &c.,

W. J. BUCHANAN, B.A.M.B., *Dept. State Med.*
Bhagalpur, India. *Captain, I.M.S.*

Communications, Letters, &c., have been received from:—

C.—Staff-Surg. J. G. Coad, R.N. (Cape of Good Hope).
E.—Dr. F. H. Edmonds (B. Guiana).
L.—Dr. W. F. Law (Belfast); Dr. H. Layng (Swaton).
M.—Dr. A. McKenzie (Durban).
S.—Mr. H. M. Stanley, M.P. (Whitehall).
T.—Dr. V. Thébault (Paris).
W.—A. H. Watson, Esq., L.R.C.S.E., L.R.C.P.E., (Peterborough).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

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ON PREVENTIVE INOCULATION.

By Dr. W. M. HAFFKINE.

A Discourse before the Royal Society.

MY LORD AND GENTLEMEN,—The study of pathogenic microbes in the laboratory has provided so far only a limited amount of information as to the character and mode of propagation of epidemics, and the bacteriologist had up to now few things to disclose which he had not learnt from direct observation of the disease in nature.

It would be futile, for instance, to hand over to the laboratory a specimen of cholera, or typhoid, or plague microbes, and to expect the bacteriologist to tell which of these diseases will spread along waterways, which will cling to the houses, or spread by food or drink, or by sucking insects.

At the same time, some of the fundamental acquisitions of laboratory research already throw considerable light upon the differences and resemblances observed in epidemics; and one of the most obvious instances is the demonstration of the fact that in certain diseases the infectious virus can grow easily in artificial media, while in others it does so only with extreme difficulty, or does not do so at all.

When we take some affected tissue from a leper, or a pustule from a small-pox patient, and place it in milk, broth, or any organic matter, it produces no modification in the medium, and in the course of time loses its infective properties and dies out. When, on the other hand, we repeat the experiment with cholera, or plague, or typhoid products, instead of dying out, the contagion begins to grow and multiply, spreads in the medium, and soon transforms the whole into one mass of infectious matter.

It is evident that such a distinction—the strictly parasitic nature of one microbe and the capacity of the other to lead both a parasitic and saprophytic life—must impress most directly the ways in which these diseases spread and assume epidemic forms, and also the measures which are likely to be effective in combating them.

In the first instance the infection must remain confined entirely, or almost so, to the body of the patient, and the disease can be propagated only directly from individual to individual, or by means of their immediate belongings. It is the inability of a virus to grow in lifeless nature that communicates to the disease a strictly contagious character.

In the second case the virus spreads rapidly around the original focus, and the sources from which infection reaches fresh individuals grow in number rapidly.

From the point of view of preventive measures, in diseases like rabies, or syphilis, or small-pox, or leprosy, where infection can be found in the patient alone, precautions of isolation taken with regard to the sick and their closest surroundings must affect directly the prevalence and propagation of the disease.

Whereas, in typhoid, or cholera, where the patient is only one, and proportionately a limited, source of danger, his isolation, and the destruction of his belongings, leaves unaffected the vast cultivations of infection which are going on in nature besides.

The above remarks are intended to determine the classes of infectious diseases in which the methods of prophylactic treatment are likely to play a more important *rôle* than in others. For it is impossible to determine the position of inoculation as one fixed among other measures, and equally valid for all epidemic diseases.

Without entering into the analysis of details, it is evident that measures taken for circumscribing the prevalence of an epidemic by isolating and destroying the foci of infection are less likely to succeed in the category of diseases where the infection is cultivated and propagated by numberless objects in nature; that attempts at eradicating an epidemic, or at protecting individuals by ways which succeed in merely contagious diseases, will be in that case easily eluded; and that the necessity of personal protection by means of a prophylactic treatment will be in this case more urgently felt than in others.

The short space of time at our disposal does not allow of any further analysis of this question, and I pass to the main subject of this discourse.

The most important modern methods of prophylactic treatment are based upon the fact than an attack of disease from which an individual recovers leaves in him a condition of resistance to another attack.

The successes of Jenner and Pasteur, who utilised cultivated virus for such a treatment, have led to a general conception that there is the possibility of creating artificial immunity to diseases by treating the organism with morbid virus rendered by some special means harmless.

This definition contains in itself a generalisation which led to a considerable amount of disappointment, as the application of the principle in a number of instances did not give the expected results.

When we cultivate a pathogenic micro-organism in

a liquid medium, two different elements are obtained mixed together: the bodies of microbes and the liquid which it has modified, and into which it has secreted its own products. A modification of the entire preparation, as represented by this mixture, can be first of all obtained by filtering and separating the two elements just mentioned and considering each of them by itself. Or else the two can be left together, and only the vitality of the microbe destroyed by some physical or chemical agent. Or the constitution and the properties of each or of both of these elements can be, to a desired degree, altered by the admixture of chemicals, or by subjecting them to physical processes. Or else the vital and pathogenic properties of the microbe can be modified by artificial breeding, and then the microbe itself, or the modified products of it, used for treatment.

All these forms of virus may or may not be useful for purposes of prophylactic treatment. Thus for a considerable time it seemed to be an unattainable problem to create artificial immunity by derivates from diphtheria or tetanus virus, or from microbes in general which cause localised infections, and which do not invade the circulatory system. Similar are the difficulties still existing in tuberculous diseases, in glanders, consumption, or syphilis.

In other instances the same modification of a virus produces different results in different species of animals.

The ordinary Indian grey, as well as the brown monkey are susceptible to the plague virus, and may contract a fatal disease from being simply pricked with an infected needle. The rabbit or guinea-pig are also susceptible to the disease.

The horse, on the contrary, contracts no fatal disease after being infected even with large doses of the living virus.

If, however, a plague culture be heated and the microbes killed in it, the relations between the monkey and the horse become the reverse of the former; the guinea-pig remains comparable with the monkey, while the rabbit shares with the horse the susceptibility which it shared first with the monkey.

It will require, according to several observers, a very large dose of such treated virus to produce in the monkey or in the guinea-pig any marked rise of temperature, or any alteration of the skin at the seat of injection. While the horse answers to the injection by almost as brisk an attack of fever as if the virus was a living one, and at the seat of inoculation a tumour is produced which, if the dose be at all considerable, may lead to a complete mortification of the tissue; the rabbit similarly answers to the injection by an attack of fever and by the formation of a hard tumour at the seat of injection.

As varied as is the immediate effect of different forms of virus upon different animals, so varied is the result of the application of such virus from the point of view of immunity conferred by it.

There are animals in which the inoculation leaves no lasting effect whatever.

In others a very temporary immunity is created, vanishing in a few days.

In other cases, again, a condition appears that produces the impression as if, after the treatment, the animal has become more susceptible to a subsequent infection than is a normal, not so treated animal.

And lastly, there may be animals in which the same virus will produce an increased and long-lasting immunity.

In general, it is admissible that in the case of every disease, and with regard to every species of animal, a form of prophylactic treatment may be found that will be useful in that particular case; but that same method of treatment may or may not be applicable to another animal or to another disease affecting the same animal. It is the not taking into account this variation of circumstances that, I believe, more than anything else, has checked the success of a number of experimenters.

The study of the anti-cholera inoculation in India has revealed a new problem in the subject of prophylactic treatment. The particular character of cholera epidemics, which appear unexpectedly, do not last, and in places where they are permanent, are spread and scattered over large areas, makes the study of that disease and the demonstration of the effect of a preventive treatment in its case a matter of much greater difficulty than is the case in localised contagious diseases, like small-pox, or in plague. And although a large amount of material has been collected already, it is desirable that a number of observations be added to the present ones confirmatory of the results obtained.

The information collected permits, however, already of pointing out very important features in the working of the anti-cholera inoculation.

The most extended and continuous observations on the subject were organised by the municipality of Calcutta, upon the enlightened initiative of Dr. W. J. Simpson, and under his continuous supervision, as well as under my own. These observations refer to the cholera-stricken suburbs of Calcutta, the so-called "busties" or groups of huts situated round the tanks in which rain-water is collected during the monsoon.

Some 8000 people were inoculated in these localities, and for two years observations were made and the results collected as to the occurrences of cholera in the huts inhabited by the inoculated.

In the vast majority of cases there lived in the same families members who had not been inoculated, together with others inoculated, and the possibility thus presented itself of comparing the incidence of the disease in individuals of the same households, exposed as much as it is possible to the same chances of infection.

During the time under observation, cases of cholera occurred in seventy-seven huts in which some of the members had been inoculated. In the subjoined table the interval is given which elapsed between the application of inoculation in each particular hut and the occurrence of cholera in it. The first row of figures refers to attacks in uninoculated, and the second to attacks in inoculated members of the same households.

Amongst the uninoculated members after—1, 2, 3, 4, 5, 6, 9, 12, 13, 15, 17, 22, 34, 37, 44, 57, 62, 63, 71, 95, 99, 109, 114, 118,

And amongst the inoculated of the same households after, 0, 2, 3, 4,

119, 120, 129, 132, 139, 143, 162, 189, 191, 203, 240, 251, 219, 271,

281, 284, 300, 309, 318, 319, 334, 356, 359, 362, 370, 372, 378, 383,

384, 389, 391, 393, 394, 401, 404, 408, 416, 433, 446, 448, 453, 472, 493, 498, 673, 720, 723, 724 and 738 days.

—— 421,——459,——512,——688——735 and 738 days.

It will be seen that, for a period of 738 days, cases of cholera occurred among the uninoculated at all intervals after the date of inoculation; whereas the figures referring to the inoculated show a striking variation of the incidence when compared at various distances from the time of inoculation. Cases continued to occur among the inoculated for a period of four days after the treatment, and then for 420 days they practically remained free from the disease, only one death having occurred among them during that time. From the 421st day up to the end of the observations six cases occurred among them again.

The relative immunity in the inoculated considered separately during those three periods shows that during the first four days the inoculated had proportionately 1·86 times fewer deaths from cholera than the uninoculated.

During the period between the 5th and 420th days, *i.e.*, for a period of nearly fourteen months, the number of deaths among the inoculated was 22·62 times smaller than amongst the uninoculated. And for the rest of the time under observation the proportion in their favour fell to 1 to 1·54.

While thus the absolute number of deaths appeared strikingly influenced by inoculation, the particularity that came out from the observations in Calcutta, as well as in other places, was that the proportion of deaths to cases was not changed by the treatment.

Thus, in the observations made in a camp of coolies of the Assam-Burmah Railway Survey, out of thirty-three attacked among the uninoculated portion of the camp, twenty-nine died, and of four attacked among the inoculated, all four died.

In the Durbhanga prison, out of eleven uninoculated attacked all eleven died, while five inoculated attacked lost three.

In the Gaya gaol, twenty uninoculated attacked lost ten, and eight inoculated attacked lost five.

In a group of tea plantations in Assam, 154 cases in uninoculated had sixty deaths, fifteen cases in inoculated had four.

This circumstance, the non-reduction of the case mortality by a treatment which influenced unmistakably the case incidents, appeared as an astonishing divergence from the result of small-pox vaccination, where both the number of attacks and their fatality are reduced by the treatment.

The new aspect of the problem of preventive inoculation which thus presented itself in the observations on human communities consisted in the possibility of a prophylactic treatment being directed separately towards the reduction of the number of attacks, leaving the fatality of the disease unchecked, and towards the mitigating of the character of the disease and the reduction of the case mortality in those who get attacked.

In analysing the nature of this particular result, the following two facts known in laboratory practice presented themselves to me as of essential significance.

In patients who recover from an infectious disease the pathogenic microbe does not disappear from their body for a considerable time after their recovery. It does not do them harm any more, though when transferred to another animal it may still cause a fatal attack. For instance, a guinea-pig inoculated with the bacillus of chicken cholera can breed for weeks, in an abscess, microbes of an intense virulence without in the least suffering in its own general health.

A condition sets in in the convalescent patient, or exists in naturally immune animals, whereby they may not suffer from the result of activity of a pathogenic microbe, from its morbid products, and from that time the presence of the microbe in the system becomes innocuous. Immunity against morbid effects generated by the products of microbes does not seem to imply necessarily the ridding of the system of such microbes. It is known now that such a resistance against the products can be originated artificially, by gradually treating the system with increased quantities of toxines. The system reacts by developing antitoxines, or substances tending to neutralise the effect of the toxines.

On the other hand, Gamaleia first drew attention to the fact that it is possible to create in an animal resistance against lethal doses of virulent microbes without that animal getting any resistance against a dose of the products prepared from that microbe in the laboratory.

One seems justified, therefore, to consider separately two kinds of immunity:

One against the living microbe, which would prevent it from entering the system and causing an attack; and another against the fatality of the symptoms of the disease, when the microbe overcomes the initial resistance and does invade the system.

In the inoculation against cholera, which is done with the bodies of microbes alone, the first result is obtained.

These considerations came to be strengthened by a set of laboratory experiments by Pfeiffer and Kolle, intended to verify our Indian results, and in the course of which they detected in the serum of men inoculated with only one dose of cholera vaccine an extremely high protective power, equal to that which, in goats for instance, could be created only after a very prolonged treatment, extended over five or six months, and inincluding injection with gigantic doses of cholera vaccine.

But on analysing the properties of that serum they found that it possessed an astonishing power of destroying the cholera microbes, but exhibited no antitoxic properties capable of neutralising the effect of the products of the cholera microbe.

When, in 1896, I was confronted with the problem of working out a prophylactic treatment against the plague, I determined to put to the test the ideas originated by the observations on our cholera patients, and to attempt, in the new preventive inoculation, to obtain at once a lowering of the susceptibility to the disease and a reduction of the case mortality.

This I resolved to obtain by a combination of bodies of microbes with concentrated products of them.

In giving the above considerations, I beg they should be considered as temporary, subject to modification or to complete refutation. There may already exist facts unknown to me which are opposed to the guesses

implied by me. It was those guesses, however, that led to the actual results obtained in the plague inoculation; and, in giving the reasoning which I passed through while working out the method, I am yielding to a demand to that effect, as I myself consider that part of my communication unnecessary; the more so that the theoretical conjectures above enumerated are not shared by very eminent experimenters such as Pfeiffer himself, to whose results I owe some of my most important premises; and the correctness of the composition of the plague prophylactic, with regard to the extra-cellular toxines added to them, to what has been termed the supernatant fluid of the prophylactic, is subjected to contest.

It is certain that no theoretical conjectures conceived by one experimenter are binding, or need even be interesting, to others; and what is obligatory to them is the acceptance of the actual results obtained.

In order to accumulate for the plague prophylactic a large amount of extra-cellular toxines, the bacilli are cultivated on the surface of a liquid medium where they are suspended by means of drops of clarified butter or of cocoa-nut oil.

The bacilli grow down in long threads into the depth of the liquid, and produce what we have termed a stalactite growth in broth, an appearance singularly peculiar to this microbe, and which, I hope, will be till further discovery accepted as the exclusive diagnostic feature of this microbe.

The products of their vital exchange—the toxines—are secreted by the stalactites into the liquid and accumulated there.

The growth is periodically shaken off the drops of oil, after which a new crop appears underneath the surface of the liquid.

Thus a large quantity of bodies of microbes is collected at the bottom of the cultivation vessel, and the liquid itself gets gradually permeated with increasing quantities of toxines.

The process is continued for a period of five or six weeks, at the end of which the bodies of the microbes get extremely detoriated.

It will be seen from this that, in my eagerness to put to test our ability of influencing the case mortality, I may have, perhaps, as will be explained, if time permits later on, paid less attention as I might have done otherwise, to the problem of reducing the number of attacks; and I have now sketched out a simple plan whereby to test this circumstance, and to try to improve our results from this point of view.

In order to render harmless the inoculation of the virus above described, I determined to kill the microbes by heating the material up to 65° to 70° C.

The virus so treated, different to what one observes in some other instances, loses at once, for the animals susceptible to the disease, almost all its pathogenic power; and it was a question to determine, whether there remained in it the qualities that were sought for, viz., the power of creating *in man* a useful degree of resistance to plague.

The plan has been contested by a number of experimenters who tried a material thus prepared on different animals and failed to detect in it any immunising properties.

(*To be continued.*)

GROUP OF CASES OF MALARIAL FEVER FROM WEST AFRICA, INCLUDING A CASE OF BLACKWATER FEVER.

By Captain M. Louis Hughes, R.A.M. Corps.
Assistant Sanitary Officer, Aldershot.

Case I.—Corporal B., of the R.A.M. Corps, arrived for first time at Lokoja, Lower Niger, on March 26, 1898. He had his first attack of fever about a month later, and had several slight attacks at intervals of two or three weeks, lasting for a few hours only and amenable to small doses of quinine. One case of blackwater fever occurred while he was at Lokoja, the patient recovering and being invalided home to England.

He left for Zebba on July 23, arriving on August 6, having an attack of fever on the journey, and leaving on August 10, he arrived at Lagazon on September 15, having two slight attacks on the way.

On November 4, he left for Yellum, arriving the same night. About November 17, he first suffered from an attack of blackwater fever, which he described as follows:—"Felt bad all day, having had a severe cold for two days before, and laid down during the afternoon. About 5 p.m., when making water, noticed it was the colour of port wine. Went to bed, and in the absence of a medical officer took 10 grains of phenacetin followed an hour later by 10 grains of quinine. Had a violent headache all night, accompanied by shivering and vomiting. I continued taking quinine and next morning felt better, the vomiting and shivering having ceased, and proceeded by canoe a two days' journey to Boussa. My urine continued to be dark in colour, and I took 10 grains of quinine every four hours when awake. On arrival I felt much better, but was very jaundiced. At 5 p.m. on the 19th, my water was almost clear, and during the 20th became quite clear. During the evening, after going to bed, I passed about 10 ounces quite dark and thick, but two or three hours later I again passed clear urine. The following day I felt much better, and two days later I returned to duty, taking Easton's syrup as a tonic."

He left for Zebba November 28, arriving on December 2, remaining in good health until January 30, 1899, when he had a second attack described as follows:—"After going to bed apparently quite well, I awoke about 1 a.m. with vomiting and shivering, which continued all night, with much pain in the back and lower extremities. In the morning my urine was quite dark, and I went to hospital. The vomiting and shivering ceased. My blood was examined by Dr. Poole, who said that the malarial parasite was present. Treatment: 10 grains of phenacetin, 10 grains of quinine, and 3 minims of tincture of digitalis, every three hours. All pain ceased on the second day, and on third day felt quite well, and urine normal. Invalided for blackwater fever. Slight relapse on February 8, urine being clear after twenty-four hours." On February 26, on journey home, one day from Sierra Leone, he suffered from a slight relapse similar to the last.

The patient landed at Liverpool on March 14 last, and proceeded to Aldershot. On the journey between London and that place, he noticed his water was

somewhat dark on the evening of November 16, and he felt ill. He reported sick on the morning of the 17th, and was admitted to the Cambridge Hospital, Aldershot, under Major Dodd, R.A.M.C., who kindly permitted me to take blood films. On March 17, his temperature on admission was 100·4° F., and he says it was higher overnight. Since then it has been subnormal, and he has quite recovered, having no symptoms in hospital of importance. Treatment: quinine sulphate for three times a day and dietary. He has since been on furlough and remained in good health.

Cases II. and III.—Two other cases from the Niger Protectorate admitted at the same time with symptoms of chronic malaria, but neither had had any symptoms of blackwater fever. One came home in the same ship and the other a day later. The peripheral blood was examined by means of films spread by cigarette papers on microscope slips, fixed, dried, and stained and by fresh specimens.

November 17, 11.30 a.m.—In Case No. I, the common tertian malarial parasite was present in fair numbers, about half grown and well pigmented. Also a few ringed parasites (malignant?).

November 18, 11.30 a.m.—Case No. I: A few tertian and two ringed parasites, after which the blood became free from parasites and remained so.

November 19, Case No. II.: Full of malignant parasites well advanced in cycle, some corpuscles being doubly infected. Also a few crescents.

November 20, Case No. II.: A few crescents present.

Case No. III.: many malignant parasites present.

November 21.—Case No. II.: Free from parasites.

Case No. III., only contained crescents.

November 22.—All three cases free from parasites.

I am indebted to Dr. Manson for kindly verifying the above. He was not able to definitely corroborate the presence of the æstivo-autumnal parasite in Case No. I. The rings found, he considers, might very well have been young tertian forms; the fact that no crescents appeared subsequently in this patient's blood supports this view as to the species of parasite. He considers that there can be no doubt as to the presence of a fair number of benign tertian parasites. Dr. Manson concurred as to the presence of the æstivo-autumnal parasite in Cases II. and III.

The above cases somewhat militate against the idea that blackwater fever is the result of malaria pure and simple, and tend to support the idea that it is a disease *sui generis*, the malarial parasites being an accidental complication. For here we have a group of cases, coming from the same endemic area, in which two of them, although infected with the more malignant æstivo-autumnal parasite, failed to develop blackwater, whereas the third, although infected with the benign tertian parasite, and probably with the benign tertian parasite only, presented the characteristic symptoms of blackwater on more than one occasion. If malaria be the cause of blackwater, then these cases oblige us to infer, what at first sight seems an improbability, namely, that the climatic conditions of West Africa can modify the properties of not only the æstivo-autumnal parasite, but also of the benign tertian parasite, and, further, modify them in exactly the same direction—that of becoming powerful hæmoglobin solvents. Moreover, the modifying influences may operate more powerfully on the benign tertian than on the malignant æstivo-autumnal, so that their relative virulences as causes of blackwater may be reversed. The writer failed to find any malarial parasites in the peripheral blood of the only other case of blackwater fever he has had an opportunity of examining.

Case I. is of further interest as proving that the benign tertian parasite occurs in West Africa, a fact which, so far as I am aware, has not been ascertained hitherto.

THE OCULAR MANIFESTATIONS OF LEPROSY.—(*Conclusion.*)

By MM. E. Jeanselme and V. Morax.

Translated and abridged, with the kind permission of the authors, from the original paper in the "Annales d'Oculistique,"

By M. T. Yarr, F.R.C.S.I.

The Iris.

The iris is very frequently attacked by leprosy; both the lesions and the symptoms being very variable. Sometimes cases of leprous iritis present no special characteristics, and differ little in signs, symptoms, or course from cases of syphilitic iritis. Such cases most frequently leave posterior synechiæ.

In two of our patients we observed a form of iritis which we have not seen previously described, and which seems pathognomonic of leprosy. The surface of the iris in these cases appeared speckled with tiny grey points which could only be made out distinctly with the aid of a lens; these spots were scattered all over the iris, but were much more numerous near the sphincter: on a small scale they resembled the eruption of miliary granulations seen in certain forms of tubercular iritis. So similar were these fine granulations to the little leprous nodular lesions of the cornea that at a first casual inspection we thought for a moment they were in the cornea: as a matter of fact, there were typical nodular infiltrations of the cornea in both cases, but a careful examination with a magnifier and oblique illumination showed clearly the presence of similar spots in the iris. In one case the pupillary reaction was normal; in the other there were strong posterior synechiæ. In the former case this miliary eruption of the iris disappeared completely within six months, while the corneal infiltration, on the contrary, pursued its course. In a third patient the same miliary nodules were present, but limited to a small area of the iris.

No histological examination of these lesions has yet been made, but it seems extremely probable that their structure is analogous to that of the little lepromas we found between Bowman's membrane and the corneal stroma in cases of leprous interstitial keratitis.

Occasionally lepromas of considerable size are found, as in a case described by Hirschberg.[8] This observer concludes his description of the clinical features of the case as follows:—"The special charac-

teristic of this pathological process, which so closely resembles tuberculosis, is the rapidity with which nodules appear and disappear (as though produced by emboli)."

The commonest form of iritis appears to be that with pupillary exudation. The pupil first becomes greyish and then blocked by an opaque exudate, rendering examination of the fundus impossible, and, of course, reducing vision to almost *nil*. Once produced the exudate remains for a long time stationary. In certain cases exacerbations come on from time to time, in others symptoms of secondary glaucoma develope, ending perhaps in distension of the globe and pronounced buphthalmus.

This form of iritis was present in the case in which we made a *post-mortem* examination. Leprous lesions were not well marked, but the pupillary opening was closed by a thin membrane composed of connective tissue fibres with long pigment-containing cells. Grains of free pigment were also found amongst the connective tissue fibres. The membrane was attached to the posterior part of the free border of the iris.

Sometimes, again, lepromas of a very large size are found at the base of the iris. One of our patients, who contracted leprosy in Brittany, presented iritic lesions of this description. In the lower external irido-corneal angle two swellings in the form of a crescent could be seen, of a rosy colour and rounded towards the pupil. The growth of these lepromas gave rise to violent irritation of the eye. These lesions closely resemble gummata of the root of the iris and certain tubercular lesions of the same region.

The Ciliary Body, Retina and Choroid.

While the invasion of the ciliary body by leprosy is frequent, signs of cyclitis are rare: in fact, it is only from the presence of precipitates on the back of the cornea (keratitis punctata) that one can affirm clinically that the ciliary body is affected. We have seen this keratitis punctata in one case, the spots disposed in triangular fashion with the apex corresponding to the centre of the pupil. This characteristic disposition contrasts well with the parenchymatous nodular infiltration of the cornea (leprous interstitial keratitis), with which it might be confounded on a superficial examination.

The published histological examinations show that infection of the ciliary body and of that part of the periphery of the retina and choroid corresponding to the anterior segment of the sclerotic, is usual in leprosy of the eye. It is very unusual, however, for observers to diagnose these lesions during life, as corneal opacities and pupillary exudates so frequently render ophthalmoscopic examination impossible. We have only found one published description of fundus changes in leprosy observed during life — that of Trantas.*

Hansen and Bull, in their published writings on leprous lesions of the eye, note the fact that alterations of the fundus rarely extend beyond the ora serrata. In the case described by Doutrelepont and Wolters (*op. cit.*) there was a well-marked infiltration of the choroid in its anterior part, corresponding to the scleral lesions; bacilli, intra-cellular for the most part, abounded in the neighbourhood of the vessels there. In Philippson's case the anterior portion of the choroid showed both large and small leper cells[9]; leper cells were also found in the vesicular and nuclear layers of the retina in the same region. In our case, choroidal lesions were few, and we found the retina affected in only one small spot. In all three cases the optic nerve and central artery of the retina were unaltered.

Pathology of the Ocular Lesions.

The presence in such abundance of Hansen's bacillus in the leprous infiltrations of the eye shows plainly, even in the absence of experimental proof, the important *rôle* played by the organism in the pathogenesis of the lesions. How does the bacillus gain access to the eye? Poncet, of Cluny,[10] seems to be convinced that the infection is from without: "Examination of the pathological process shows that lesions proceed from without inwards, from the cornea to the iris, from the iris to the ciliary body, and from the ciliary body to the choroid, the fundus remaining healthy, while the superficial parts fall a prey to the bacilli. It would seem that the organism establishes itself in the connective tissue and lymphatic spaces after traversing the epithelium." Babes (*op. cit.*) propounds the same theory.

The facts, however, are against this hypothesis. So far, the lesions in ocular leprosy have invariably been secondary, and have made their appearance some years after the cutaneous or nervous lesions. In addition, a careful study of the anatomical distribution of the lesions shows that when the conjunctiva is attacked it is always *after* a subjacent lesion: a fact first noted by Hansen and Bull, and confirmed by Philippson, which we were able to verify in two of our cases.

The disease begins in the region of the limbus and especially in the episcleral portion of this region. Thence it spreads to the sub-epithelial and deep layers of the cornea, to the surface of the sclerotic, to the ciliary body, iris, and anterior parts of the retina and choroid. If, then, the bacilli are found in the episclera, while the conjunctiva above is normal, it seems impossible to admit the theory of Poncet and Babes, and we are forced to the conclusion that the infection of the eye in leprosy is from within.

How and why does Hansen's bacillus localise itself in the anterior scleral region? It seems extremely probable that it reaches this region through the blood-stream and localises there by means of emboli. Thomas[11] maintains that it is the blood which acts as the disseminator of the bacilli in the organism, and Doutrelepont and Wolters (*op. cit.*), relying on the preponderance of lesions in the neighbourhood of the vessels traversing the sclerotic, express the same belief. On the other hand, although the dissemination of the bacilli by the blood-stream furnishes a

* *Translator's note.*—I published an abstract of this interesting case in the JOURNAL of last October. The ophthalmoscopic picture of the fundus included a large patch of choroidal pigment near the macula, several tiny round spots (retinal exudates?) in the same situation, and one or two yellowish patches of choroiditis towards the periphery.—M. T. Y.

probable explanation of the invasion of the eye, it leaves unexplained the localisation of the disease in the anterior segment of the globe. Why are emboli of the bacilli so rare in the central artery of the retina and its branches, and so common in the anterior ciliary arteries? Ocular tubercle presents a similar problem—a problem the elucidation of which we must leave to others. The preference of certain micro-organisms for particular tissues is well known, and our clinical work furnishes us with daily examples, but the question of a satisfactory reason for this preference in this case is too complicated for us to think of endeavouring to solve here.

While the blood-stream is undoubtedly the chief means of disseminating the bacilli, it seems also certain that other media may serve the same purpose; witness the spread of the disease in the cornea: here free bacilli are found in the interlamellar spaces, the presence of which can only be explained by admitting the possibility of their being carried by the lymph or by means of leucocytes, as they are often found in cases in which interstitial vascularisation of the cornea does not exist.

We cannot conclude without again drawing attention to the striking analogy presented by leprous keratitis to the corneal manifestations of tubercle and hereditary syphilis. It seems to us all the more necessary to insist on the resemblance between the three processes in view of the belief held by some that interstitial keratitis is the expression, not of syphilis *per se*, but of a dyscrasia or cachexia induced by syphilis. We have the three infections, leprosy, tubercle, and syphilis, each capable of producing analogous ocular lesions. In leprosy and tubercle we know the pathogenic micro-organism and believe that its presence in the cornea is the direct cause of the infiltration and opacity. Why, then, invoke dyscrasia or cachexia to explain the phenomena of interstitial keratitis merely because the micro-organism of syphilis has not yet been discovered? Would it not be more logical to admit that here we have to deal with an active process of the same nature as that of leprosy and tubercle? Histology and microscopical anatomy show, we believe, that the process in all three diseases is identical, although clinically there are differences in the subsequent evolution of each. Tubercle infiltrates the cornea slowly and progressively, not affecting the whole for several months. Once developed, it does not appear to subside. Leprous infiltration proceeds slowly, may remain limited to a portion of the cornea for months or years, and is liable to exacerbations and subsidences without disappearing entirely. Syphilitic disease affects the whole cornea with comparative rapidity, the lesions usually attaining their acme within a month. Once developed, the infiltration remains stationary for weeks or months, regression in many cases takes place rapidly, and in some the cornea is completely restored within two or three months from the commencement of the disease. Often, of course, more or less extensive opacities remain.

Translator's note.—Considerations of space have compelled me to omit the valuable notes of cases illustrating the various forms of ocular leprosy with which the distinguished authors have enriched this monograph. Those specially interested in the subject would do well to peruse the original paper (*Annales d'Oculistique*, November, 1898) in its entirety.—M. T. Y.

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(*Continued from p. 269.*)

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THE ETIOLOGY AND TREATMENT OF BLACKWATER FEVER.

By L. W. Sambon, M.D. (Naples).
London.

Prophylaxis.

It is obviously impossible to dictate appropriate preventive measures against blackwater fever without a thorough knowledge of the natural history of the disease, and therefore the first step is to urge and assist investigation.

We already know in a general way that blackwater fever has its peculiar topographical distribution and its particular seasonal prevalence. The accurate investigation of these two points may lead to important discoveries bearing on the etiology of the disease; it will certainly enable us to adopt useful prophylatic measures. To arrive at a knowledge of the topographical distribution and seasonal prevalence of blackwater fever in its various endemic areas it is necessary to register only first attacks and obtain from each patient minute information as to his whereabouts in the two or three months previous to the attack. This length of time will certainly cover the longest incubation period of the disease. Scott, in British Central Africa, noticed that "the attack showed itself about eight days after having been in the low river country," but numerous observations prove that a much longer period may elapse before the manifestation of a striking hæmoglobinuric paroxysm. Meanwhile we may state in a general way that swampy districts should be avoided in the hot season after the rains.

The prophylactic use of quinine has been strongly recommended by Dœring. He believes that quinine prophylaxis minimises the chances of blackwater fever infection by warding off repeated malarial attacks, the predisposing influence of which is generally recognised. He further surmises that "the irritation which quinine exercises upon the red blood-corpuscles during the paroxysms is much less when the drug has been constantly administered during the afebrile period."

I certainly believe that the systematic and daily employment of quinine may ward off malarial paroxysms, and thereby remove one of the conditions most favourable to blackwater fever, but I think that quinine prophylaxis is often carried to an unnecessary and injurious extent. It should be restricted, as far as our knowledge allows, to the season and immediate localities in which malarial diseases are rife.

Treatment.

We have no specific remedy against blackwater fever. The patient may get well without any medica-

tion; therefore if we cannot help him towards recovery we should certainly not jeopardise his condition by inappropriate treatment. Numberless drugs have been tried, but they have proved to be useless or harmful. Their employment has been generally empirical or based on erroneous theories as to the pathology of the disease.

Those who conjecture blackwater fever to be a more severe manifestation of malaria propose that it should be treated by large doses of quinine. Those who believe the disease to be nothing more nor less than quinine poisoning of course declare that the administration of quinine is dangerous in the extreme.

Judging from recorded facts we find: 1) that quinine has no specific action in blackwater fever; 2) that, in a proportion of cases, it appears to be injurious.

Bastianelli, who accepts both the malarial and quinine theories of blackwater fever, suggests that the drug should be administered only in malarial hæmoglobinuria in which malarial parasites are found in the blood. Certainly the hematozoa of tertian and summer-autumn fevers have at times been found in the blood and viscera of blackwater patients, but rarely in such numbers as would lead to infer that they were more than a complicating feature.

Ordinary malarial paroxysms may recur soon after an attack of blackwater fever, but Doering and others have observed that patients who recover from blackwater fever frequently remain free from malaria as much as six months or more. Doering thinks that "through the destruction of the red blood-corpuscles in a paroxysm of blackwater fever some substance is apparently set free in the blood which is capable of making the blood immune against malarial parasites for a certain length of time." I believe that in some instances the latent malarial infection is destroyed in the more active reaction which is caused by the blackwater paroxysm, and that the subsequent length of immunity depends entirely on the chances of reinfection.

The evidence lately brought forward against quinine in blackwater fever is so great and convincing that I think the use of this drug should be forthwith discontinued. In a mixed infection of blackwater fever and malaria, in which the parasites of the malarial disease were unmistakably recognised, one is justified in administering quinine.

If ordinary malarial fever were to follow an attack of blackwater fever without giving rise to hæmoglobinuric paroxysms quinine should again be administered, because if blackwater fever infection had been still latent in the system it would have probably reappeared under the influence of the malarial paroxysms.

The microscope is indispensable in the diagnosis and treatment of tropical diseases, and the medical man unprovided with a microscope or unable to use it is seriously handicapped in tropical practice. A set of preparations of the most important micro-parasites to serve as reference or control slides would be invaluable to the young practitioner.

Some authors, unpardonably ignorant of the pathology of blackwater fever, have suggested the use of styptics such as ferric chloride, gallic acid, ergot and digitalis; others, in the name of "symptomatic treatment," have endeavoured to check vomiting and reduce temperature; others again, wishing to assist nature in its various eliminative processes, have tried all the known emetics, diaphoretics and diuretics. Thus a long list of drugs might be compiled, amongst which subnitrate of bismuth, tincture of iodine, perchloride of iron, tannic acid, ergotin, cantharides, ipecacuanha, chloroform, chloral, opium, antipyrin, phenacetin, strychnine, nitro-glycerine, pilocarpine, digitalis, mustard and pig's bile have been vaunted to be as infallible in the treatment of blackwater fever as bleeding and blistering seemed to be fifty years ago.

We have as yet no remedy known to be of the slightest value in blackwater fever. The paroxysm can neither be cut short nor can its recurrence be prevented, and the practitioner has no right to make experiments on his patients; he should be content to treat the disease by careful nursing and ordinary hygienic measures until a specific treatment be sanctioned by laboratory experimentation. In this same position we are at present with regard to typhoid fever and most infectious diseases.

Purgatives have at all times been considered useful at the outset of most diseases, and in black-water fever heroic doses of calomel, with or without jalap, have been constantly used. This preliminary calomel purge is not essential, but I see no objection to a moderate dose of calomel (5 grains) to clear the primæ viæ and check the formation of products of decomposition in the digestive tract. In most cases an irrigation of the colon will be preferable.

The patient should be allowed to drink freely. We have no better diuretic than water, and it is necessary to keep the kidneys flushed so that the eliminated hæmoglobin should be washed out from the tubules. Warm alkaline drinks, by favouring emesis, will be useful in effecting the discharge of the great quantity of bile which is secreted during the paroxysm, and will thus greatly relieve the persistent and exhausting retching. Vomiting is no contra-indication to drinking.

The patient should be carefully sponged with tepid water, and this should be done with as little disturbance as possible.

It is obnoxious to plunge the patient into a cold bath and it is useless to wrap him up in blankets and hot-water bottles which cause great discomfort. He should be kept warm, but the clothing must not be heavy nor tucked in tightly, lest the respiratory movements be in any way hindered. The room in which the patient lies should be thoroughly ventilated.

Hot fomentations and turpentine stupes may be applied to the loins to alleviate pain and prevent suppression of urine. Constipation should be relieved by a daily enema.

The food should be liquid, consisting chiefly of milk and broths. Barley-water, oatmeal-water, lemonade, and the juice of oranges may be given freely. Alcohol is not necessary in all cases, but it should be given when the weakness is marked and the pulse is failing.

The patient should be kept in bed in the horizontal position, as if the subject of severe hæmorrhage, until convalescence is well established.

NOTE ON "PRICKLY HEAT."

By Frederick Pearse, F.R.C.S.Eng.; M.R.C.P.Lond.
Bombay.

Prickly heat is usually described as a non-specific disease of the skin occurring chiefly in hot climates, and connected with excessive perspiration. It consists of an eruption of extremely minute red papules, which appear at first as red points, and then become raised into tiny hillocks. The numerous minute, closely set and slightly inflamed papules produce an appearance not unlike that caused by an irritating liniment. The small papules essentially discrete become, in severe cases, almost confluent, and some of them in severe cases develop an opacity at their apices. By pressure or rubbing a very minute quantity of a creamy material can then be expelled. The rash is extremely irritable, and attended with severe pricking and tingling sensations. A few spots resembling acne are generally also to be observed and occasionally a large sized inflamed acne spot gives the appearance of a boil. The little vesicles at the apices of the papules do not contain clear fluid, but, beyond the most minute quantity of a creamy material, there is nothing like suppuration. I believe this to be pent-up sebaceous material. In the same way the acne spots contain at first sebum, although they may subsequently inflame, and ultimately form and discharge pus.

Although anything which excites perspiration "brings up" the rash and aggravates all the symptoms, the disease does not appear to be directly connected with the sweat glands. It is here that I join issue with many authorities. It is not a form of malaria, as some assert, but a seborrhœa. There are seldom any sudamina vesicles such as are so frequent in rheumatic and other perspiring diseases, and in the excessive perspirations of active exercise. The rash of prickly heat is distinctly limited to those parts of the skin containing sebaceous glands, and will be found, for example, to show a sharp margin between the back of the hand and the palm, where the hairs and sebaceous glands suddenly leave off. It seems to have a special predilection for those parts where the hairs are soft and downy. The front of the chest and abdomen, the shoulders and back and the arms, are the most frequently and most severely affected regions. It occurs also on the face and legs, but not generally to the same degree. It is not a matter of clothing, because it occurs on the backs of the hands and on the face, and on the bare legs of children. A very fine desquamation often follows from those parts which have been most severely affected. The irritation is sometimes so intense that sleep is seriously disturbed, and the restlessness produced in the daytime is to delicate persons very wearying. There is no definite course. It may last for a few days, or it may go on for months. It varies from day to day, and a short spell of cool weather will sometimes clear it temporarily almost all away.

While excessive perspiration will very quickly produce sudamina, this is not the case in prickly heat. Many days' experience of hot weather is required before the rash develops and sudamina are seldom observed. It seems that the prolonged sweating excites at the same time the activity of the sebaceous glands. Certain of these smaller glands, not being accustomed to such activity, get their ducts choked up, and are apt to become inflamed. It is the obstruction of the ducts of the larger sebaceous glands which cause acne—those glands which are generally associated with medium-sized hairs, while the vast number of smaller glands probably connected with the downy hairs are affected in seborrhœa. I look upon prickly heat as an acute seborrhœa, and maintained in a more or less acute and active state by the continued irritation of excessive perspiration. Bacteria flourish more or less on the surface of the skin, and perhaps find the conditions of prickly heat favourable for their development. I have not, however, discovered any particular variety. The moist surface, again, is favourable for fungous growth, but this, like the bacteria, is probably the accidental associate of the disease, and not the cause of it.

Treatment.—In the way of prevention it must be remembered that as the complaint is so intimately associated with excessive perspiration, anything which tends to cause this must be avoided. Hot drinks are especially bad, but even iced drinks will excite perspiration in a heated atmosphere. A large quantity of any drink is injurious. Close, ill-ventilated rooms, too much clothing and exercise will, of course, naturally excite perspiration. These matters must therefore be attended to. The clothing should be as scanty as possible, and not made of wool or silk. Open-wove cotton material is the most comfortable. The bed must not be luxurious with soft mattresses and warm pillows, but such as will offer rapid conduction away of the body heat. A punkah at night seems necessary for many people—it is certainly a great comfort. Excessive bathing, especially with the use of soap, seems to aggravate it. It is not sufficiently borne in mind that soap removes sebaceous matter from the surface of the skin; that the skin, thus freed from its natural oil tends to become dry, rough and hard, that the sebaceous glands are thus unduly stimulated to produce more secretion, while at the same time the excessive perspiration is also irritating them to lubricate the surface. The removal of the natural grease of the skin under these circumstances is distinctly disadvantageous, and bathing should be performed with plain water. Soap is only required when bathing is neglected.

Curative treatment follows on these lines. The body should be anointed with oil for the purpose of protecting it against the irritation of the exuded sweat. It also keeps the skin soft. The best preparation is perhaps a mixture of almond oil and lanoline in the proportion, say of 8 to 1, and scented according to fancy. The body should be anointed with this night and morning, and gentle massage of the skin used at the same time relieves the distressing symptoms. The skin is sometimes so excessively tender that anything like rubbing must be avoided.

The domestic remedies of bran or ammonia in the bath are of little value. Lemon juice smeared over the skin is of doubtful benefit. Lotions of Goulard water or astringents afford but very temporary relief, and the same may be said of the numerous antiseptic dusting powders. The excessive perspiration is liable

to cause eczema intertrigo, so that drying powders for the axillæ, under large mammæ and between the skin folds of fat people are useful. On the prickly heat itself they seem to have little control. Temporary relief can be obtained by lotions of carbolic acid, of menthol, and of many other drugs, but lasting improvement is secured by the use of oil. It need hardly be said that vaseline is not the equivalent of oil for this purpose. Lanoline, however, when freely used (and it must be very freely used) and when combined with a pleasant oil, is the most successful application.

LEPROSY IN INDIA.[1]

By H. A. Acworth, C.I.E.

(Continued from page 277.)

Mr. Jonathan Hutchinson: I will now try to show that the extirpation of leprosy by natural measures—by such, I mean, as aim at removing its cause—is not perhaps so hopeless as some have imagined. The data do exist which should enable us to form a confident opinion as to the nature of its cause, and it is not one which is irremediable. Of these data, the non-medical mind is almost as well capable of judging as the trained physician, and it is much to be desired that the general public should look at the facts for itself. Surely it does not require much medical knowledge to appreciate the bearing of some of the facts, which I will now mention. Leprosy is identically the same in Iceland, Norway, the Sandwich Islands, the East and West Indies. Inference: that it has nothing whatever to do with race or climate. Leprosy affects the rich as well as the poor, and may develop itself in Europeans who go to live in leprosy districts, and it is in the army officer or the missionary precisely the same disease as in the poorest native. Inference: it has no necessary connection with poverty or personal neglect. Leprosy prevails extensively in some places, as, for instance, the Sandwich Islands, in which, as regards good clothing and climate, no sort of hardship exists. In leper asylums, the doctors, nurses, and servants never contract the disease. Inference: it is not contagious. When lepers are imported into countries where the disease does not prevail, as, for instance, from Norway into the United States, they never communicate the disease to others. Inference: It is not contagious. In further confirmation of the last proposition it may be stated that no year passes without the introduction into England of a certain number of lepers (Englishmen who have developed the disease abroad). In all these the nature of the malady is carefully concealed, and they mix with their friends without any special precautions. No single instance of any suspicion of contagion has occurred. Dr. Abraham has already referred sufficiently to a single case which happened in Ireland. It is a solitary one, and open to doubt. I have on the table before me a globe, on which I have endeavoured to mark the districts at present the homes of leprosy. The lessons of this globe are most instructive, but I must not venture to speak of them excepting in the most general terms. You will find that there are black marks on the seaboard of almost all well-populated countries. The chief exceptions are parts of Europe, the British Islands, and the United States. At many of the spots marked, however, there are but few lepers, not more perhaps than might be counted on the two hands. These few have, in most such instances, had their representatives for many generations; and although no segregation, as a rule, is attempted, their number does not increase. I ask does this look like contagion? In many other places, however—Japan, China, the Malay Peninsula, India, Ceylon, the West Indies, parts of South America, the Sandwich Islands—I have been obliged to use the brush very freely. In most of these places, however, although the number is large there is reason to believe that it is either stationary or declining. Were it capable of spreading by contagion we should—since the precautions taken are in most places of the weakest possible kind—expect it to attain universal prevalence. Surely it does not need a medical education in order to see that a disease which affects half-a-dozen people at fifty different places on the shores of the Baltic, another half-a-dozen in New Brunswick, and a few more in Crete, and which never spreads, has far closer analogy with a dietetic disease such as gout than with a contagious one like small-pox. Just as gout prevails in certain districts among certain classes and certain families and does not spread, so it is with leprosy. The legitimate inference is that both are dietetic diseases, and the legitimate hope is that as gout is yielding before tea, coffee, and temperance, so will leprosy yield before the substitution of cereals, potatoes and flesh food for salted fish. Another argument of greater cogency is, I think, fairly obtainable from examination of the leprosy globe now before us, when it is explained that in nearly all the places where the disease prevails now it had done so from time immemorial. It is not the fact that European intercourse introduced leprosy to aboriginal populations, as has been the case with many other maladies. Everywhere we found it there already, and everywhere the same. Go where you will—Japan, China, Borneo, Sumatra, New Zealand, the East and West Indies, everywhere, provided you keep near the coast, and amongst races which have learned the art of fishing—and there you will find reason to believe that leprosy has existed from the most ancient times. Quite recently, in the Fiji Islands, "Leprosy Stones," regarded with superstitious reverence, have been found in many districts (Dr. Corney). I do not see why it should be held to demand the acumen of a physician to determine the bearing of this remarkable fact. If a disease is found to be one to which the whole human race, when residing in certain districts and adopting certain habits, has from all times been liable to suffer, and if that disease is found to prevail independently in many countries, islands, as well as continents, which, through long ages, have had no communication with each other, does it not follow that it is a malady capable, so to speak, of spontaneous origin? Is it not proved almost to demonstration that it cannot owe its origin to contagion,

[1] Read at the Indian Section of the Society of Arts.

but must rather take its rise in some usage common to the human family? No usage can, I affirm, be suggested which does not concern an article of food. In this matter I am half inclined to make appeal to the intelligence of the public against the prepossessions of my own profession. I shall be told by the latter that it is absurd to compare leprosy with gout, because in the one there is a bacillus and in the other none, and that where a bacillus is present the malady can be none other than contagious. The medical mind is at present engrossed by the study of microscopic organisms, and is dazzled by the flood of new light which has dawned upon us respecting them. In a little time we shall see more clearly, and I much mistake if we shall not then come to recognise that these organisms are themselves fed by food, and in many diseases their development denotes only a stage in the causation. To say that because bacilli are present, therefore a disease must be contagious and contagious only (whether it be done in case of tuberculosis or of leprosy), is, I cannot but think, to go far beyond what has been proved. Mr. Acworth would possibly call it having regard for logic. I would myself rather say that it is a lapse of logic, since it rushes to an inference before it has been ascertained that the premises are true. The bacillus may be in latent forms ubiquitous, and may take its energy and its special form of development from the food with which it is supplied. There is nothing that I know of that has been proved in reference to the life history of these organisms to disprove such an hypothesis, and very much in its favour. For the present, however, it will probably be caviare to most zealous bacteriologists, and for that reason I venture to hint that intelligent lookers-on are entitled to form their own opinions. That I may, if possible, avoid the risk of being tedious, let me hasten to aver my own couclusions. The chief of them is this, that if a malady has been proved to be almost universally prevalent, to pay no regard to climate or to race, to riches or to poverty, and to be at the same time neither hereditary nor for practical purposes contagious, there is but one inference possible, and that is that it must be caused by some article of food. I defy anyone to accept these propositions and to arrive at any other explanation of the facts. My next conclusion is that there is but one article of food which can be plausibly suspected as the cause of leprosy, and that is *uncooked fish*. Again, I defy anyone accepting the food hypothesis to mention any other article. Just as we may be quite sure that it is no telluric or climatic influence which causes leprosy, because the disease prevails in districts which vary widely as to these; so we may be sure that it is neither flesh of pigs, rancid butter, or any kind of vegetable, for we find leprosy amongst people who vary exceedingly as to their use of all these. On the other hand, as regards uncooked fish, the evidence is *primâ facie* overwhelming. In almost every district where leprosy prevails, fish has been suspected. The disease prevailed in aboriginal communities, and in times when fish was extensively used; it still prevails in large fishing centres and along sea-coasts. Excepting under special conditions, it is unknown in inland districts. One of the most recent writers on the subject (Mr. Ehlers), speaking of its present distribution in Europe, has used the expression, "it forms, roughly speaking, a ring round the continent." It has disappeared from numberless places simultaneously with the progress of agriculture and the substitution of other forms of food for fish. It rose in Europe to its highest point when the supremacy of the Roman Catholic Church, with its numerous fish fasts, was at its height (in the time of Hildebrand), and was coincident with a low state of agriculture. It began to decline, it is true, before the days of the Reformation, but the rate of decline was apparently very much helped by that event. It still apparently, so far as Europe is concerned, has a great predilection for Catholic communities. It did not follow the Greek Church into the interior of Russia, and it does not occur there now, for that Church forbids the use of fish as well as of flesh on its fast days. In recent years it has increased largely in the Sandwich Islands and at the Cape, and at each place coincidently with the establishment of a company for salting and drying fish. The fish hypothesis covers well, I believe, the facts as to the prevalence of leprosy in all parts of the world, with the exception of certain districts in India. In many the evidence in its support is very strong indeed. Respecting certain inland districts of India which suffer, I well know that I shall be assured that fish is not there to be had, and if it were obtainable the religion of the race forbids its use. To this I am able to reply that the evidence which has been given me by those who have resided in those districts has been most contradictory, and that I have been assured by many who had good right to give opinions, that nowhere in India is fish-food inaccessible; that amongst no classes does religious prohibition wholly avail to exclude it, and, further, that the statements made by natives as to their dietetic habits are absolutely untrustworthy. The fish theory does not imply a large consumption of fish, but rather the use of it—it may be in only small quantities—but in very deleterious form. From such use I do not believe that any district or any race in India is wholly exempt. Further, it may be added that the degree of prevalence is almost always in ratio with what is probable in this respect. Of Dr. Vandyke Carter, because, as Mr. Acworth asserts eulogistically, his practice and his opinions were inconsistent, it has been said that he was "the master of logic and not its slave." I am not so stupid as not to see that there is something epigrammatic in this expression, but I am too dull to appreciate its force. Most willingly would I be, if I might, the slave of logic, and deeply should I suspect the correctness of my opinions if I could not base my practice upon them. The leprosy question has above all others suffered from this masterful disregard of logical thought. Surely it is not in accordance with either logic or common sense to go on asserting that the disease spreads by contagion, when the evidence is overwhelming in the opposite direction. Nor is it logical (and if not logical, is it wise?) to continue to act as if the disease were in some direct association with poverty and neglect, when it has been shown that the rich and cleanly suffer in precisely the same

manner. Nor is it logical to go on repeating, and trying to support by reference to authorities, statements which have been refuted over and over again by reference to facts. I understand by logic the application of sound reasoning processes to the subject in hand, and although it is very certain that nothing like "mathematical demonstration" can be attained in that with which we are now dealing, yet I feel sure that those who are weak enough to let their prejudices and prepossessions override the conclusions derived by the careful investigation of facts, will never arrive at the truth. My own theory as regards the nature of leprosy is that it is a malady closely allied to tuberculosis. Its bacillus is one that can hardly be distinguished from that of tubercle, and the two diseases touch at more points than one. It might probably be termed "fish-eaters' tuberculosis," such name being allowed to imply that the bacillus of tubercle is in the leper modified by fish-food. The remedy I would suggest is an inexpensive one, and would not in any way curtail the food of the community. It would be simply to prevail upon those living in leprosy districts never to partake of partially raw or decomposed fish. At the same time, that no effort should be spared to diffuse information; and to secure a change of habits in this matter it is very desirable that leper asylums should be founded wherever there are lepers. It should, however, be clearly understood that they are for the benefit of the leper, and not for the extermination of leprosy. To suggest that they will have any efficacy in the latter direction, and to base such suggestion on the creed that the disease spreads by contagion is to proceed on a false hypothesis, and can only lead to disappointment. The true friends of the leper are those who conscientiously seek to discover its cause and to remove it. In conclusion, I have but to repeat what was said at the outset, that I feel most grateful to the author of this paper for a most praiseworthy example of energetic benevolence, whilst, at the same time, I cannot agree with him in thinking that the measures he advocates would produce the results he anticipates. By all means, let us have leper homes, but let us abstain as much as possible from resort to the policeman and "barbed wire," and conduct them rather on the principle of the farmer who tethers his flock by their teeth. Make the homes comfortable and attractive, and the poor creatures will gladly resort to them. Any legislation which proposes to deal with leprosy as a contagious disease and to enforce isolation on that pretext deserves, I must contend, the strenuous opposition of all who are well informed as to the details of the subject. On the other hand, legislation would be well warranted which should restrain as being "unfit for human consumption," the fabrication and use of certain articles of fish-food now common in India. It would be justified on the plea that it would strike at the real cause of leprosy, diminish the terrible sufferings it produces, and in time do away altogether with the need for leper homes.

Mr. H. M. Birdwood, C.S.I., said that it was undoubtedly a courageous act on Mr. Acworth's part, in the conflicting state of scientific opinion as to the advantages, or even the possibility, of segregation, thus to start an institution in which the segregation of lepers was to be compulsory. He was not indeed without the warrant of strong scientific opinion in support of the action taken by him. He had acknowledged his indebtedness to the guidance of Dr. Vandyke Carter, whose preponderating influence had also been felt in the discussions of the Committee of the National Leprosy Fund. Dr. Bidie, of Madras, was on the same side; and so, among others, were Dr. Stephen, the Sanitary Commissioner of the Panjab, and the Public Health Committee of Calcutta. Dr. Bidie pointed out, in an important contribution to the Blue-book already referred to, that leprosy had appeared in certain countries for the first time only after the arrival of lepers from other countries. Thus leprosy was introduced into Australia by Chinese lepers; into South Africa by Kafirs; into South America by the early Portuguese colonists; into the West India Islands by negroes; into the United States of America by Chinese and Norwegians; and into Canada by Norwegians. In Norway itself the compulsory segregation of lepers had apparently resulted in the reduction of 20 per cent. of the leper population in 20 years; and Dr. Bidie, also expressed the opinion that the direct result of restrictive measures in Great Britain was the extinction of leprosy, though that result might also have been due to some extent, to other causes; improved hygienic conditions, and a higher standard of personal comfort having in time fortified the people against attack. There would, then, appear to have been some ground for accepting the direct communicability of leprosy as "a good working hypothesis" for a civil administrator, on which to base measures of reform; but Mr. Acworth was the first to put the question to a practical test, under the authority which he held to be given him by a local Muncipal Act; and his action had now been endorsed by the Indian Legislature, after a full and careful consideration of the opinion of the Leprosy Commissioners, as recorded in their able report, of the members of the committee of the National Leprosy Fund, and of members of its own experienced staff of civil and medical officers in all parts of India. No doubt this action had been taken in view of administrative considerations, rather than on strictly medical grounds; but there could be no question that the inmates of the Matoonga Asylum had benefited in health; and their state was infinitely better now than at any previous time. Nor had it been difficult, except at first, and for a short time, to detain the patients within bounds. Even in his most anxious moments, Mr. Acworth had never thought of such stringent measures as had commended themselves, in a former age, to the authorities of the Leper Asylum at Greenside, near Edinburgh. The inmates of that asylum—as stated in Dr. Bidie's report—were forbidden on pain of instant death, to leave it, or even to receive visitors; and a gallows was erected outside the hospital to show that the authorities were in earnest! Mr. Acworth was content with gentler methods; and had his reward in witnessing the complete success of his plans for the "purgation" of the City of Bombay, and in the knowledge that the unfortunate lepers, whose welfare he had at heart, were in the enjoyment of comforts to which they had all their lives, till then,

been strangers. The legislative measures adopted hitherto were marked by extreme caution; but that was only right in the present state of our knowledge of leprosy. He (Mr. Birdwood) hoped that the time would come when leper farms, which had been strongly recommended by competent advisers, would be established in selected districts throughout India. Lepers, suffering from black leprosy, ought to be withdrawn from large towns. Employment on agriculture and horticulture could not fail to improve their general health, and to cheer their last years, so far as that was possible. Leper farms were said to have succeeded in Cyprus and elsewhere; and it was satisfactory to know that the question of their establishment in India had been engaging the attention of the Government.

Sir CAVENDISH BOYLE, K.C.M.G. (Government Secretary, British Guiana), said he was not there as a controversialist or as an experienced officer of the Indian Civil Service, but merely as a humble representative of the Colonies. The more he heard of this great subject, and the more he saw of it, the more certain he was that how to deal with it was a question at present beyond the power of any man to decide, whether he were a member of the Royal College of Surgeons or of the Imperial Institute. He was quite sure they had not yet got to the bottom of it, and he feared they would not for a long while to come. His experience was simply in connection with some of the West Indian Colonies, and more recently with the colony of British Guiana, where there were not only negroes, Portuguese, and Chinese, but a considerable number of coolies. Though that colony was larger than the whole of the British Isles, it had a population of only 300,000 of whom more than a third, 108,000, were Indian coolies. During 1897-8 they dealt with 482 lepers; of course, there were many more, but these 482 were isolated, being nearly 2 per 1,000 of the population. What would be thought in London if there were a similar proportion of lepers in her population of 4,500,000; and if provision had to be made for hospitals or asylums for 9,000 leper patients? He rather thought they would look at things somewhat differently. He did not suppose that many present really knew what a terrible scourge leprosy was, and how absolutely necessary it was to do all they could to prevent bad cases mixing freely with the ordinary population. They had heard something about the disease being completely hidden, and that there were a few lepers in England whom no one suspected; but what was to be done with a poor creature who could scarcely be called a human being, who was so horribly afflicted by the disease that you could not hide him? Either you must lock him up in a cell, or let everyone who came near him know what he was suffering from. He had not seen such a case in England, but he had seen hundreds of them in the Colonies. He could not say anything on the controversy whether segregation would stop it or not, but isolation of these poor people in asylums, where they would be looked after and attended to as they could not be elsewhere, and where the rest of their miserable lives would be led as easily as possible, was an essential in every colonial Government. Dr. Abraham had referred to the apathy of the colonies with regard to the Berlin Conference, but he could assure him that the only reason why his colony was not represented was lack of funds. What he had heard that day, and what he had heard from the medical staff in the colony, however, led him to the conviction that if the members of that conference came back with honour they did not come back with peace, so far as controversy was concerned, for they did not seem to have decided who was right and who wrong. He would say, in conclusion, that coming from a colony where they had people from almost every country in the world, the Government of which was as benign and considerate as possible, when lepers were found exposing themselves to the detriment of the community, it had been thought necessary to isolate them, and he hoped that would continue.

Professor W. J. SIMPSON, M.D., said:—I wish to add my testimony to the splendid work done by Mr. Acworth. I took a special interest in leprosy in India, and I well remember that it was due to his influence and energy that a magnificent leper asylum was established on the outskirts of Bombay, that powers were put in force for the compulsory segregation of wandering and vagrant lepers, and that the streets and markets of the city were cleared of a loathsome and dangerous nuisance. I am accordingly surprised to hear to-day from the paper read that the Bill of 1896 should have been introdnced by such a speech as has been mentioned, and that no credit was given to Mr. Acworth, the Municipal Commissioner of Bombay. I did not notice it at the time. I had the advantage of going over the Matoonga Asylum when the arrangements were completed and in full working order, and I had no difficulty in concluding that perhaps, with the exception of the excellent leper asylum near Colombo, in Ceylon, it was the finest in the East, and that everything was done to ameliorate the hard lot of the lepers, and make their lives as comfortable as possible. Thoroughly in sympathy with those who hold the views that leprosy in the towns of India can only be efficiently dealt with by compulsory segregation, I was anxious to see erected on the outskirts of Calcutta a leper asylum established on similar lines to those of Bombay and Ceylon. But progress in some things is slower in the capital of the Indian Empire than in the first city of India, and it took several years to get the first important point decided, and this only after a special note was addressed by me to the Bengal Government in 1893, on the subject that compulsory segregation was absolutely necessary in any new asylum that might be built for Calcutta, and it took several years more to decide on a suitable site. Before my departure from Calcutta plans for the new asylum had been begun, but I fear plague in India has distracted attention from the leper, for I have not heard of the buildings having been completed. There was an old asylum in Calcutta, which admirably illustrated the disadvantages and evils of an asylum near dwelling-houses, and worked on the voluntary system. The asylum was in one of the best residential quarters of the native town, and was the source of frequent complaints from the neighbours, who were subjected to much annoyance by the constant passing to and fro of the lepers; but this grievance was small compared with the fact that the lepers' sores attracted

to the asylum a plague of flies, which used to invade the neighbouring houses and alight on the food. The voluntary system, moreover, was of no practical use for the purposes of detention. The lepers would be put into the asylum one day and would be out the next, and the majority used the asylum only as a shelter for the night. With this voluntary system, the same results were to be seen in Calcutta as in Bombay before the compulsory system was introduced. The streets, markets, bazaars, doors of the rich men during feasts, marriages and festivals, and the approaches and entrances to the principal temples were thronged with lepers; wherever begging and the soliciting of alms could be profitably carried on there would the lepers gather exhibiting their hideous deformities with the object of exciting pity. Alms were given to them partly from pity but mainly through fear of the leper coming too near, for the natives of India, notwithstanding any views held to the contrary in this country, have a wholesome fear of the leper and of his disease. I have always been opposed to voluntary segregation, because, however advantageous it may be in theory, it breaks down in practice. The Leprosy Commission, in advocating voluntary isolation, had no practical experience of the subject; and, after a careful perusal of their report, I have, like Mr. Acworth, been unable to understand on what grounds they recommended it, because they came to the conclusion that leprosy was not hereditary nor contagious, but arose *de novo*, and that the virus was widely distributed through space. I attribute this extraordinary conclusion, which is contrary to the experiences of all those who have given special attention to the subject in leprous countries, to the fact that the Commission tried to do too much in the limited time at their disposal. If, instead of visiting the whole of India, and seeing in different parts 2,500 lepers out of a reputed number of 105,000, or less than 3 per cent., they had visited and studied the disease in some of the worst-affected parts of the country, I believe they would have come to a different conclusion. One of the Commissioners visited Burdwan, which is one of the worst localities, remained two days, and saw 23 cases out of 4,118 or less than ½ per cent. Chamba, which, according to the last census, contained 20 to 30 lepers per 10,000, was not visited at all. Attention should have been concentrated on such places as as Ellichpur, whose leprous population increased in twenty years from 5 to 16 per 1,000; Goalpara, which increased from 7 to 19 per 1,000; Manbhum, which increased from 5 to 12 per 1,000; and Pooree, which increased from 3 to 15 per 1,000. This increase is very remarkable if it be remembered that the life of a leper is only nine to twelve years, and that, consequently, in ten years, 70 or 80 per cent. of these would die, and that, therefore, a maintenance of the average points to a fresh supply of lepers equal to that rate, to take the place of those removed by death; while any increase in the ratio indicates a much larger proportionate increase of the disease, and this is intensified by the fact that the birth-rate among lepers is abnormally low. Notwithstanding Dr. Abraham's views, I am inclined to think that all that is positively known regarding leprosy is against the conclusions of the Commission. Dr. Vandyke Carter, in an inquiry, showed that out of 1,564 lepers, into those family history he had made inquiries, 64 had a family taint in the direct line, that is, in father, mother, or grandparents; over 14 per cent. in the collateral line, that is among the uncles and aunts; and 21 per cent. in the co-equal line, *i.e.*, among brothers and sisters. This positive information in no way favours the view that the virus is widely scattered through space. On the contrary, it indicates that the virus has limitations of a very marked character, and has a very intimate relationship with family life. This investigation was made before Hansen's discovery of the leprosy bacillus, and before the relative *rôle* and proportion which heredity and communicability play in the spread of leprosy was differentiated. It is now generally conceded that heredity does not play a very important part in the production of the disease, and under these circumstances, the facts brought out by Vandyke Carter, as to family disposition, points to communicability by close and sustained intercourse. This view is supported by facts in another direction. Over and over again instances have occurred of a leper coming to live in a village or house where there was no leprosy, and sooner or later forming a focus or centre of infection to the villagers and members of the household. As it has been stated that no case of inoculation of the disease has been experimentally effected, I will mention one accidental experiment, which shows that the disease is inoculable. It is a case recorded by Dr. Tache, of New Brunswick, Canada, in which a young man was inoculated from the body of a woman whom he helped to carry to her grave. "The day was hot, and, on a sudden, liquid matter began to ooze out through a joint of the coffin, wetting the shoulders of one of the carriers. The wet, combined with the heat and the pressure of the sharp edge, produced an abrasion of the skin of the young man. The contract of the liquid with the abraided surface lasted a part of the time of the procession, and the whole length of the service, as it was only on his way home that the young man washed his sore shoulder and changed his clothing. Some months after, that man, whose health had always been robust, began to feel unwell. In a short time the symptoms of leprosy made their appearance, and he died of the disease eleven years after the occurrence. There had not been any case of leprosy in the family, whose ancestral genealogy is traced for several generations back; in fact, the disease was not yet known as leprosy, being of recent appearance in the locality and among those people. He was the fourth case in that place, the three others being the woman spoken of, the husband and sister of the woman, in the ancestry of whom there had never been any trace of the disease; the fifth case in that locality was the sister of the young man." Both Chinese and Indians believe in the communicability of leprosy, and this is no new doctrine with them. It was held years ago in India at a time of high civilisation and enlightenment. Susruta, the great Indian physician, sums up the observations of his time, which are just as applicable to-day as they were then, as follows:—(1) "Women and men having their blood and seminal fluids corrupted from leprosy (not of the white kind) get their offspring lepers." (2) "The diseases named below are transmitted from one

PHAGEDÆNA.

An illustration of a condition which is seldom, if ever seen in European practice would seem at first sight merely an attempt to preserve from oblivion a disease of a by-gone age. This may be true, as far as European hospitals are concerned; but in military practice, and in practice in the tropics amongst natives, phagedæna is unfortunately anything but rare. The newly-arrived medical practitioner in the tropics, if attached to a native hospital, or if his practice brings him in touch with native ailments, will meet with a condition of ulcerated surfaces which is new to him, and not a little puzzling.

Dr. le Dantec, whose article we publish in the current number of the Journal, has done well to remind us of the fact that phagedæna is of frequent occurrence in tropical practice, and that, although in communities where Listerism obtains it may be a negligible quantity, such is far from being the case in less favoured countries. Phagedæna was a frequent concomitant of surgical wounds in British hospitals before the days of improved sanitation. Our hospital wards, and especially our "operation" wards, were but pest houses compared with their present-day types. Phlegmonous erysipelas, gangrene, and phagedæna were in daily evidence up to the fifties, but the advance of sanitation in our houses, hospitals, and cities eradicated the more flagrant of these, and it wanted but the finishing stroke of Listerism to well nigh expunge them all. In most parts of the continent of Europe, however, sanitation did not gain the foot-hold it did in Britain, and up to the seventies, when antiseptics became general, these blots in surgical practice were still rife.

Most of our European hospitals in warm climates are models of perfection as regards sanitation and modern equipment, but to them come natives suffering from all the ills of an insanitary period. Amongst these, sores covered with the "false membrane" characteristic of phagedæna are common, and the younger men going out from European hospitals, unaccustomed to see such an ailment, are slow to recognise the conditions.

In military practice, however, even at the present day, phagedæna does, and may in future, occur. In campaigns in uncivilised countries, buildings have to be occupied which may be insanitary of themselves, or become so at any moment by the overcrowding of the sick and wounded. Overcrowding is a primary cause of phagedæna, and whilst in time of peace such a state of things would be criminal, in war it may be impossible to prevent it.

Phagedæna, as distinct from "hospital gangrene," "spreading traumatic gangrene," "phlegmonous erysipelas," "septicæmia," &c., indicates a local affection. It is a superficial infective process supervening upon open wounds or sores. Its presence is revealed by the formation of an opaque tough membrane, beneath which the tissues are infiltrated for some distance. It is associated with some febrile disturbance, but there is neither blood poisoning nor specific constitutional infection. The "false membrane" and the infiltrated tissues when stained and examined microscopically show swarms of micrococci existing singly or in chains. The bacilli are described by Dr. le Dantec as "straight, sometimes bent, and motionless," measuring, when fully grown, on an average, 7 to 12 μ.

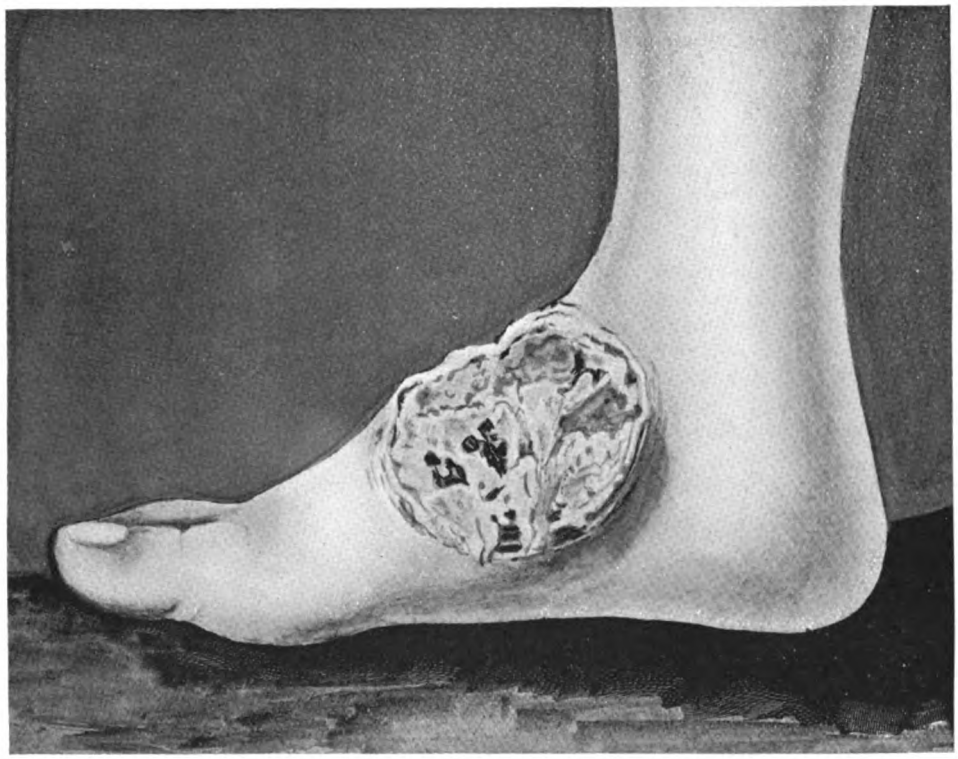
The ætiological relation these micrococci bear to the disease and its contagiousness was fairly well proved by Koch many years ago.

The preventive treatment of phagedæna it is scarcely necessary now-a-days to dilate upon, seeing that sanitation and Listerism are so well understood. Phagedæna is caused by bad drainage or overcrowding, and by any insanitary state; but, by the observation of what are termed general hygienic rules and surgical cleanliness, the disease has been eradicated.

Should phagedæna, however, appear during a military campaign, or in the ward of a native hospital, the spread of the disease is to be combated by isolation, and treating infected persons under canvas or in temporary huts. The infected ward of a building must be purified by fumigation, by washing the wood-work with a strong antiseptic solution, and by scraping and whitewashing the walls. If the building is of a temporary character, it had better be destroyed.

Local treatment consists in placing the patient under an anæsthetic and scraping the surface and edges of the wound thoroughly by a sharp spoon. The exposed surface is to be further mopped out by carbolic lotion (1—20), and then packed with cotton wool dipped in chloride of zinc moistened with water. The pack is allowed to remain for ten minutes, when a simple antiseptic dressing is applied. Should recurrence take place in any part of the wound, the process must be repeated.

The constitutional treatment required is a liberal supply of good food and medicinal or alcoholic stimulants.



PHAGEDÆNA OF WARM CLIMATES.

By Dr. LE DANTEC.

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person to another by sexual intercourse, by touch, by taking meals in the same dish, by sleeping in the same bed and sitting on the same seat, by putting on the same garments, by wearing the same garlands, and by using the same ointment—leprosy, fever, consumption, ophthalmia, and small-pox of different kinds." A fear has been expressed that compulsory segregation might produce rioting similar to that which happened when plague patients were segregated. This apprehension is based on a misapprehension of the nature of the two diseases, and the point of view from which they are looked at by the people in general. Segregation of lepers is on a totally different footing with segregation of plague patients. Leprosy is a chronic disease, loathsome in its nature, and a general object of dread. The family, as a rule, soon get tired of the patient and are glad to get rid of the lepers—in fact, he or she as often as not becomes a wandering outcast. Plague is an acute and very fatal disease; its effects are sudden, and the patient is removed to hospital at a time when the emotions of grief are most acute, and the friends are swayed by their feelings. As so many who are taken to hospital die, it appears to the Indian mind, as it would to the poorer classes of other countries, that there is a direct connection between segregating their relations in a hospital, and their death, and it is easy to understand in their excited frame of mind the revolt against segregation of plague patients. No such fears need be entertained regarding the segregation of lepers; and if the lepers are treated kindly in the asylums, and everything done for their comfort, remembering they are there, not as prisoners, but as unfortunates, for whom every care should be taken, I believe compulsory segregation will do much good, especially if, after the removal of the leper to the asylum, the house in which the leper lived is thoroughly disinfected, or, if a hut, it is burnt down.

CLIMATIC BUBOES.*

By Dr. B. Scheube,

Sanitary Adviser, Imperial Physician and Medical Referee in Greiz.

Translated by P. Falcke.

During the last few years a series of publications have appeared on an illness which has been termed, and not unsuitably, "Climatic Buboes." In 1896, Reinhold Ruge (1) issued a report on thirty-eight cases of inflammation of the inguinal glands, which, during 1888 and 1889, he had observed on the Zanzibar coast, on the East African blockade squadron. These cases, which could not be traced as having been originated by the usual causes (sexual diseases, injuries, &c.), were looked upon by the author as being climatic. The appearance of the disease was very varied. Sometimes the illness began with fever, or the same set in during its further course, when the swelling increased or suppuration set in. The fever then by no means exhibited any typical characteristic. Sometimes the affection ran its course completely without fever. The swelling of the inguinal glands mostly had its location on one side only; occasionally both sides were affected simultaneously, or one after the other. The glandular swelling generally developed rapidly within the course of a few days, and sometimes attained the size of a goose egg. The swelling consisted sometimes of a parcel of glands, sometimes of separate adjacent glands. In 60·5 per cent. of the cases the swellings, although often of considerable size, retreated; but in 39·5 per cent. of the cases operations were necessitated, when either an abscess had to be opened, or when the patient suffered greatly, or was weakened by the long duration of the illness, the diseased glands had to be excised. The duration of the illness was very diverse, and varied between a few days and a few months. None of the cases had a fatal conclusion. In a few cases there were relapses, sometimes on the same side, sometimes on the other.

Two English publications are quite independent of Ruge's statement. Charles C. Godding (2) reports the occurrence of the "non-venereal bubo" in the English marine, and ascribes climatic influence as playing an important part in its ætiology. The symptoms of the disease consist in swelling of the inguinal glands of one side, rarely of both; indisposition and fever, which, according to the chart given, is of an irregular remittent type. As a rule, reabsorption of the swelling takes place, suppuration is rare, but is frequently simulated by softening and fluctuation of the bubo. The illness may last from a few weeks to a few months. In some cases a slight sprain seems to indicate the incidental cause; in others a superficial grazing of the penis or toes; but in most, no cause at all can be traced. Age seems to exercise no influence. The affection is most often observed on the East Indian and Chinese stations of the English marine. During the period from 1888 to 1894, 733 cases yearly came under observation in the English marine, there being an average force of 56,180 men. Most illnesses occurred in the East Indies (31 cases to 1,000 men), China (25 per 1,000), and the West Indies (22 per 1,000). Fewest illnesses occurred in the Mediterranean (8 per 1,000), in Australia (9 per 1,000), and on the West Coast of Africa (13 per 1,000); England itself and the Channel yielded 10½ per thousand illnesses. In a later publication (3) Godding reports that the illness is most severe on the East African coast, but he gives no computation as to numbers.

Skinner (4), in the Presidency of Bengal, observed numerous cases of inguinal buboes of unknown origin. Of forty-nine cases that came under observation in a regiment and a battery, 28 occurred in Calcutta, 13 in Hong Kong, 4 in England, 2 in Allahabad and 2 in Malta.

Usually the appearance of the buboes was preceded by fever, which was often remittent. Frequently the patients were cachectic, and had previously suffered with malaria. Sometimes the buboes set in whilst the patients were under treatment for climatic fever or even dysentery.

Furthermore, it may be mentioned that Halfhide, according to Schön (5), mentions that idiopathic

* Reprint from the German *Archiv. für Klinische Medicin*, vol. lxiv.

inguinal glandular suppurations, often complicated with peritonitic symptoms, occur in Coronie (Surinam).

There is a further statement by O. Nagel (6), who, in German East Africa, saw a number of cases of buboes amongst officials and planters. In these all other ætiological possibilities were excluded, and thus the illness could only be ascribed to the effect of climatic influence. Most of the patients had been in East Africa over a year; a few had suffered, and two were still suffering with malaria. The fever in no case exceeded 39° C., and in only two patients had incision of the glands to be resorted to in consequence of suppuration. The swellings mostly developed rapidly, and were painful. In one case for about a week before the swelling was visible the patient suffered with severe spontaneous pain, and pain on pressure in the inguinal region affected. The cure was relatively quick; the fever ceased after the local affection had been cured.

There need be no doubt that in all these statements it is the question of the same illness. Even if the pictures of the disease as presented by the various authors—which I have reproduced in all essentials—do not coincide on all points, still the same do not present any more striking deviations than every single case observed would do. A connection of the illness with malaria is unanimously decided against by each observer. Ruge denies that malaria is the originating cause, because it could be repeatedly observed that the fever, which had previously defied quinine, departed as soon as the diseased glands were removed, and amongst the numerous cases of malaria which were simultaneously being observed on the blockade squadron, not one was complicated with inguinal glandular inflammation. Godding asserts that splenetic enlargement was only mentioned in one case, Nagel found that quinine has not the slightest effect, and Skinner sought vainly for the malaria parasite in several cases.

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[2] Charles C. Godding, "On Non-venereal Bubo," *Brit. Med. Journ.*, 1896, Sept. 26, S. 842.

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(*To be continued.*)

The Indian coolies engaged at work on the Uganda Railway have been attacked by the chigoe—the pulex penetrans. In popular language the parasite is termed the chiggir, and its introduction into India is much dreaded. The parasite is endemic to South America and the West Indies, but it reached the West Coast of Africa some time ago, no doubt following the trade route. How it reached hence to Uganda is unknown, but, with the people on the western shores of the Indian Ocean infected, it would appear to be only a question of time for it to spread eastward to India and the Malay Peninsula.

Epidemic Dysentery in East Prussia.

A few deaths are annually reported from all parts of Germany under the head of "*Ruhr*," but so few from the western, central, and southern provinces as to suggest that, as in this country, dysentery has disappeared before the progress of urban sanitation and agricultural drainage, and that the cases so designated are properly one or other form of diarrhœa. It is, however, quite otherwise in Eastern Prussia, where the bulk of the population is not Teutonic, or even Germanised Slavs, but Polish or Cassubian, poor, dirty, and ignorant, resembling in character, habits, and diet, in their fondness for potatoes and whisky, as well as in their religion, the population of western and south-western Ireland. There, in the cold, low-lying, often marshy plains traversed by the Vistula and the Oder, and extending northwards to the Baltic coast, the sanitary conditions, especially the water-supplies of the villages and smaller towns, are as bad as could elsewhere be found a century ago. There, too, dysentery prevails, associated, as it generally is, with paludal fever, and from time to time it assumes the character of an epidemic, as was the case in the province of Dantzig in the years 1895 and 1896. The official report on the outbreak, by Dr. Borntraeger, published in the *Hyg. Zeit.* for July last, is a masterpiece of methodical investigation, and would reward a careful study by one called on to inquire into an outbreak of cholera or other epidemic. The report brings out very clearly not only the connection of the disease with insanitary conditions, polluted water supply, etc., but its direct communication by personal intercourse, even where these factors were not present, as in the household of a country gentleman, and in the wards of a hospital at Dantzig, a town the sewerage and water-supply of which are among the very best. Thus the first centre whence it spread was the manorial estate of Krangen, with a resident population of 165, largely increased at times by the introduction of labourers from elsewhere.

Here a farmer's daughter, who on Whit-Sunday had been to church at Pr. Stargard, a town about four miles distant, was seized on her return by what was called "*Ruhr*" by the club doctor, but she soon recovered, and as no one else was attacked, the nature of her illness remained doubtful. A fortnight later five out of twelve girls sleeping three in a bed at the house of a man named Hennig, whose duty was to engage labourers, and two of his children, were attacked with undoubted dysentery. Three of the girls went home to as many different villages, in each of which they sowed the disease. Then a housemaid, the coachman's wife, and two children of the house-steward at the mansion, contracted it, though the establishment was supplied from a private well apparently free from any risk of contamination. All recovered except the housemaid, who went to her home in a distant village, and communicated the disease to her parents and sisters, the housemaid and her father dying. Several other families on the estate were next attacked, and Hennig's brother-in-law, a butcher, having attended the funeral of one of the children, was attacked on his return, and the village where he lived was decimated. Here the sanitary conditions were atrocious. This man, Sklowe, ran the blood and washings of his slaughter-house into a pit dug in the ground, and threw the offal into his cesspit, both of which, as well as a ditch receiving the soakage or overflow from other privies, were in close proximity to the village pump. The same story was repeated over and over again: persons who contracted the disease in one village carrying it to another, where the water-supply becoming infected, created a focus of disease, while soiled clothing, vegetables "freshened" by polluted water, and like agencies co-operated, until an epidemic was set up throughout the district that persisted for a twelvemonth. It was probably by these last-named vehicles that the members of the household at Krangen received the infection, their own water-supply being unobjectionable.—*Public Health* (May, 1899.)

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PROFESSOR HAFFKINE ON PREVENTIVE INOCULATIONS.

WE publish in this issue the important lecture on Preventive Inoculation delivered by Professor Haffkine on June 7 before the Royal Society. Those who had the privilege of hearing the lecture could not fail to be favourably impressed by the manner in which Professor Haffkine dealt with the subject. The clear discernment which he displayed as to the issues at stake, the significant facts brought to bear on those issues, and the highly successful results which he was able to show, were such as would appeal with particular force to his audience. Starting with the differences and resemblances observed in epidemics owing, as laboratory research has shown, to the parasitic nature of some microbes, and the parasitic and saprophytic life of others, this knowledge, it was stated, threw light on the ways in which diseases spread and assumed epidemic proportions, and on the measures likely to be effective in combating them. With certain diseases, in which the cause was due to a microbe capable of leading a parasitic and saprophytic life, there was one fact which had been the basis of modern methods of prophylactic treatment, and that fact was that one attack leaves after recovery a condition of resistance to another attack. Jenner and Pasteur had used cultivated virus for this treatment, and their successes had led to the idea that there is the possibility of producing artificial immunity by treating the organism with morbid virus rendered harmless. Professor Haffkine showed that this proposition has to be accepted with caution, and because its application had been made too general it had led to disappointment. In general, it is admissible that in the case of every disease, and with regard to every species of animal, a form of prophylactic treatment may be found that will be useful in that particular case; but that same method of treatment may or may not be applicable to another animal or to another disease affecting the same animal. It is not taking into account this variation of circumstances which has checked the success of a number of experimenters. The study of anti-cholera inoculation in India revealed a new problem in the subject of prophylactic treatment. The Calcutta results showed that for a period of nearly fourteen months the number of deaths among the inoculated was 22·62 times smaller than amongst the uninoculated, and for the rest of the time under observation the proportion in their favour fell to 1 to 1·54; but while the absolute number of deaths appeared strikingly influenced by inoculation, the special feature which was noted in connection with the inoculations was that the proportion of deaths to cases was not changed. The non-reduction of the case mortality, which influenced unmistakably the case incidence, was a new aspect in the problem of preventive inoculation; and it was thus in 1896, when confronted with the problem of working out a prophylactic treatment against plague, that Professor Haffkine determined to endeavour to obtain not only a lowering of the susceptibity to the disease which he had succeeded in obtaining in cholera, but also a reduction of the case mortality. With this double object in view he resolved to obtain the result by a combination of bodies of microbes with their concentrated products. In order to accumulate for the plague-prophylactic a large

amount of extra cellular toxines, the bacilli are cultivated on the surface of a liquid medium, where they are suspended by means of clarified butter or of cocoanut oil. The bacilli grow down in threads into the liquid, giving a peculiarly characteristic appearance, which at present may be accepted as the exclusive diagnostic feature of this microbe. The growth is periodically shaken off the drops of oil, after which a new crop appears. The process is continued for five to six weeks. The microbes are afterwards killed by heating the media from 65° to 70°C.

Subsequent to the necessary experiments on animals this virus was first proved to be harmless on man by inoculation of the officers of the laboratory, the Principal and Professors of the Grant Medical College, a large number of leading Europeans and native gentlemen of Bombay and others. Professor Haffkine then described the results of some of the principal applications of the prophylactic. The first was at the Byculla Jail, in Bombay, where, when plague broke out, 147 of the prisoners were inoculated and 172 left uninoculated, and the results were as follows:—

	Number	Cases	Deaths
Uninoculated ...	172	12	6
Inoculated	147	2	0

The dose given was 3 cc., which in later inoculations was reduced to 2½ cc.

The next experiment was at Umer-Kadi Common Jail, and the results were as follows:—

	Number	Cases	Deaths
Uninoculated ...	127	10	6
Inoculated... ...	147	3	0

Again, in the village of Undhera, the figures stand thus:—

	Number	Cases	Deaths
Uninoculated ...	64	27	26
Inoculated	71	8	3

Other observations, such as those at Lanowlie, at Kirkee, Dumaon, Hubli, Dharwar and Gadag, were attended with the same results, and the difference in the mortality from plague in the inoculated and uninoculated part of the communities was estimated to average over 80 per cent., approaching often 90. The duration of the effect of the plague inoculation has been ascertained to extend at least over a few months, the usual length of one epidemic.

In concluding, Professor Haffkine pointed out that the following problems had still to be worked out in the laboratory.

(1) The turning out of large quantities of material, and avoiding the variations due to the character of the plague microbe, and to the differences in the composition of the cultivation media.

(2) The further investigation of the different constituents of the plague prophylactic.

(3) The possible mitigation of the reactionary symptoms after inoculation.

(4) The study of the effect of antiseptics used for preserving the prophylactic, and along with these the important general problems connected with the theory of the curative treatment of plague, and of the life history of the plague microbe in nature.

Professor Haffkine is to be heartily congratulated on the splendid results which he has attained; and we thoroughly endorse Lord Lister's statement that, for the further solution of the problems connected with preventive inoculations, Professor Haffkine is the man qualified above all others to undertake the task and accomplish it sucessfully.

PASTEUR FILTRATION AND LARGE WATER SUPPLIES.

About two years ago the first installation of Pasteur filters for water-works supplies was opened by the Governor-General at Darjeeling. Laboratory experience and the practical results derived from the application of large numbers of the same filters on a small scale led to the expectation that the use of this filter on a large scale would prove a practicable means of permanently purifying the water. This expectation has not been disappointed. Dr. Neild Cook, the Health Officer of Calcutta, has made a large number of experiments on behalf of the authorities concerned, and has found that the Darjeeling filter is still discharging sterile water.

The peculiar material of which Pasteur filters are made, and the process by which they are pre-

pared, yield a product which, for all practical purposes, is indestructible and unaltered by any process of cleaning. The experimental difficulties, therefore, of checking the efficiency of such a filter are vastly less than in the case of filters whose filtering surface is destroyed, and has to be renewed after each cleaning, or of those in which the outer layer wears away, exposing a fresh filtering surface.

The filtering medium, owing to the wise restraint which Dr. Chamberland has put on the manufacture, retains the size and shape of the well-known tube in filters of every capacity; and the circumstance that the compressed air test will instantly show either mechanical or bacterial faults, if they exist, prevents the multiplication of units from having the slightest influence on the efficiency of the filter, as the Darjeeling installation has shown. The curve representing the output of a Pasteur filter usually drops very rapidly when the filter is first used after being cleaned, until it reaches an output from which the decrease is exceedingly slow. This output is therefore maintained for a relatively long period, and a gain in convenience far exceeding the increase of cost is obtained by calculating the capacity of a Pasteur filter, not on the initial output, but on its ultimate output.

The installation which has been recently sent to the ss. *Oceanic*, the largest steamship afloat, has been calculated on this basis. The main filter supplying the first-class saloon and the ice-making machinery occupy a ground space of only 4 ft. by 2 ft.; but by a convenient construction, in which two filtering batteries discharge into a common receiving chamber, the installation is of sufficient output to make it unlikely that the filters would ever have to be cleaned at sea, even if the voyage was longer than the short American crossing. The water is delivered to the filter from the adjacent fresh-water tank, and is drawn from it by a small steam-pump, which delivers filtered water to an adjoining covered tank provided with an air-sterilising inlet. From this point it is allowed to pass to those parts of the ship where it is required to be used. The filters for the second- and third-class passengers are distributed, and work simply under the pressure due to the head of water between the fresh-water tanks and the filters. It is very satisfactory to learn that Messrs. Harland and Wolff, who are well known as pioneers in the building of liners, have realised the necessity for greater protection of water supplies on board ship than is at present afforded. Shipowners may be congratulated on having spontaneously recognised the duty of providing bacterial filtration without the inevitable demand which would have been bound otherwise to be made upon them. It is to be hoped that soon distillation or Pasteur filtration will form the ordinary procedure for preparing drinking water on board ship.

PLAGUE IN EGYPT.

THE appearance of plague in Lower Egypt is a circumstance calculated to cause anxiety, and not without reason, in every country of Europe. At first reported to exist in three centres in Lower Egypt—Zagazig, Ismailia and Alexandria—plague is now found to have obtained a hold in Alexandria alone. Up to the present time, however, the extent of the seizure has been immaterial; not more than 30 persons have been attacked; whilst the number of deaths would seem to indicate a mildness in virulence altogether exceptional.

There are one or two circumstances about this outbreak which may be set down as peculiar. In the first place, the proportion of foreigners attacked in Alexandria is very much larger than we are accustomed to find in China or in India. We hear of Greeks and French being victims of plague, and that, too, when the virulence is mild and its extent infinitesimal. It would seem, therefore, that Europeans are not more likely to escape plague in Egypt than the native, and the consolation they derived from the history of the Hong-Kong and Bombay epidemics, that natives were the principal, practically the only severe, sufferers from the plague, receives a rude shock. We are apt to assume that Europeans are in some way immune, and that there is no danger to Western Europe, at all events, of an inroad of plague from the East. A very superficial glance,

however, at the subject of plague and its victims will suffice to dispel this notion.

British residents, in the East generally, live in a very different style to the poorer, or even the lower middle, classes in our large cities. They occupy for the most part well-built and well ventilated houses apart from the native quarter. If, therefore, plague is a filth disease, British residents in the East are, for the most part, removed from liability to infection even when an epidemic prevails around them.

Large areas of many of our great cities in Britain possess slums as bad as Calcutta or Canton can show, more abject poverty, more drunkenness, and less provision for ventilation. Public sanitation has done much to improve the lot of even our poorest neighbourhoods, but on the Continent of Europe public attempts at sanitation in many of even the fair-sized cities are but rudimentary; and in the smaller towns and villages it is scarcely attempted. We have, therefore, in every part of Europe, quarters of city habitations which will harbour plague, if it invades them, with a persistency such as the Mandvie district in Bombay can alone equal.

Egypt, moreover, has come to be the highway between the west and the east, and the presence of an infectious disease within its gates is a factor of the first commercial and sanitary importance. Alexandria, however, is some distance from the Canal, and how plague reached the city whilst other places more in the line of traffic escaped is remarkable. During the severe cholera epidemic of 1883, Alexandria was the last place in Lower Egypt to be attacked, and it escaped with a very slight outbreak. Now it is the first to be infected by plague, and how the disease was first introduced has not yet been ascertained. The infection of Bombay remains a mystery, unless it was carried by merchandise or passengers from Hong Kong; and although this possibility is feasible, seeing that there is direct communication between the two, there is little or no direct communication between Alexandria and Bombay.

Returning pilgrims from Mecca are suggested as having brought it, but no returned pilgrim has been known to have been attacked so far in Alexandria. The mildness of the outbreak resembles the Calcutta seizure, where, although every condition of native insanitation obtains, the disease has aborted. It is to be hoped that in Alexandria, as in Calcutta, the microbe will find an unsuitable soil for development. Yet it behoves the Egyptian Government, and the European ports within seven days' sail of Alexandria, to be vigilant; otherwise a portion of Europe may suffer from plague as the towns in the Bombay Presidency have suffered and are suffering still.

Translations.

THE PHAGEDÆNA OF WARM CLIMATES; ITS IDENTITY WITH HOSPITAL GANGRENE, ITS PATHOLOGY, SYMPTOMS, AND TREATMENT.

By Dr. Le Dantec.

Translated by P. Falcke.

The phagedæna of warm climates is the name applied to a complication of sores presenting a pulpy, greyish, sloughing surface, and a fetid exudation which infiltrates and destroys the surrounding tissues.

Vinson was the first who, in 1857, described an epidemic of ulcers which befel the coolies which the s.s. *Mascareignes* was carrying from the coast of Mozambique to Réunion. This ulcer received the name of "ulcer of Mozambique." Chapuis informs us that during the year 1861 the phagedænic ulcer caused great suffering to the galley-slaves taken to Guiana, there having been 1812 cases of phagedænic ulcer out of 8,373 persons admitted to the hospital; he confirms the identity of the Mozambique ulcer with the Guiana ulcer.

At the same time the expedition in Cochin China was being made, and phagedæna claimed numerous victims amongst the expeditionary force. In fact, after the taking of Tourane, out of a total of 6,000 men, there were 700 suffering with ulcers, entailing 100 deaths and 30 amputations. This ulcer was called *Annamite sore*.

In 1862 Le Roy de Méricourt and Rochard agreed in considering the Annamite sore, the Mozambique ulcer, and the Guiana ulcer as one and the same disease, under the name of the phagedænic ulcer of hot countries. Since this time the phagedænic ulcer has been identified in several other colonies, in the Antilles, Senegal, and in the Soudan. In one word, the medical geography of the phagedænic ulcer extends throughout the tropical zone.

Being in Guiana in 1884 I had the opportunity of observing many phagedænic ulcers on the persons of condemned Arabs. I was struck by the presence of a characteristic exudation on the surface of the sores, and the idea occurred to me to examine this exudation under the microscope after staining with aniline.

I discovered a swarm of bacilli—in fact, in such considerable numbers as if they had been in pure culture.

These bacilli were straight, sometimes bent, and motionless. When they had attained their full length they measured on an average 7 to 12 μ. Examination of the blood obtained from a prick of the index finger demonstrated that the microbe did not invade the general circulation. My preparations were submitted to M. Roux, who concluded that "it is very probable that the bacillus predominating in all cases of ulcer is the cause of the disease; in any case, while awaiting the proof of the same through inoculation by pure cultures, it is important to call attention to the presence of this bacillus." My investigations were soon confirmed by Clarac in Martinique in 1885, by Petit in Mayotte during the same year, and more recently by Boinet in Tonquin. In fact, the two latter investigators have even added that they had been successful in cultivating the phagedænic microbe; it is easy, however, to believe that they fell into error through the presence of saprophyte microbes which sprang up instead of, and in place of, the specific microbe. As a matter of fact, the phagedænic bacillus cannot be cultivated on the soil of ordinary cultures, such as bouillon, gelatine, agar, potato, &c. It possesses another important characteristic, that is, that it does not take Gram's stain, whilst all the bacilli which we have isolated by culture of the phagedænic exudation distinctly take this stain. Having been unable to obtain cultures we tried to inoculate the exudation into animals into wounds made by an incision with a knife. Our results, however, on dogs, rabbits and guinea pigs, were always negative.

In 1896 Vincent had the opportunity of observing 47 Arabians returning from Madagascar, who were suffering with hospital gangrene sores. These patients hailed from a country where the phagedænic ulcer is endemic. Is there any relation between the phagedæna of warm countries and hospital gangrene? It is this point we wish to elucidate.

Vincent, in his remarkable memoirs published in the *Annales de l'Institut Pasteur*, describes a motionless bacillus, which most often is straight, but sometimes curved, the shortest forms measure 3 or 4 μ; but filamentous elements exist. This bacillus is not in the blood generally, it does not take Gram's stain, it is not culturable on the usual soils; and finally, the inoculation of the exudation has no effect on the rabbit, guinea pig, or white rat. Vincent obtained positive inoculations on cachetic tuberculous rabbits, or by inoculating beneath the skin of a normal rabbit a mixture of dried pulp, coli-bacilli, and yellow staphylococci.

Coyon confirmed the researches of Vincent in a case of hospital gangrene observed in the practice of M. Ricard in Paris, on the person of a groom who had come from abroad. Thus, if we compared the microbe found by Vincent in the exudation of the purulent ulcers with that which we have met with in the phagedænic ulcer of Guiana, it would be impossible to differentiate them, both being the same colour, same dimensions, and both being impossible to cultivate or inoculate.

Some doctors, who doubtless have had the opportunity of seeing Hospital gangrene in France, have confirmed the identity of the same with the phagedæna of warm lands. Thus Thorel in his Mekong expedition compares phagedæna to hospital gangrene. Monestier (1867) distinguishes two kinds of Mozambique ulcers:—(1) The simple atonic ulcer; (2) The gangrenous ulcer or hospital gangrene. Finally, Fontan, speaking of phagedænic ulcers of warm countries (*Archives de Médicine Navale*, 1888), remarks that they *are sometimes anæmic, sometimes sphacelic, often covered with membranous exudations resembling then the covered sores of hospital gangrene.*

Clinical and bacteriological study thus leads us to admit the identity of phagedæna of warm climates with hospital gangrene.

In recapitulating the various phases through which the question of phagedæna of warm countries has passed we see that, in the first period, every observer describes, as special to the region in which he happens to be, a complication transforming simple sores into formidable ulcers; hence the names Mozambique ulcer, Annamite sore, sore of the Yemen, &c. In the second period Le Roy de Méricourt and Rochard confirm the identity of these diverse varieties of ulcers and unite them under the generic name of "phagedænic ulcers of warm countries."

Finally, during the third—the present period—the identity of phagedæna of warm countries with hospital gangrene seems to find double support in bacteriology and in clinical experience.

Hospital gangrene has become a very rare affection in temperate climes, and apart from the contingencies of great wars, it would be difficult to study it in Europe. Such is not the case in hot countries, where, so to say, it is met at every turn. It is therefore indispensable that it should be recognised at a glance. We shall therefore discuss the conditions that give birth to phagedæna.

It is necessary, primarily, to distinguish the phagedæna which comes as a complication of the wounds caused by fire-arms, and which has nothing special in tropical zones from the phagedæna which I shall call spontaneous. It is the study of this last form which permits us to investigate the pathology of the phagedænic ulcer. Spontaneous phagedæna has a very marked predilection to attack the lower limbs; 95 times out of 100, in fact, the phagedænic ulcer has its seat on the feet or legs. Two conditions appear necessary for the development of phagedæna in the colonies:—(1) The existence of a continuous discharge on the skin; (2) the exposure of such a sore to the impurities of a damp soil.

Sores on the lower limbs are of great frequency in hot countries. When passing through certain forests of Indo-China one is exposed at each step to the bites of leeches. On the East coast of Africa and in Guiana it is wise to attend to the feet every evening to remove the sand-flies as soon as they commence to perforate the epidermis. Finally, on long marches the friction of shoes at the ankle allows of a channel of entrances for the virus. Bamboo splinters may be pointed out likewise as predisposing causes for phagedænic sores.

By the enumeration of these causes it is easy to guess that natives who walk bare-footed are more

liable to attacks of phagedæna than Europeans, who are better shielded against these incessant dangers. Thus, though Europeans suffer but little with ulcers, this fact should be ascribed not to immunity from phagedæna, but to the hygienic precautions which they take during a march.

Given a skin abrasion, a second condition is necessary to transform the common sore into the "Annamite" sore; that is the contamination of the sore with damp earth. In the hospital of Ji-Cau, Mathias, in 1886, treated thirty phagedænic ulcers which had developed on the sappers employed in constructing a bridge and who worked with their legs in mud. The sampan men, who in their business are often obliged to walk in the water and mud to fetch or carry their loads, are particularly liable to phagedæna. In the same way the Annamite peasant, who, at ordinary times is affected on the lower limbs, is attacked on the upper limbs at the time of the rice-harvest, for then the arms come into contact with the soil of the rice-fields. It is thus probable that the natural abiding place of the microbe is the damp earth, not the hospital.

The soil contains three microbes dangerous to the sores of man:—

(1) The septic bacillus which produces gangrenous septicæmia.

(2) The bacillus of Nicolaïer which sets up tetanus.

(3) The phagedænic bacillus which generates phagedæna in warm climates and hospital gangrene in temperate countries.

Any sore coming in contact with damp soil in certain districts may become a phagedænic ulcer; this explains the frequency of "Annamite" sores on the feet of all the natives who walk bare-footed. The dorsal surface of the foot and the anterior surface of the leg are the most habitual seats of the ulcer, then comes the thigh and the upper limbs, but it may be seen on all parts of the body, particularly where there has been an abrasion of the skin. Thus Jourdenil has observed the disease on the elbow, the pubis, the lobe of the ear, &c. Collomb cites a case of phagedæna succeeding a hypodermic injection of quinine, and Good confirms an analogous case on the person of one of his companions, Dr. Guirriec.

The invasion of a sore by phagedæna is announced by some particular symptoms; the sore becomes painful, the granulations take a violet tint, the suppuration becomes abundant, fetid, sometimes sanguinous, and finally after a few days, on removing the bandage, a greyish exudation is revealed on the surface of the sore; sometimes this is greenish, and it adheres strongly to the adjacent tissues. This pulpy exudation infiltrates all round and disorganises the tissues, causing them to slough; it generates a brownish ichor, with a penetrating fetid and nauseating odour which may be compared to the smell of putrid fat. In the case of natives who are robust and have good constitutions, phagedæna limits its ravages, and it is not a rare thing to see natives who have been subject to phagedænic ulcers of several months' standing. These chronic ulcers are circular, cup-shaped, the edges are projecting, irregular and hard; the bottom of the sore is occupied by a pulpy, greyish mass, a mixture of sphacelated tissues and parasites. But in the case of an unhealthy native the disease makes rapid strides; a putrid, whitish discharge, sometimes several inches thick, spreads, over the sore, infiltrating the surface and gaining depth; it forms gangrenous patches, dissects between the muscles and tendons; the bones, through necrosis, appear black; the articulations are open, veritable disarticulations being produced.

Some authors state that there is a diminution of cutaneous sensitiveness on the edges of the sore; in contradiction, we have confirmed the fact that the marginal zone, of the colour of winedregs, is always painful when the phagedænic ulcer threatens. As soon as the ulcer becomes chronic, the edges of the sore become covered with scabs which naturally diminish the sensibility of the adjacent parts, but we have never verified real anæsthesia except in the case of leprous ulcers. In the case of anæsthesia of the edges of the sore it is well to examine for other signs of leprosy.

It is easy to distinguish the phagedænic ulcer from other ulcerations, such as tonga, pian, &c., which are met with in hot countries.

Tonga is an ulceration which most frequently is tuberculous; Pian, on the other hand, is a fleshy excrescence in shape like a strawberry. As to the numerous atonic ulcers to be met with in the colonies, they are not distinctive, and resemble the atonic ulcers of temperate countries. These atonic ulcers are frequently seen on anæmic persons, and like all other sores may become phagedænic, but they do not essentially constitute phagedænic ulcers. In one word, and on this point I insist, an ulcer is not to be called phagedænic unless covered by a greyish exudation, a sort of false membrane which adheres to the subjacent tissues and disintegrates them commensurately with its infiltration of them. In doubtful cases a microscopical examination of the exudation speedily elucidates the nature of the ulcer. In fact, it suffices to place a small particle of the grey mass between two cover-glasses, to dry over a spirit lamp, and to stain with aniline. There will then be seen on the field an innumerable quantity of bacilli accompanied by some micrococci. The latter, somewhat numerous in the superficial parts of the false membrane, become rarer and rarer in the deep parts. The ichor, though rich in microbes, contains fewer microbes than the membrane. If this purulent liquid be kept in a pipette it will be found that the staphylococci and streptococci multply but not the bacilli. We advise those who wish to try to cultivate the phagedænic microbe to remove the membrane by means of the lancet or curette, to place it on a cover-class and to let it dry in the sun at a temperature of 30°C or 35°C. In this way it may be sent to Europe for examination, or one may lay in a stock with a view to ulterior researches. We have received consignments of this sort from the Soudan in a perfect state of preservation from my pupil and friend Dr. Boyé.

We have already said that our inoculatory experiments on dogs, rabbits and guinea pigs proved abortive. Gayet was not more successful with the monkey, nor Jourdenil with man. Notwithstanding these negative results, comfirmed by experiment, contagion appears possible, as proved by certain clinical

facts. Winson admitted that flies going from one sore to another were the propagatory media of this illness. Bassignot cites a peculiar case of auto-inoculation. An Annamite had a phagedænic ulcer on his heel, and squatting in the indigeneous manner made a positive inoculation on the corresponding ischium. Col was on board the *Garonne*, and on a return voyage from Cochin China witnessed many cases of contagion. Finally, Boinet has observed the same fact in Tonquin. Thus, a Muong merchant, who had an enormous phagedænic ulcer on his left leg, inoculated four little ulcerations which he had on his right leg by bandaging them with linen defiled by the ulcer.

If we now try to investigate the causes which may favour inoculation, we see that in this the soil plays an important part. Clinical experience demonstrates that contagion is the easier the more anæmic the subject. Malarial cachexia in particular is a potent predisposing factor. The experimental investigations of Vincent have now proved that tuberculous cachexia plays a similar part. The same author has called attention to the predisposing power which several microbes possess in regard to infection. In this there is some analogy to what is observed in those two other telluric infections, septicæmia and tetanus. The investigations of Vaillard and Vincent, and those of Besson, have in fact shown that the presence of predisposing microbes is necessary to produce tetanic and septic infections, when the pathogenic microbes of these illnesses are deprived of their toxin.

From a clinical point of view and in the order of morbid associations, we must signalise still another curious characteristic in the history of phagedæna; this consists in a violent increase of the phagedæna coincident with the breaking out of every access of intermittent fever. It is also no rarity, on removing the bandage from a wound the day after an attack of fever, to see the ulcer enlarged by an inch or more.

Such is the affection which one has often to treat in the colonies. In military campaigns it is sufficient to arrest the progress of our troops, and we must employ every means at our disposal to rid ourselves of it. It is a sickness widespread among the natives of Cochin China, Madagascar and Dahomey, and, as in these very colonies we recruit soldiers for the formation of our colonial infantry, it is our duty to minutely examine the men, to be assured that they have no sores complicatcd by phagedæna. We thus eliminate men who not only would be worthless from a military point of view, but would be impediments on the march, and would doubtless form the starting point of the epidemic on the arrival of troops in barracks or in hospital. It does not suffice to take precautions at the moment of enlistment; during expeditions other measures must be adopted to avoid the various dangers to which men are exposed when crossing rice fields and small fords. A movable puttee of supple material, worn in the style of a light sandal, answers the purpose. This footgear must be very easy, or the native will carry it in his knapsack, not on his feet. During the last campaigns in Tonquin the Annamite soldiers enveloped their legs with bands of material forming puttees. This simple precaution diminished the frequency of the ulcer in a notable degree.

When phagedæna has attacked a sore, how are we to set about to cure it? The first measure to take is to remove the false membrane or exudation, which is composed largely of phagedænic microbes. We have often destroyed this gangrenous putridity by two or three applications of the actual cautery. When the cauterisation was thorough, the scab fell off three or four days after, revealing a pink sore free from all complication. Bichloride of mercury compresses protected the sore from the return of the offensive evil, and brought about a rapid cure. Clarac employed the curette to get rid of the false membrane, and his example has been followed by many surgeons. All treatments employed at the present day to cure phagedæna have as their basis the scooping out of the sore; they only differ as to the antiseptic employed at subsequent dressings. Clarac employed bichloride compresses; Fontan, powdered antiseptics: Vincent, after the scooping out, scattered chloride of lime freely over the sores one in ten parts; Ricard used camphorated ether; others used tincture of iodine. In one word, each surgeon has given preference to one antiseptic, but it may be said that each and all are successful, and thanks to them, one can easily master the most terrible of the sores of hot countries. When the sore is covered with healthy granulations, one may graft the epidermis or dermis, following the method of Thiersch, but as a rule it is not necessary to have recourse to grafting, if one can encourage cicatrisation by means of appropriate dressings. To attain this result I can highly recommend the use of strips of diachylon following Baynton's method. In order that this dressing may produce the best effect it is necessary to wait till the granulations are at the same level as the skin. If the edges of the sore are slightly hardened, as often is the case in chronic ulcers, one must not hesitate to cut down these borders with the scissors or knife, for before putting on diachylon everything must be ready for cicatrisation.

Baynton prepared his dressings as follows: he divided a piece of diachylon plaster into strips, two or three inches wide, and long enough to go once and half again, round the limb. He trimmed down the neighbouring parts of the ulcer and applied his strips; placing half width of each over the healthy side of the limb, he crossed the ends over the ulcer on each side, which they thus drew together. He commenced by placing the first strip a little below the ulcerated spot, he then applied the others, each covering the other by a third of its width. He then enveloped all with compresses folded several times and covered the limb by a roller bandage from the toes to the knee. Baynton's dressings have the advantage of exercising a continual compression on the sore; but it has the disadvantage of taking a long time to apply; it is therefore more convenient to content oneself with a simple diachylon bandage made with strips covering the sore without going round the leg.

One may exercise gentle compression by means of a pad of cotton tightened by a rolled bandage. With syphilitic subjects it is often an advantage to replace the strips of diachylon by bandages of plaster of Vigo. But with all patients one must not forget to apply the general principles of surgery. Half the treatment of affections of feet and legs consists in maintaining the

horizontal position. It is unnecessary to add that one must not neglect general medication, more especially in the case of malarial cachexia, which one must treat with quinine, or, if one can, by sending the patient home. To summarise, the treatment of phagedænic ulcer should be as follows :—

(1) Place the patient in the horizontal position, the leg somewhat elevated.

(2) Remove the false membrane by means of the curette.

(3) Destroy any germs which may have escaped the curette by means of antiseptics.

(4) Encourage cicatrisation of the ulcer, now become a simple sore, by means of grafting, or more simply, by strips of diachylon.

Such is the history of the disease named "phagedæna of warm countries." It is similar to that of hospital gangrene in temperate zones. Hospital gangrene has been ousted from our hospitals by antisepsis. Let us hope that phagedæna will disappear in its turn from the tropical zone, thanks to the hygienic measures which it is our duty to disseminate amongst the exotic populations.

Recent Literature on Tropical Medicine.

TROPICAL OPHTHALMOLOGY.

Pigment Stains in the Conjunctiva.—Steiner (*Centralbl. f. Augenheilk.*), when residing in Soorabaya, Java, frequently observed curious pigment stains in the conjunctiva of Malays which appeared to be sequelæ of trachoma. These patches, black as ink and often very extensive, mainly affected the tarsal conjunctiva of the upper lid, although occasionally seen on the globe. In one case the patch developed into a veritable tumour, a "benign pigmented adenoma." He also noticed similar pigment stains among the Chinese living in Java.

I have frequently seen pigmentation of the conjunctiva amongst the Chinese in Hong Kong, not, however, as a sequela of trachoma, but of epithelial xerosis; the patches were neither so dark nor so extensive as those described by Steiner, and were usually situated in the lower lid. I notice that Herbert, of Bombay (*Transactions of Ophthalmological Society*, 1898), describes pigmentation of the conjunctiva as a characteristic sequela of xerosis in the dark-skinned races, and states he has seen cases in Bombay in which the conjunctiva became uniformly pigmented.

"Tarsostrophy."—The *Annales d'Oculistique* for April gives prominence to a paper by Dr. Bitzos, of Cairo, advocating the treatment of trachoma by an operation which he names "tarsostrophy." Believing that surface treatment of granular lids does not lead to a sure and certain cure, and that to combat the disease radically and effectively it is necessary to treat the tarsus itself, he advocates nothing less than turning the tarsus inside out in order to destroy its anatomical aptitude for producing trachoma (*dé manière à détruire ces aptitudes anatomiques pour la pathogénie du trachome*). The operative procedure, which he describes at length, consists in dissecting out the tarsus of the upper lid and replacing it with the internal surface (*la surface trachomatogène*) external, and its external angle internal, fixing it in its new position with fine sutures. This operation is stated to give excellent results, and M. Bitzos thinks it should be performed in all cases of trachoma occasioning any inconvenience (*si la maladie tourment taut soit peu la personne*). In very severe and chronic cases, in patients indifferent to cosmetic considerations, he removes the tarsus altogether. This last procedure, by the way, was first described by Kuhut at the Moscow International Congress, and has recently been strongly recommended by C. A. Wood in certain cases (*Annals of Ophthalmology*, July, 1898).

"Tarsostrophy" seems a somewhat startling and heroic method of treating an ordinary trachoma, and I am not sure that I am convinced of the soundness of the theory on which it is based. However, the opinions of a surgeon with such extensive experience of trachoma as practice in Cairo affords are entitled to every consideration. Further reports from M. Bitzos, with full details of cases (omitted in the present paper) will be awaited with interest.

Strong Solutions of Corrosive Sublimate in Trachoma.—Hardly less drastic than "tarsostrophy" is the procedure adopted by Dr. Querenghi, of Milan (*Ann. d'Oc.*, April 1899), and first advocated by Guaita in 1886. Dr. Querenghi states that for the last five years he has treated all trachoma cases by means of 4 to 5 per 1000 solution of corrosive sublimate, "with splendid results." The lids are everted, and the granular surface thoroughly rubbed with cotton wool soaked in the solution. This procedure is carried out every other day, and invariably, according to the author, cures the most obstinate cases. Corneal complications (pannus, ulcers, &c.) do not constitute a contra-indication; at the same time it is necessary to avoid touching the cornea with the solution. The pain produced by the application is naturally severe, but "not insupportable." Dr. Querenghi, however, admits that patients occasionally "desert."

The dangers of this energetic mode of treatment appear to me to be very great; the cornea is exposed to considerable risk, and I cannot help thinking the liability to subsequent cicatricial contraction and entropion is much increased. The ordinary modes of treating trachoma afford a safe and certain means of cure in the vast majority of cases, if carefully and conscientiously carried out by the surgeon or a trained attendant; hence the practice which is becoming so common amongst distinguished oculists, of lauding severe and dangerous procedures as suitable to all cases, can hardly be sufficiently deprecated.

M. T. Yarr.

INDIA.

Anti-Choleraic Inoculations of Tea Coolies.

In a letter from Mr. J. Buckingham, Chairman of the Assam Branch Indian Tea Association, to Mr. B. V. Nicholl, Deputy Commissioner, Sibsagar, regarding registers to be kept of cholera cases among inoculated coolies, the Chairman says:—"There are many obstacles at present in the way of inoculating coolies, and I should deem it a great favour if you would send a copy of this letter to the Commissioner of the Assam Valley urging the necessity of inoculation being started in the recruiting districts on a more satisfactory and systematic basis, for until this is done, returns cannot be of much value. The planters of Assam are fully alive to the importance of inoculation for cholera, and the Administration Report fully bears out the inestimable value of it, the death-rate from cholera among the general body of immigrants *en route* to the Assam Valley being 16·7 per cent. as compared with ·49 per cent. among inoculated coolies during 1897-98."—*Indian Medical Record* (May).

Relapsing Fever (Sunjar) in the Kumaon Himalayas.

Surgeon-Captain Leonard Rogers, M.D., M.R.C.P., B.S., F.R.C.S., Officiating Imperial Bacteriologist to the Government of India, contributes an interesting note on this subject to the May number of the *Indian Medical Gazette*. He writes:—"As I recently had an opportunity of investigating an outbreak of relapsing fever some 60 miles from the Muktesar Laboratory in the Kumaon Hills which had been reported as an outbreak of 'sunjar,' which is thought by some to be a mild form of plague, a brief record of the facts ob-

served may be worthy of being placed on record. The disease was greatly on the decline at the time of my visit, which was a flying one on account of other important work, and the following details are taken from my notes which were made on the spot.

"On my arrival at the scene of the outbreak, I was informed that the epidemic had nearly died out, but a number of patients who had recently recovered were collected for my inspection, and a careful questioning of them elicited the following facts. In the village of Suwal, which is situated not far from Champawat in the south-east of the Almora district, the first person to be attacked was a woman aged 40, who had not recently left the village, and she died on the seventh day of her attack of fever. Seven days after this woman fell ill, a man, who lived in the next house, which was built side by side with the first one and in contact with it, was attacked by fever, and within the next few days the whole of his family, consisting of a wife and five children, between the ages of 4 and 13, also got it. The fever lasted in these cases from eight to ten days, and then after an interval of four or five days of freedom from fever, it recurred, and lasted from four to seven days. It is not necessary to follow in detail all the cases, but it will suffice to mention that in ten other cases the average duration of the first attack of fever was 7·7 days, of the interval of freedom from fever was 6·1 days, while the relapse averaged six days, figures which correspond closely with those of the classical descriptions of relapsing fever, while the extremes met with in the whole of the cases were within those given by Fagge for this disease. The other symptoms met with may be briefly summarised as being a sudden onset of fever accompanied by headache and pain in the back and limbs, loss of appetite, and often diarrhœa shortly before the cessation of the fever in either the first or second attack. In the interval there was an entire absence of fever, but considerable weakness and inability to do any work remained. The recurrence of the fever was also sudden, but the second attack did not usually last quite as long as the first one, and in no case did a third attack occur. After the fever finally ceased, the patients only slowly recovered their strength, being unfit to do any hard manual labour for two or three months, another characteristic of relapsing fever. There were no other fatal cases among the few villages that I was able to investigate, but several fatal cases had been reported in other villages affected, which I was unable to visit, and which extended over an area of about 20 miles, and other cases beyond these limits have since been reported.

"With regard to the infectiousness of the disease, the following figures are interesting and conclusive. In six households attacked, of which I have the figures, there were thirty persons. As soon as the first case occurred in three of these houses containing twenty persons, twelve of the occupants were removed to other houses, and all of them escaped the disease. Of the sixteen persons who remained in the affected houses fourteen got the fever and only two escaped. The incubation period varied from one to ten days after exposure to the chance of infection. All these facts again are typical of relapsing fever.

"Finally, only one patient had not had a relapse at the time of my visit, and on examining him closely he appeared to have a little fever. The thermometer revealed a temperature of 100·2°, and on examining his blood the spirillum of relapsing fever was found in small numbers, two being present in a field of an oil emersion lens in one specimen, while single ones could be easily found in a short time in every specimen examined. Inoculations of agar and gelatine tubes gave negative results, which is in accordance with the fact that this organism has not yet been cultivated outside the body. No monkeys were available for inoculation, so no further observations could be made on the organism. The proof of this outbreak being one of true relapsing or famine fever was then complete, and on inquiry it was found that these people were well off and had not suffered at all from privation.

"I mentioned at the commencement of this note that the above described outbreak was called 'sunjar' by the natives of these parts, and a few remarks on the question of what are 'mahamari' and 'sunjar' may be appropriate in this place. From inquiries that I have recently made in the Kumaon hills it appears that the former term is applied in these parts to any epidemic disease which is attended by a very high mortality, and under this name I have had a typical outbreak of cholera described to me by villagers. On the other hand, any infectious disease which is attended by a comparatively low mortality is called 'sunjar,' which term would probably include such diseases as measles and chicken-pox, etc., if they occur in these parts.

"From an examination of the records, it appears to be certain that some of the outbreaks of so-called 'mahamari' are exactly similar to true plague, notable those of 53-54, which spread down to the districts of Bijnor and Moradabad, and of 1876-77. It is, however, equally certain that this form of the disease may be absent from the hills for several years at a time, as for instance between the years 1878 to 1881, when, although the whole of the villages of Kumaon and Garhwal were carefully inspected yearly and a special report made, no cases of this disease were found. Further. it seems very likely from the descriptions that some of the outbreaks that have been returned as 'mahamari' have been outbreaks of typhus fever and not of plague, and that the latter are comparatively rare. Again, the outbreaks of the plague-like disease nearly always begin in the higher parts of the district, which border on Tibet, and it seems very probable that they originate in that country, which together with Yunan appears to be the home of the plague, from whence it spread to Canton and Hong-Kong in 1893, and that it is more likely that the plague reached Bombay in that roundabout way than that it travelled direct from Garhwal to the western part of India."

AMERICA.

Some Remarks on Typhoid Fever among the American Soldiers in the Recent War with Spain.

The paper on this subject, by Dr. Victor C. Vaughan, was the result of an investigation into the causes of the introduction and spread of typhoid fever in some of the regiments encamped in this country. It was stated that the true number of cases of typhoid fever was not contained in the official reports, the nature of many undoubted cases being masked by the diagnosis of malaria in many cases of protracted fever, in which examination of the blood showed the absence of any malarial hematozoa, by the diagnosis of indigestion (the mortality from which in some regiments was 15 per cent.), by the diagnosis in other regiments of dengue, and again of enteric fever as distinguished from typhoid fever. It was evident from the investigation that the majority of the men were infected when they reached the camps. More than 90 per cent. of the volunteer troops affected at Chickamauga were infected when they reached that camp. Some were infected in their State camps; others brought the infection from their homes. There was no evidence that the water supply was infected prior to the arrival of the troops. In some cases in which the same water was partaken of by the inhabitants of the neighbouring town and the encamped soldiers the latter were affected with typhoid fever, the former not. It was concluded that water infection played a very insignificant *rôle* in the spread of the disease among the troops. It was equally clear that infection came through an improper disposal of fecal matter and urine. It was impossible to walk through some camps without soiling one's shoes with fecal matter. Over this flies swarmed and subsequently infected the food. Previous intestinal disorders seemed to have little effect in causing an attack of typhoid fever. But many of the intestinal disorders diagnosed otherwise were undoubtedly typhoid fever. Another potent factor in the spread of the infection was the fact that the disease was often far advanced before its rea

nature was recognised. Methods for the proper disposal of fecal matter were recommended, and care in the selection of medical officers, as well as the delegation of more authority to them, was earnestly recommended.

In the discussion, Surgeon-General Sternberg deprecated the occurrence of typhoid fever among the soldiers, stating that it was attributable in large part to the non-appreciation on the part of the regimental officers of what they were content to designate the "fads" of the doctors relative to sanitation. In addition, the spread of the disease was to be ascribed to the non-recognition, on the part of the physicians, of the true nature of many cases of disease, and to the non-disinfection of excreta. The inadequacy in point of numbers of the medical department, even in time of peace, was also commented upon. He stated also that although most of the medical officers of the volunteer army were equal to the average physician of their neighbourhood, most of them had to learn even the rudiments of camp sanitation, and he advocated that the medical schools should devote more attention to hygiene.

Dr. Francis P. Kinnicutt, of New York, detailed some experiences at Montauk Point and in the New York hospitals, mentioning the inefficient sanitary measures. He advocated greater knowledge and greater power on the part of the medical officers.

Dr. George L. Peabody, of New York, stated that Dr. Vaughan's paper was a great indictment of the medical officers, and advocated the distribution of the paper among these officers and the adoption of a requisite to subsequent appointment that all applicants for the position of assistant surgeon in the army be required to familiarise themselves with the paper.

Dr. George Dock narrated some experiences, particularly at the Sternberg General Hospital, and exhibited several instructive charts. He spoke of the lamentable lack of knowledge of the clinical course of typhoid fever on the part of many of the medical officers coming from civil life. A great amount of quinine had been given to many of the patients with mistaken diagnoses, and although the cases continued the diagnosis was not altered. The protracted cases almost without exception were instances of typhoid fever.

Dr. Abram Jacobi deprecated the appointment of incompetent regimental surgeons by the governors of States and the necessity of their acceptance by the surgeon-general.

Dr. D. W. Prentiss, of Washington, asked if any ill effects had been observed from large doses of quinine.

Dr. Dock replied that sometimes such had been observed and sometimes not. Large doses at times seemed to depress the mind and disorder the stomach.

Dr. S. Solis Cohen, of Philadelphia, spoke of a Pennsylvania regiment in which satisfactory sanitary measures had been ordered by the regimental surgeons, and faithfully carried out by the regimental officers, and no typhoid fever developed. In other regiments sanitary measures were not carried out and typhoid fever developed. He also spoke of the occurrence of mixed typhoid and malarial infection. In camp all cases of fever should be considered typhoid fever until proved otherwise. If a case of fever does not yield to two hypodermic injections of fifteen grains each of the double hydrochlorate of quinine and urea it is not malaria.

Dr. Vaughan stated that the average medical officer in the volunteer army was as good as the average surgeon, but that he is powerless to prevent or cure disease unless the line officers follow his directions, and this the latter are often not disposed to do.—*Boston Medical and Surgical Journal*, May 18th.

Poisoned Arrows.

Malber and Bourgeois state that the poison used upon the arrows used by the natives in Venezuela is curare, and although sometimes mixed with other substances, this is the basis of all the arrow poisons of South America. (*Revue mensuelle de l'Ecole d'anthropologie*).—*Janus*, April, 1899.

Venin and Antivenin.

Martin has made the following calculations with regard to the quantity of antivenin required to neutralise a given amount of snake poison. If the snake poison be injected under the skin, the dose of antivenin must, if also injected under the skin, be 10 or 20 times as great as would be required to neutralise the poison in vitro. If, however, the antivenin be introduced into the circulation, then the dose is identical with that required for neutralisation in vitro. The action of the one substance upon the other is chemical, but the snake poison diffuses much more rapidly than the antidote. The writer recommends that in the treatment of snake bite by antivenin the antidote should be injected into a vein.

Prophylaxis of Texas Fever.

Texas fever is an epizootic or infectious disease of cattle, which has been long prevalent in Texas, where it decimates the herds, especially during the hot season. As early as 1869, Dodge pointed out that it appeared to be propagated by the tick, *boophilus bovis*, which infests cattle, but it was not till 1886 that the pathogenic agent was discovered by Smith and Kilborne, who succeeded in isolating a hæmatozoon resembling, though distinct from, that of malaria. They also confirmed Dodge's suggestion that the *boophilus bovis* was the propagating agent by showing that, if these parasites were transferred from infected cattle to others in regions where Texas fever was unknown, the disease soon broke out there also. There are still, however, important blanks to be filled up in the history of the parasite, for the hæmatozoon has not yet been detected in the body of the tick, nor has it been successfully cultivated. Still, enough is known for therapeutic purposes, and the 1898 *Report of the Secretary of Agriculture*, recently published at Washington, announces the discovery of a substance a solution of which will destroy all ticks on animals washed therewith. We may therefore look forward hopefully to a rapid extinction of the disease.

The Food Supply of the American Army in the Philippines.

In a report on sanitary conditions at Manila, Major H. W. Cardwell, chief surgeon First Division, Eighth Army Corps, under date March 18, 1899, writes as follows:—

"There is at present absolutely no fault to be found with the supply of food, and its preparation is excellent. Great ingenuity has been exercised by the various companies of this regiment (Fourteenth United States Infantry) in the erection of stone and brick ovens in the court-yard of the Cuartel, for use in bread-making and roasting; the tops of many being used as huge stoves. The issue of fresh beef, as regards its quality, is of the best. Companies which do not cook their own bread buy from those that do, or from regimental bakeries, and the quality is equal to that of the best American city bakeries.

"Potatoes and onions, fresh, with the usual canned goods from the commissary and such green produce as can be obtained in the markets, supply an ample vegetable ration. The issue of rice in additional quantity by the commissary is appreciated, and, strange to say, those most liable to complain as to insufficiency of quantity are newly arrived organisations. I find that many, especially among the officers who have been here six months or more, crave and eat as much meat as they did in the United States."—*Boston Medical and Surgical Journal.*

The medical officers of the United States Army and Navy find the treatment of dysentery very unsatisfactory in the Philippines, and recommend that all patients suffering from diarrhœa, dysentery, and rheumatic affections should be sent to the United States for treatment.

News and Notes.

THE Harben Medal of the Royal Institute of Public Health was presented to Lord Lister by the President of the Society, Professor W. R. Smith, at the annual dinner of the Institute on June 7.

The new School of Tropical Medicine will not at present undertake the analysis of arrow poisons, as suggested by Mr. Hedderwick. The Secretary for the Colonies thinks the expense would be undesirable at present, as only one instance of the use of poisoned arrows in West Africa has been brought to his notice.—(*Brit. Med. Journ.*, May 20, 1899).

A case of Malta Fever, giving the serum test with the micrococcus melitensis, was reported in the United States in the *Philadelphia Medical Journal*, Dec. 31, 1898.

SEAMEN'S HOSPITAL SOCIETY AND THE LONDON SCHOOL OF TROPICAL MEDICINE.—The Committee of the Seamen's Hospital Society, at a meeting on May 26, elected the following additional officers on the staff. Physicians: R. Tanner Hewlett, M.D., M.R.C.P.; Guthrie Rankin, M.D., M.R.C.P. Surgeons: James Cantlie, M.B., C.M., F.R.C.S.; J. Brian Christopherson, M.D., B.C.Cantab., F.R.C.S. Ophthalmic Surgeon: L. Vernon Cargill, F.R.C.S. Superintendent and Medical Tutor of the School of Tropical Medicine: D. C. Rees, M.R.C.S., L.R.C.P. For the time being the Medical Officers are distributed as follows:—at Greenwich: Physicians; Drs. Hewlett and Rankin. Surgeons: Messrs. Johnson Smith and W. Turner. At the Victoria and Albert Dock:—Physician: Dr. Patrick Manson. Surgeon: Mr. Cantlie. Surgeon to out-patients: Mr. Christopherson. Mr. Vernon Cargill attends the Ophthalmological department of both Hospitals.

Professor W. J. Simpson has been elected an Honorary President of the 13th International Congress of Medicine to be held in Paris in August, 1900.

THERAPEUTIC NOTES.

BORIC ACID used in an enema has, according to Grumpelt, produced severe toxic symptoms, consisting of erythematous, papulous, or bullous eruptions on the hands, and in one case a marked dryness of the skin. Intense fever, nausea and headache were also observed. The trouble followed the use of about a pint (500 cc.) of a 1 per cent. solution.—*Merek*, May.

FOR FISSURES OF THE TONGUE.—The *Riforma medica* for April 10 gives the following formula:—

℞. Carbolic acid ... ... 22½ grains;
Tincture of iodine ... ... 75 ,,
Glycerine ... ... 225 ,,

M. For local application.

A VERMIFUGE POWDER FOR CHILDREN.—Dr. Albert Veillard (*Journal de Médecine de Paris*, April 9) gives this prescription for the destruction of round worms and threadworms:—

℞. Calomel ... ... 2½ grains;
Santonin ... ... 1½ ,,
Powdered sugar of milk ... ... 15 ,,

M. The whole to be given in the morning, before breakfast, in a coffeespoonful of honey, to a child about four years old.

THE TREATMENT OF INFECTIVE INFANTILE DIARRHŒA.—The *Journal des Praticiens* for April 22 recommends the following:—

℞. Eau sucrée ... ... 300 grains;
Wine of opium ... ...from ½ to 2 drops;
Dilute hydrochloric acid ... ... 2 ,,

M. To be given in one dose. It may be repeated several times a day, according to the indications [remembering that children are especially susceptible to opium].

TANNALBIN IN DYSENTERY.—Waedemon has used tannalbin in 200 cases of diarrhœa with excellent results. In nurslings particularly, and in very young children, tannalbin was found to be especially useful, being successful in cases where other remedies had failed. Among the 200 cases were 171 children, of whom only two died, having come under treatment too late. Among the adults equally good results were secured, 14 out of 15 cases of dysentery being cured. Excessively frequent and bloody stools in a case of typhoid were immediately checked by the tannalbin. The best results were obtained by recurrence to large doses. To nurslings, 3 gm. (45 grn.) were given daily, administered in a mucilaginous vehicle; adults received up to 10 gm. (2½ dr.) even, daily, without the slightest inconvenience.

The author recommends tannalbin in all cases of infantile diarrhœas, and believes it to be a veritable specific, and much superior to the remedies ordinarily employed in this class of affections.—*Merek's Archives*, March, 1899.

HÆMOGLOBINURIC FEVER of miasmatic character has caused considerable disagreement among clinicians on the question of treatment, especially on the use of quinine, which certain authors consider even to be the cause of the hæmoglobinuria and hæmorrhages from the mucous membranes. Whatever may be thought of it, it is quite certain that quinine alone is powerless to provoke "black fever." Dr. F. Du Bose (of Selma) has found that by associating quinine with morphine and atropine, and administering calomel in large doses, all threatening symptoms of hæmoglobinuric fever disappear, and cure rapidly follows. As soon as the diagnosis is made out, he makes an injection hypodermically of the following solution:—

Quinine bisulphate ... ... 2½ grains.
Morphine sulphate ... ... ⅛ ,,
Atropine sulphate ... ... 1/200 ,,
Boiled distilled water ... ... 30 min.

Use for one injection, in the thickness of the muscles. Repeat every eight hours.

If the patient cannot be seen often, give in powders, as follows:—

Quinine sulphate ... ... 4½ grains.
Camphor monobromide ... ... 1½ ,,
Morphine sulphate ... ... 1/13 ,,
Atropine sulphate ... ... 1/100 ,,
Capsicum powder ... ... ½ ,,

One powder.

Take one such every four hours. The morphine and atropine may be increased if necessary to the amount used hypodermically. From the first calomel is given in a dose of 9 to 10 grains. Then at intervals of two hours, four or five doses of 4½ grains calomel, which causes abundant diuresis without causing excessive purgation or ptyalism.

If fever is high, the following powders:—

Phenacetine ... ... 2½ to 4½ grains.
Caffeine ... ... ½ ,,
Sodium bicarb ... ... 1 ,,

One powder. Take a similar powder every two hours till temperature is favourably lessened.

Review.

THE NURSE'S AND CLINICAL CASE BOOK, WITH DAILY AND FOUR-HOURLY TEMPERATURE CHARTS. London: John Bale, Sons & Danielsson, Ltd., Great Titchfield Street. Price 6d.

The firm of John Bale, Sons & Danielsson have done so much in the direction of facilitating clinical reports that we welcome their new "Clinical Case Book." On ample sheets provision is made for exact reports by the nurse, not only as regards the condition of the patient, but also for recording the amount of the various kinds of food and stimulants administered during every twenty-four hours. The temperature charts are a model of clearness and precision. The only suggestion we would venture upon is that the separate leaves of the book should be perforated, that they may, if so required, be readily removed and transferred to the physician's case book.

Correspondence.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—I would suggest that the "second body" joined at right angles to the main body of a guinea worm, observed by Dr. W. M. Elliott, was part of the uterus of the worm which had prolapsed through a wound of her musculo-cutaneous structures. Similar appearances are readily produced in many nematodes. The body could not have been the male worm in coition, as, in the first place, the male worm has not hitherto been discovered; and, in the second, his presence at the stage at which the female guinea-worm has attained when she appears at the surface of the body is physiologically unnecessary, her uterus being then packed with embryos at the term of their intra-uterine life and she herself about to die.

I am, &c.,

PATRICK MANSON.

May 19, 1899.

Birth.

CANTLIE.—On June 15, at 46, Devonshire Street, Portland Place, London, W., the wife of James Cantlie, M.B., F.R.C.S., of a son.

Communications, Letters, &c., have been received from:—

C.—Dr. Closky, Selangar; Dr. P. T. Carpenter, Brit. Honduras; Dr. J. T, Clarke (Perak).
D.—Major T. A. Dixon (Punjab).
E.—Dr. F. G. H. Edwards, Royal Colonial Institute; Dr. W. M. Elliott (Gold Coast).
H.—Staff-Surg. P. B. Handyside (Simon's Bay).
J.—Dr. A E. Jerman (Uganda).
P.—Fleet-Surg. J. Porter, R.N. (Cape Station).
R.—Dr. Duncan J. Read (Shanghai).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents.'

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Original Communications.

YELLOW FEVER IN WEST AFRICA.

By W. M. Elliott, M.D.(R.U.I.), M.Ch., F.R.C.S.E.
(Gold Coast Medical Service).

The subjects of malarial and blackwater fevers have recently occupied a good deal of space in the columns of The Journal of Tropical Medicine, and it may not be out of place to draw some attention to another even more deadly fever which crops up now and then in certain parts of the British Empire.

There are many points in the pathology of malarial disease which are still *terra incognita*, and the application of the microscope to the exact diagnosis of malarial fevers is not yet adopted universally. The result is a tendency among practitioners in tropical climates to include all fatal cases of fever of uncertain nature in the category of pernicious malarial fevers. This is particularly the case in regions where, as in West Africa, malaria is constantly seen in its worst forms. Some time ago, in conversation with an eminent authority on tropical diseases, I mentioned that I had seen many cases of yellow fever on the Gold Coast. He received the statement with polite incredulity, but asked for some details as to the symptomatology and *post-mortem* conditions of the cases I referred to. My replies seemed to incline him to my belief that yellow fever is indigenous to the Gold Coast. There are but few references in recent English medical literature to the occurrence of this disease in West Africa—apart from brief bald statements in text-books—and it is with a view to directing attention to a fact which in recent years seems to have been ignored, that I venture to publish these notes.

There is an old doctrine that yellow fever had its original home on the West African coast, and that its existence in the tropical regions of the American continent and in the West Indies is the result of the slave traffic. On the other hand, certain French authors, notably Beranger-Ferraud, contend that the disease was first imported into Africa from America. However this may be, there is no doubt about the fact that yellow fever has frequently shown itself in epidemic form in Senegambia, and that the British possessions on the West African littoral are liable to outbreaks of a peculiarly malignant form of fever which is confined to the European residents, and which, after thinning their numbers to an appalling degree, and within a brief period, suddenly disappears.

I cannot speak from personal experience of the epidemics which from time to time have scourged the European populations of the colonies of Lagos and Sierra Leone; but of equally fatal outbreaks on the Gold Coast I have had sufficient experience to enable me to come to a definite conclusion as to their nature.

My four years of professional experience in this colony have been limited, practically, to its western province, and during that time I have seen many Europeans die; but of those who were under my own immediate care I am convinced that in only three cases could the cause of death be certified as malarial fever. In all the other fatal cases of climatic fever the cause of death was, "to the best of my knowledge and belief," yellow fever. I may, perhaps, mention the immediate cause of the fatal issue in the cases which I attributed to malaria, viz., one, hyperpyrexia; one, suppression of urine; one, acute nephritis following hæmoglobinuric fever. And at the mention of blackwater fever one naturally thinks of the various points of diagnosis between this disease and yellow fever which have been laboriously tabulated by various authors. But to those who have seen both diseases the diagnosis presents no difficulty when the condition of the urine and skin, and the nature of the almost inevitable vomit, are considered.

Some of the cases that I have met with corresponded in every detail to the excellent description of yellow fever given by Surgeon-General Sternberg, in Davidson's "Hygiene and Diseases of Warm Climates." Unfortunately, I have not the notes of these cases at hand, but the following account of three cases which occurred at a small station within a period of five weeks, although perhaps not in every respect typical, will serve to illustrate the yellow fever of West Africa. It will be noted that quinine was freely administered in these cases. The bogey of malaria is ever present to the mind of the medical practitioner in West Africa, and he regards quinine as the sheet anchor of his treatment in most cases of fever of any description. But in the cases under consideration nothing was more obvious than the absolute uselessness of this drug. I should also mention that the European residents of the station at which these cases occurred numbered only eleven, so that the European mortality from this form of disease was 27 per cent. within five weeks.

Case I.—European mercantile agent; age about 24; has been about three months on the coast, and three weeks at this station; first attack of fever.

1st day.—Acute onset in the evening, with severe headache and vomiting. Temperature 103°.

2nd day.—M.T. 102·4°; has not slept; stomach very irritable; bowels opened freely by aperients; skin dry. E.T. 103·4°; pulse 104; quinine 20 grs. 10 p.m., temperature has fallen to 102°, and there is some deafness (cinchonism).

3rd day.—Still sleepless, and vomiting nourishment and medicine; marked congestion of conjunctivæ. M.T. 104·4° and rising. Gave antifebrin to modify temperature, but although latter fell to 102·4° at noon, the drug produced cyanosis with drowsiness and bilious stools. In the afternoon 20 grs. quinine. E.T. 104°; pulse 100; still vomiting at intervals; some cinchonism; no specimen of urine yet available for examination; cold sponging at intervals.

4th day.—M.T. 100·4°; pulse 84, soft and weak; cyanosis has quite passed off; has passed a sleepless night, having vomited sedative draught. There is no headache, and the mind is quite clear; tongue more moist and a little cleaner. Throughout the day temperature stood practically at 104° in spite of the internal administration of phenacetin, and the application of a cold water sheet. Towards evening vomiting was not so troublesome; kept down some chicken soup and 15 grs. quinine; had a hypodermic of strychnine and 30 grs. of sulphonal.

5th day.—M.T. 104°; pulse 82; has slept a little after the sulphonal; tongue cleaner; lips dry. E.T. 104°. During day had 25 grs. quinine, which produced cinchonism, but did not affect the temperature. Passed a fair quantity of bile-coloured urine, and three black liquid stools. About 9 p.m. mind began to wander.

6th day.—6.30 a.m., copious flow of bilious urine. M.T. 103·4°; pulse 105, weak and unsteady; slight icterus; black vomit. 11 a.m., a tarry motion. In the evening black vomit recurred three or four times; hiccup set in, also free perspiration; patient was now comatose, and died at 9 p.m. The icterus rapidly deepened after death.

Note the pulse-temperature ratio on the 4th and 5th days.

Case II.—European; mercantile clerk; about nine months on coast, and about two months at this station.

1st day.—Seized with shivering about 9 a.m. Temperature 105·2°; pulse 126; no severe headache or pains in back or limbs; face puffed and conjunctivæ congested; urine non-albuminous; tongue moist, and slightly coated; gave phenacetin and a strong purgative; perspired freely, but bowels were obstinate. 5 p.m., temperature has fallen to 101°, but is rising again. 9 p.m., bowels have acted; temperature 104°; free perspiration set in.

2nd day.—M.T. 105°; pulse 108; bowels acted again during night; repeated phenacetin, and during day patient took 24 grs. quinine. E.T. 100°; pulse 120, weak; stomach very irritable.

3rd day.—At 4 a.m. temperature was 103°. 9 a.m., temperature 101·4°; pulse 90; had slept a little; vomiting had ceased; bowels not opened during night. Noon, temperature 102·4°; slight conjunctival icterus. 7 p.m., temperature 99·4°; pulse 84, very weak; took 25 grs. quinine during the day.

4th day.—9 a.m., temperature 100°; pulse 88; no vomiting; no epigastric tenderness. Patient said he felt "all right," but there was a slight primrose tinge all over his body, and my estimate of his condition differed from his own. No specimen of urine obtained. 4 p.m., temperature 100°; pulse 100; has had one small motion, non-bilious and of a greyish colour; quantity of urine doubtful, no specimen kept; jaundice well marked, and some tenderness over liver area; has had 25 grs. of quinine; latter to be discontinued; was treated with calomel and saline aperients, and sinapisms over liver. 6.30 p.m., copious dark coffee-ground vomit. 7 p.m., hot pack. 10 p.m., temperature 101°; pulse 104; no motion, urine or vomit since last note. 11 p.m., copious black vomit. 11.20 p.m., comatose; black vomit; fæces discharged unconsciously; patient did not recover consciousness; black vomit recurred; liver could be felt reaching down nearly to umbilicus. Death took place on the *5th day*, without convulsions. There had been suppression of urine since early on the 4th day, but *in articulo mortis* a little normal-looking urine escaped.

Note.—The remittent course of the temperature suggested malaria, but the onset of icterus without hæmoglobinuria or bilious vomit was against any recognised form of malarial fever, and the occurrence of the typical black vomit made the diagnosis clear.

Case III.—European; mercantile agent; age about 25; had been about two years on West Coast, and had suffered from several malarial attacks, in some of which I had attended him.

1st day.—Found him with well marked febrile symptoms; ordered aperients and phenacetin; bowels were well opened.

2nd day.—M.T. 105·2°; pulse 124; tongue and breath very foul; skin slightly moist; gnawing pain in epigastrium; respiration rapid and shallow. E.T. 103°; pulse 104; has had small motions, with a little urine (not preserved); respiration as before; lungs clear; frequent vomiting. Treatment: calomel, phenacetin, quinine, mixture of hydrarg. perchlor. and soda bicarb., sinapism to epigastrium. 11 p.m., has passed two ounces of urine, dense, opaque (urates) and highly albuminous; temperature 104°; skin dry.

3rd day.—Has had frequent small stools during night; says he also passed urine; voice very weak and tremulous; tongue moist, with a dark fur; still has gnawing pain in epigastrium, and there is vomiting when anything is swallowed; temperature 102°; pulse 98, and soft; respiration as before; slight general icterus, which deepened during the day; motions watery and non-bilious; urine, two ounces. 9.30 p.m., skin and hands cold; temperature 100·2°; pulse 100; motions tarry. 11 p.m., first black vomit.

4th day.—At morning visit found temperature above normal, and pulse very feeble; patient did not vomit again during night, and passed no urine, but occasionally a black liquid stool; voice very feeble; mind clear; icterus not any deeper than yesterday; suppression of urine continued throughout the day; pulse became gradually weaker; patient could take nothing without bringing on black vomit, and death took place about midnight without convulsions, and preceded by a very short comatose stage.

Two of these patients occupied houses in the centre

of the native town, and the residences of all three were close to the banks of one of the most pestilential lagoons to be found along the Gold Coast littoral.

It is almost superfluous to discuss the diagnosis of these cases, but as bearing upon the point the following clinical features, among others, may be noted:—

(1) Cases I. and II. were new arrivals.

(2) Late appearance of icterus, a symptom which in hæmoglobinuric fever and the so-called "bilious remittents" is of early onset.

(3) The "black vomit" to be in no way confounded with any "bilious vomit," such as is met with in malignant forms of malarial fever.

(4) The character of the motions in the late stage of the disease, viz., either "tarry," indicating hæmorrhage in the upper part of the gastro-intestinal tract; or of a grey colour, without bilious staining, indicating collapse of the hepatic function.

(5) In two of the cases blood films were examined (under a $\frac{1}{12}$th inch immersion objective) for malarial parasites, with a negative result.

(6) Quinine useless.

To many, an attempt to establish the existence of yellow fever in West Africa may almost seem like endeavonring to prove an axiom, but there are, on the other hand, many practitioners in West Africa itself who are exceedingly sceptical as to the existence of this disease in their immediate neighbourhood.

I do not propose, in the present paper, to discuss any of the interesting problems which the history and pathology of yellow fever suggest. My immediate object is to direct attention to a fact to which, in my opinion, sufficient weight is not at present given in estimating the dangers to which Europeans are exposed in this climate, the fact, namely, that yellow fever is indigenous in certain low-lying districts of the Gold Coast.

THE CLINICAL ASPECTS OF BERI-BERI.

By P. T. Carpenter, M.R.C.S., L.R.C.P.Lond.

(*Late Medical Officer to Sundry Tobacco Plantation Companies in British North Borneo and to the Government of British North Borneo, March*, 1889-1892.)

In contributing the following my hope is that it may be found of some interest, at least to those practitioners in the tropics who have not had many opportunities of observing cases of beri-beri.

It is not a description of any individual case; it is intended to be a composite picture of beri-beri as I saw it in Borneo. Most of it was written when I was actively engaged treating the disease amongst the Malay and Chinese coolies working in the tobacco plantations of British North Borneo, and I have thought it as well to avoid all but the most necessary alterations, preferring rather to leave it as a simple record of my three years' experience.

Beri-beri is a very fatal disease, characterised especially by general dropsy, paralysis, and tendency to dyspnœa, by muscular tenderness, loss of electrical contractibility and absent knee-jerks.

It is favoured by privation and exposure in a tropical climate, especially in low, swampy and malarial districts. It attacks chiefly the poor and low-conditioned.

The disease commences insidiously with very few exceptions. The clinical history is generally as follows: A month or so before, the patient has probably had repeated attacks of malarial fever which have left him weak, and in bad general health. He had noticed some puffiness of the face and feet, cramps, twitchings and jerkings in the legs at night; he was easily fatigued, and suffered from shortness of breath on exertion. There was a general loss of power, but he was still able to do his work by exerting himself. If seen at this early stage the medical man will notice beyond the puffiness a peculiar cachectic appearance, difficult to describe, but hardly to be mistaken when once seen. This differs, of course, with the natural colour of the patient's skin. The patellar reflexes will not yet be found absent. These premonitory symptoms frequently subside for a time, for a week or even a few months. At the end of this time the patient usually seeks advice. He now comes complaining of weakness and pain in the legs, and difficulty in walking, though he is able to walk tolerably well and with certainly no characteristic gait except a little stiffness, which might be due to almost anything. He will tell you that he has a difficulty in walking on account of a "dragging pain"—the Malays invariably use the expression "tārek-urat," *i.e.*, the nerve (urat) is stretched (tarek). He will point to the popliteal regions as the particular part where he feels this stretching. He has generally swollen feet and ankles, perhaps a slight general œdema. He complains, besides, of a shortness of breath and palpitation, a painful tightness in the epigastric region, and a swollen belly. Night-blindness is not at all uncommon. He has "pins-and-needles," a tingling and a numbness in the lower extremities; sometimes a burning sensation in the throat. Marked drowsiness, reminding one of the sleeping-sickness of Africa (so-called), invariably accompanies the above symptoms. The patient, moreover, feels a general sense of heaviness and weight.

On examination, most probably the patient will present a fairly normal appearance, with the exception of the peculiar cachexy and a slight general œdema, which latter is by no means always present. There may be some aphonia, but this, generally, not till later. Anæmia is the exception rather than the rule. There is always present a marked tenderness of the muscles and even of the bones of the calf of the leg. There is also unmistakable loss of power in the limbs. The grasp is feeble, frequently more on one side than the other, but the paresis of muscles in the anterior tibial and femoral groups and the extensors of the forearm is always symmetrical. These muscles have lost their power of response to the faradic current. The galvanic current (I always used a Storher's battery) gives the following reactions, not the true reaction of degeneration:—

$$\left.\begin{array}{l}\text{A.C.C.} > \text{K.C.C.}\\ \text{A.O.C.}\\ \text{K.O.C.}\end{array}\right\} \textit{Nil.}$$

The knee-jerks, which in the earliest stages were present or even a little exaggerated, are now absent.

Frequently one side is lost before the other. Ultimately, at any rate, in every case of beri-beri that I have seen, and I must have seen hundreds, the knee-jerks are always absent. On the other hand, the superficial reflexes are invariably present. Cutaneous sensibility is impaired in the feet and legs. I have even seen complete anæsthesia in both lower extremities, the rest of the body being unaffected. One case I remember of complete hemi-anæsthesia, and I was much puzzled to account for it as there were none of the concomitant signs of hysteria present. Impaired sensibility only is, however, the rule; a numbness of the feet and ankles, and a feeling, when the patient walks, as if he were treading on wool or soft mud. No special nerve tenderness is present, though pressure in the popliteal space causes pain. The gait is not even yet characteristic, although the patient walks as if he were very feeble, and, moreover, he frequently staggers. The organs of special sense are rarely affected. The functions of the brain are intact in all cases, and there is no tremor, nor is there any tremor throughout the whole course of the disease.

The digestive system, save for some slight anorexia and the almost constant complaint of swelling and tightness in the epigastric region, which is noticed to be swollen, is normal. The tongue is clean and moist and the taste is normal; the bowels regularly moved, frequently a little too often. Constipation is not common in these cases, and I mention this point because costiveness is described as a more or less constant symptom in anchylostomiasis, a disease formerly often confounded with beri-beri. The fæces are generally dark-coloured and soft, and the ova of the ascaris, trichocephalus and anchylostoma, are found occasionally, the former frequently, the latter rarely, and then only in small numbers.

The genito-urinary system I have never found deranged; never albumen, never sugar, and the quantity normal.

The circulatory system as yet presents nothing very noticeable. The heart perhaps commencing to be hypertrophied. The pulse low-tensioned, small, slow and weak. A pulmonary hæmic murmur at left base is commonly found early, however, as is also a reduplication of second sound at aortic cartilage.

The respiratory system, except for the subjective complaint of constant shortness of breath, is fairly normal. Air enters freely, and there are no physical signs of disease.

The cutaneous system is likewise normal. Skin is perhaps too greasy and perspires less than it should, but nothing in the way of an eruption or serious skin lesion is present.

This state of affairs may continue for a short time, or some improvement may take place; the œdema may subside, and often does, and the general health may slightly improve. But soon, it may be in a day or two, it may be a week or two, the disease makes another stride, and this advance is often accompanied by a rise of temperature or even a severe attack of fever. The voice becomes altered to a hoarse whisper, the patient experiences a sense of increased weight (*brat*, the Malay word meaning "heavy", is a very common expression of the sufferer), and he is now hardly able to walk. The gait has now become characteristic (sledge-hammer gait). The man walks with his body bending well forward in order to keep the centre of gravity over the line of support, and to prevent himself falling backwards, his common extensors of the thighs being too weak to do their work. On account of the popliteal pain, which has increased, the legs are held stiff at the knee-joint; the feet are thrown down with violence, toes touching the ground first, on account of the paresis of anterior tibial muscles, and the patient cannot dorsal-flex his feet. The gait is not a typical ataxic gait. Wrist-drop develops more or less, but in slight cases it may be easily overlooked. In bad cases a marked clawing of the hand, due to paralysis of the interossei muscles, appears. The flexors of the limbs are never more affected than one would expect from the general waste of the muscular elements. Curiously the muscular tenderness, so well marked before, now disappears. The cachexy increases. Anæmia is not noticeable necessarily even now.

The paresis of muscle now becomes a paralysis, and wasting of the special groups affected is now becoming painfully evident. The patient becomes helpless, he is unable to stand or even raise himself to a sitting posture, he is unable to feed himself, for he cannot hold anything in his paralysed hands. He cannot extend his legs, for as a rule they are contracted and rigid, the heels drawn up by the unopposed calf-muscles, and he cannot for same reason dorsal flex his feet. He can, possibly, feebly flex the thighs on the trunk. The skin continues dry but greasy, and there are no lesions or tendencies to bed-sores. Dyspnœa and the painful sense of oppression in the epigastric region, as well as over the heart, now gives great distress, and is worse in cases of marked dropsical forms. The feeling of drowsiness, noticed in the earlier stages, gives way to restlessness. The anorexia increases, and sometimes ptyalism comes on. Fluid begins to accumulate in the pericardial, peritoneal, and pleural cavities, sometimes calling for paracentesis. The brain, and other organs of special sense continue to perform their functions. The heart's action becomes violent, and signs of right heart dilatation supervene. The pulse becomes weaker and smaller until it is imperceptible at the wrist.

The respiratory system may now present morbid changes, œdema of the lungs frequently supervening.

The patient may linger in this truly pitiable state for weeks, even months, but death at last comes to release him (the large majority of cases died in Borneo, and I am taking the usual course of the disease as far as my experience goes). The patient is probably as well to-day as he has been for the last week or month. Suddenly, almost without any warning, the dyspnœa becomes urgent. The patient complains bitterly of the great burning in the epigastrium, and he frequently rolls himself from side to side in vain attempts to obtain some relief. He gasps hoarsely "Help! I am dying!" He is evidently *in extremis*, and in the greatest agony for want of breath; his face is blue. Yet air enters his lungs, and the chest and abdomen move somewhat. He struggles, according to his strength, and in a few

minutes is dead. Death is probably due to pulmonary thrombosis. The patient, unfortunately for himself, is invariably conscious to the very end. . . . As in most diseases, there are many variations in symptoms and progress of beri-beri. Nothing is mentioned in the foregoing account but what I have actually seen, though it is not to be expected that any one case will present all the symptoms. I might add, too, that nothing has been taken from books, indeed there were no books to refer to in Borneo in 1889-93 where I was stationed.

There are cases in which only very slight dropsy occurs, in which there is no excess of fluid in the cavities. These are described as distinct varieties, and dubbed "dry beri-beri." The only distinction, to my mind, between "dry" and "wet" beri-beri is in the accident of dropsy and its consequent effects. In the dry form the death agony is less and the patient lives longer. In the dropsical forms death is more rapid and the patient, being less enfeebled, struggles harder and more piteously for life. There are cases in which, from the beginning to the end, œdema is the most orominent symptom, hiding in a great measure the pther conditions. Cases in which the thorax and abdomen are loaded, or it would be perhaps better to say over-loaded, with fluid, and the whole body enormously swollen. Yet these cases do not in other respects differ very much from the description given above. The anæmia is in these cases generally more or less marked.

Other cases there are which run an exceptional course, cases of so-called "acute beri-beri." An example of this is the following:—

A strong, healthy-looking Chinaman was admitted to hospital for malarial fever and cachexy. He was four weeks in hospital before he had apparently sufficiently recovered to return to his work in the tobacco fields. It was then noticed for the first time by the Chinese apothecary, an excellent and well trained man from Hong-Kong, that the patient exhibited calf tenderness. No other symptom was noted; four days after I saw the man and he had all the symptoms of beri-beri. Two days after this he was prostrate, and the next day he was dead.

There are many cases, of course, that get well under treatment. However I propose to defer the account of such for a future occasion, when I may also have an opportunity of making some remarks on treatment, prognosis and so on, including differential diagnosis and probable causes.

A CASE OF HÆMOGLOBINURIA? IN CONNECTION WITH MALARIAL CACHEXIA AND QUININE AT BARRACKPORE.

By R. R. H. Moore, M.D., T.C.D.
Major R.A.M.C.

The patient, Gunner G., had five admissions for ague before the present illness, as follows:—Nov. 2, 1898, ten days; Nov. 19, 1898, four days; Dec. 29, 1898, two days; March 12, 1899, twenty days; April 19, 1899, nineteen days.

When admitted on March 12 he had fever of the tertian type for fourteen days. He got no quinine until the fourteenth day; he then got fifteen grains, after which the fever disappeared.

When admitted on April 19 he again had fever of the tertian type; it persisted for five days; on the fifth day he had fifteen grains of quinine and the same amount on the two following days; the fever disappeared after the first dose. He had no enlargement of the spleen, but was anæmic; he never complained of feeling ill.

He was again admitted on May 13, with a temperature of 100·4 F. The next day the temperature rose to 104·8° F.; it then ran a normal course till May 29.

He was given no quinine and had no splenic enlargement.

It was remarked that he was passing a rather small quantity of urine, so a record was kept, and it was analysed daily from May 16, as shown in the following table:—

DATE.	AMOUNT.	SPECIFIC GRAVITY.	ALBUMEN.
May.	*oz.*		
16	48	1012	0
17	20	1016	0
18	28	1020	0
19	25	1012	0
20	32	1012	0
21	44	1014	0
22	46	1020	0
23	38	1020	0
24	46	1022	0
25	32	1012	0
26	60	1014	0
27	46	1014	0
28	—	1024	0
29	24	1024	0
30	24	1026	¼
31	—	1034	⅓
June.			
1	25	1018	⅕
2	54	1012	Trace

His temperature remained normal up to May 29, when he had an attack of fever in the afternoon, temperature 102·4° F. Tne next day he got fifteen grains of quinine, ten in the morning and five in the evening. He had no other attack of fever and got no more quinine.

The urine he passed on the 30th was dark brownish in colour, and contained one-fourth of albumen. On the 31st it was still darker and contained one-third of albumen. It was better on June 1, and on the 2nd had got almost normal again. The quantity also increased. These symptoms did not recur, and he was discharged in good health on June 22.

I record these facts just as they occurred, not being able to offer any satisfactory explanation. No microscopic examination of the urine was made. He had no treatment beyond demulcents and diuretics.

ON PREVENTIVE INOCULATION.

By Dr. W. M. Haffkine.

A Discourse before the Royal Society.

(*Continued from page* 292.)

Among other forms of plague virus, which were tested by us and by other experimenters, a large number were found to be too dangerous to use; in other instances the mode of application was inadmissible in the case of men; in others, again, the effect appeared too evanescent to be of practical use.

The Properties of the Plague Prophylactic.

The immunising effect of the plague prophylactic as above described was worked out on domestic rabbits, and its actual efficiency on these animals were since verified and confirmed by a number of experimenters, by trying to infect with virulent plague protected and unprotected animals. Comparing the rabbit with other laboratory animals, such as the rat, the guinea-pig, the mouse, and the monkey, one may consider the rabbit as the one that perhaps required the least amount of protection, as its natural resistance to plague is relatively high. The most altered virus—*i.e.*, such as was rendered the most harmless of all—was found to confer on the rabbit a very considerable degree of immunity, enabling it in a few days to resist ten or fifteen-fold lethal doses of virulent plague microbes. The same treatment applied to animals of a more susceptible nature would, on the contrary, in many instances fail.

The Questions which were to be Solved by Experiments on Human Beings.

At the end of our laboratory experiments a very definite set of questions stood before us, which were to be solved by direct experiment on human beings. Those questions were: (1) Would man behave with regard to the prophylactic like the animals upon whom its protective power had been worked out? (2) If it so happens that the answer is affirmative, what would the dose of the prophylactic and the method of administering it be; and would not the dose required be so high, and the reaction to be produced so severe, or the number of inoculations to be repeated so great, as to render the treatment inapplicable to men, or impracticable? (3) How many days, counting from the date of inoculation, would it take to produce in man a useful degree of immunity? (4) How long would that immunity last? And lastly, there followed two questions, to which my experience of the anti-cholera inoculation entitled me to give a reassuring answer, but which it was necessary to verify in plague again, viz.: (5) During the period of reactionary fever and all the other symptoms produced by inoculation, will the resistance of the inoculated exposed to plague be, for the time being, reduced, or remain the same, or be increased, *i.e.*, would it constitute a danger to apply the inoculation in localities actually affected with plague? and (6) When a man who happens to be incubating the plague, or to have initial symptoms of the disease already, chances to be inoculated, would it aggravate his condition, or have no effect, or, on the contrary, help him?

Demonstration of the Harmlessness of the Inoculation.

The perfect harmlessness of the inoculation was first of all demonstrated by the officers of the laboratory, the principal and professors of the Grant Medical College, a large number of leading European and native gentlemen of Bombay, and their families and households being inoculated; and after that when, in the last week of January, 1897, the plague broke out in Her Majesty's House of Correction at Byculla, in Bombay, the option of inoculation was offered to the prisoners.

Date of Occurrence of Plague.	Occurrences in uninoculated			Occurrences in inoculated		
	Number of uninoculated present	Cases	Fatal	Number of inoculated present	Cases	Fatal
Jan. 23 to 29, 1897, previously to the day of inoculation	—	9	5	—	—	—
30/1/97, the day of inoculation — Forenoon, before inoculation	—	6	3	—	—	—
30/1/97, the day of inoculation — After noon, after inoculation	—	—	—	—	3	3
1st day after inoculation, 31/1/97	177	2	1	151	1	—
2nd day after inoculation, 1/2/97	172	1	1	150	—	—
3rd day after inoculation, 2/2/97	173	1	1	146	—	—
5th day after inoculation, 4/2/97	171	1	1	146	—	—
6th day after inoculation, 5/2/97	169	2	1	146	—	—
7th day after inoculation, 6/2/97	169	5	1	146	1	—
Total after the day of inoculation	172 uninoculated, average daily strength	12 cases	6 deaths	147 inoculated, average daily strength	2 cases	No deaths

The Experiment in Her Majesty's Byculla House of Correction, Bombay.

The Byculla Jail is a long term one. There are no children or very young people among the inmates, there being in Bombay a separate establishment, the Sassoon Reformatory, where minor criminals are sent. The prisoners of the House of Correction present a well-fed, well-clad, regularly worked, and almost as uniform a set of people as can be seen in a regiment, amongst whom one could scarcely see a single infirm or very aged individual. At the appearance of plague the prisoners numbered 346. The inoculation was introduced after nine cases of plague had already occurred, five subsequently ending fatally; there remained thus 337 individuals to be dealt with. Of these, 154 only volunteered for inoculation, and 183 remained uninoculated. On January 30, in the forenoon, before the inoculation was applied, six more cases occurred, of which three afterwards proved fatal. The inoculation was applied in the afternoon, and afterwards it was discovered that one more prisoner had already a bubo on him when inoculated, while two prisoners developed buboes in the same evening

after their inoculation. These three inoculated, attacked in the evening of inoculation, proved also fatal. After that the difference observed in the fate of the two groups, the inoculated and uninoculated, is seen from the subjoined table :—

For seven days, except the fourth, after inoculation cases of plague continued to occur among the uninoculated group, their average daily strength throughout the week being 173 ; altogether, 12 cases occurred among them with six deaths ; while in the 148 inoculated there was one case on the next day after inoculation which rapidly recovered, and one on the last day of the epidemic which recovered also.

Analysis of the Results of the Byculla Jail Experiment.

A glance at the above table will show the progress which was made in our information by that initial experiment, and how far it carried us ahead from the state of uncertainty which surrounded the question originally. The dose of prophylactic administered to the prisoners was three cubic centimetres. They all had the customary attack of fever from the operation, with the discomfort accompanying that condition—a headache in many cases, nausea, loss of appetite for a couple of days, a feeling of fatigue and lassitude, reminding one of a mild atttack of influenza, and with swelling and pain in the inoculated side. Did, however, all this make them more susceptible to the disease than were their non-inoculated fellow inmates? It is certain that the table testified unmistakably to an opposite result. Further, the incubation period in plague appears to be on the average five days, extending, however, not unfrequently up to ten. Of the twelve prisoners in the uninoculated group who developed plague during the next few days after the date of inoculation a large proportion, if not all, must have been already incubating the disease on that day ; and seeing the perfect similarity of conditions under which the inoculated and the uninoculated, who came from the same crowd of people, were living, one could infer safely that a similar group of individuals incubating plague was present among the inoculated also at the time when the inoculation was performed on them. The inoculation, however, did not aggravate their condition, as the number of inoculated who developed plague, counting from the first twelve hours of inoculation, was proportionately five times smaller than the corresponding number among the uninoculated ; and the two cases that appeared among the inoculated, one on the very next morning after inoculation, both ended in recovery. As far as that first experiment went, therefore, men behaved like the laboratory animals which derived protection from the inoculation. For communicating that protection one injection of prophylactic appeared sufficient with a dose of three cubic centimetres, which dose, however, in our subsequent operations was further reduced to two and and a half cubic centimetres. The difference in favour of the inoculated appeared within some twelve hours after the operation, but the man who was inoculated with plague on him and the two who developed clear symptoms of plague the same evening did not benefit by the operation. This completed the first information gathered with regard to five of the six questions enumerated above. No answer could be given as to the final duration of the effect of inoculation, except that the operation appeared to be useful in a localised already existing epidemic extending over seven days.

The Experiment in the Umerkadi Common Jail, Bombay.

In the next case the strictness of the conditions of the last experiment was enhanced further. This was in the second Bombay jail, the so-called Umerkadi Common Jail. The plague broke out there at the end of December, 1897, and by January 1, 1898, three prisoners were attacked and all of them subsequently died. In the interval between the operations in the two jails some 8,000 people in the free population of Bombay had already availed themselves of the inoculation. This time the whole of the prisoners, numbering 401, appeared willing to undergo the preventive treatment. In view of the novelty of the operation, however, and of our responsibilities before the Government and the public, and the necessity of demonstrating clearly the effect of inoculation, the prisoners were not allowed to undergo the treatment in a body and it was resolved that only one-half of them should be permitted to do so. The manner in which that half was selected guaranteed the elimination of all possible errors usually inherent to observations on free human communities. The population of a jail in India is gathered into several groups, the largest being the ordinary convicts divided into simple prisoners and convicts sentenced to hard labour ; then there is a group of civil prisoners (debtors), then a group of prisoners under trial, of convict warders, of cooks, of bakers, of men employed in the infirmary, &c., and a separate group of female prisoners.

On the morning of January 1, 1898, in the presence of Major Collie, I.M.S., and Dr. Leon, the medical officers, and of Mr. Mackenzie, the superintendent, and of all the officials of the jail, the above groups were brought one after the other into the jail-yard and asked to seat themselves in rows ; and after all had been so seated every second man without further distinction was inoculated, excepting two of them who did not volunteer for the treatment. From this moment the pair numbers, the inoculated, were left to live with the uninoculated under as identical conditions as were those in which they were living before. They had the same food and drink, the same hours of work and of rest, and they shared with them the same yards and building, &c.

In this case fatal attacks continued to occur in the jail for thirty days, during which time an almost equal number of prisoners, inoculated and uninoculated, were discharged from jail and thus excluded from further observation. The average daily strength of the uninoculated who remained in the jail up to the end exposed to the plague was 127, and of the inoculated 147. In the smaller number of uninoculated ten cases of plague occurred, six of them proving fatal ; while the larger number of inoculated produced three cases, of whom all recovered. In these three cases, however, in the inoculated, the character of the disease was so much mitigated that the authorities

of the Government Hospital at Parel, Bombay, where they were sent, hesitated to return them as plague cases, and the Director-General of the Indian Medical Service who examined two of them diagnosed them as mumps. They were returned as cases of plague in order that no possibility of error in favour of inoculation should be admitted.

The Experiment in the Dharwar Jail.

On the third and last occasion when the plague broke out in a jail one did not feel justified in withholding the inoculation from any of the inmates, and all of them were permitted to be inoculated. This was in Dharwar during the terrible outbreak of plague in that town and district, the news of which must have reached you even here. Five cases of plague occurred, of which one was imported, and four in old residents of the jail, and all five ended fatally. The prisoners, then numbering 373, submitted in a body to inoculation and only one case of plague followed, in a man who had been attacked two days after the operation and who recovered, being the only one who did not succumb.

The Experiment at Undhera in a Free Population.

The most carefully planned out and precise demonstration of the working of the prophylactic system in the free population which was exposed to a greater amount of infection than the prisoners in the jails was that made in the village of Undhera, six miles from Baroda. The following was the mode of operation adopted: A detailed census was made by the authorities of all the inhabitants of the place, and on February 12, 1898, when a committee of British and native officers arrived to carry out the inoculation, the people were paraded in the streets in four wards, family by family. Major Bannerman, I.M.S., of the Madras Medical Establishment, and myself, accompanied by the Baroda officials, went from one household to another, and within each inoculated half the number of the male members, half that of the females, and half that of the children, compensating for odd figures that happened to be in one family by odd figures in another. I personally and the officers who were with me, directed special attention to distributing the few sick in the two groups of inoculated and uninoculated as equally as our judgment permitted us to do. The plague, which had carried away before inoculation 79 victims, continued afterwards in this instance for forty-two days, and appeared in twenty-eight families, in which the aggregate number of uninoculated was 64, and of inoculated 71. The total number of attacks in those families was 35, and they were distributed as follows: The 64 uninoculated had 27 cases with 26 deaths, and the 71 inoculated had 8 cases with 3 deaths, thus showing 89·65 per cent. of deaths fewer in the inoculated members of the families than in the uninoculated. There were no deaths from other causes in the inoculated of the village, while among the uninoculated there were 3 deaths attributed to other causes than plague.

The subjoined figures show the number of days which elapsed between the date of inoculation and the occurrence of a death from plague in these families. The first row of figures refers to occurrences in uninoculaled members, the second to occurrences in inoculated, while the small figures show the number of deaths which occurred in each group on those days.

Deaths from plague occurred in uninoculated and in inoculated:

$3^2\ 4^1\ 5^3\ 7^2\ 8^3$—$10^3\ 11^3\ 12^1$———$15^1\ 16^1\ 19^1\ 20^1\ 21^1\ 24^1\ 32^1$ and 42^1

———9^1———12^1 and 14^1———

days after date of inoculation.

There elapsed, therefore, eight days during which 11 deaths from plague occurred among the uninoculated members of the families before the first death took place in an inoculated case. The inoculation has again acted, so to say, immediately; or, as we have adopted to generally formulate the result, has acted within the time necessary for the subsidence of the *general* reactionary symptoms produced by the inoculation.

The investigation in this village was carried out by Surgeon-General Harvey, the Director-General of the Indian Medical Service, and a committee of British and native officials. Every member of the family who survived was seen, his particulars verified from the documents, and every detail was confirmed from the registers kept at the time and from the testimony of the whole of the villagers who were present throughout the inquiry.

Experiments on a Large Scale and Averages of the Results Obtained.

I have dwelt so long upon the description of the above experiments, not because they were the largest in volume or the most striking which were made, but because they were the most precise of all and, as far as I am aware, free from any possible loophole of mistake. I made prolonged and detailed observations in very severely affected communities—of Lanowlie in a population of 700 people, and of the followers of the artillery at Kirkee, numbering at the time 1,530. Very complete data were collected by Professor R. Koch and Professor Gaffky of the German Government Commission, by Major Lyons, I.M.S., and by myself in the Portuguese colony of Damaon in a population of 8,230 individuals, during a frightful outbreak of plague there, extending over four months, in 1897. A minute investigation, extending over several months, was made by me in the Khoja Mussulman community of Bombay, numbering some 10,000 to 12,000 people, where about half of the total number were inoculated under the auspices of His Highness the Aga Khan. A most comprehensive inoculation campaign, and with widely reaching and most satisfactory results, was carried out under the Collector of Dharwar by Captain Leumann, I.M.S., Dr. (Miss) Corthorn, Dr. Hornabrook, Dr. Foy, Dr. Chenai, and others in the three adjacent small towns of Hubli, Dharwar, and Gadag, where some 80,000 people were inoculated. The latter was the most magnificent piece of work done, from the point of view of practical application of the method. With the extension of the number of inoculated the exactitude and precision of observation must, however, suffer.

A number of doubts and possible objections with

regard to many particular points arise which it is not always possible to answer with certainty. Such observations are required to enable us to judge whether the application of the method as a general measure answers to the expectations formed, but the exact extent of the results is to be gathered from such mathematically precise experiments, imitating the conditions of laboratory practice, as were those which I have detailed above. The difference in the mortality from plague between inoculated and uninoculated parts of communities was estimated to average over 80 per cent., approaching often 90, as was the case in Undhera, the lowest proportion ever observed in the experiments which I made personally having been 77·9 per cent. This was at Kirkee.

Case Mortality Reduced by 50 per cent.

A very accurate set of data were collected in almost all the larger hospitals where inoculated plague cases were admitted upon the fatality of the disease in the inoculated. These were to the effect that the case mortality among the inoculated was some 50 per cent. lower than among the uninoculated plague cases. A number of documents on this point have been collected by the Indian Plague Commission and will appear, I trust, in their records.

Minimum Duration of the Effect of the Plague Inoculation.

As to the duration of the effect of the plague inoculation the statement which can be made for the present is, that it lasts at least for the length of one epidemic, which on the average extends from over four to six months of the year. The Government of India have recognised the inoculation certificates, entitling the holder to exemption from plague rules, to be valid for a period of six months, with the understanding that if accurate data are forthcoming of the effect lasting longer, the holders will be permitted to exchange their certificates for another period without being reinoculated.

The Further Problems Pursued in the Bombay Plague Research Laboratory.

The task which the officers of the Bombay Plague Research Laboratory have proposed to themselves in connection with the prophylactic inoculation comprises now the following problems: (1) the working out of proceedings for turning out uniformly large quantities of material, and avoiding the variations due to the character of the plague microbe and to the differences in the composition of the cultivation media; (2) the further investigation of the different constituents of the plague prophylactic with a view of enforcing those which produce definite and beneficial results; (3) the possible mitigation of the reactionary symptoms after inoculation; and (4) the study of the effect of antiseptics used for preserving the prophylactic; while their most important general problems concerning plague refer to the study of the curative treatment of plague, and to the life-history of the plague microbes in nature.

The Typhoid Inoculation.

Permit me now to enter a pledge in favour of a new inoculation campaign which has been inaugurated already, and which I hope will be carried out successfully for the benefit of a large number of soldiers of this country residing in India, and of white men in general in all tropical countries. The problem of typhoid inoculation has quite a special interest for Europeans, as much as cholera has for the natives of India. Typhoid fever proved to be a more difficult disease to eradicate from military cantonments than cholera. It is possible that the explanation of that lies in what is already known of the character of the microbes of these diseases. The typhoid bacillus when subjected to different chemical and physical agents, such as acids or antiseptics, or a high temperature, or desiccation, or the admixture of other microbes, appears far more resistant than the cholera microbe. Such a character would ensure for the typhoid bacillus the existence in more varied media, under more various climates and a greater independence from seasonal or local changes than is the case with the cholera microbe. Outside the endemic area the cholera remains in one and the same place for a few weeks, and in any given part of a town often for a few days only. It is rare that it visits one and the same barrack more than once in five years, sometimes ten years, and when it occurs a temporary evacuation of the place puts a stop to the disease. The typhoid virus, on the contrary, sticks to an infected locality for years and causes a continuous incidence of the disease, for which occasionally nothing short of a complete desertion of the station is effective. At the same time, while the cholera infection seems to be almost exclusively confined to the water-supply, in typhoid fever the improvement of the water seems to leave intact a large number of other sources of danger, which up to the present time have escaped recognition. Differing thus in their life-history in nature, the bacilli of cholera and typhoid fever present important common features in the manner in which they behave in the human and animal body. The chief centre of infection in both instances is the intestinal canal, the circulatory system remaining free from invasion. When inoculated into animals both microbes admit of the same kind of transformation by passages from animal to animal, and against both immunity can be created in the laboratory by the same preparation of virus as used in the inoculations for cholera, while when examining the tissues of immunised animals the same modifications are detected in them as those observed after the anti-cholera inoculation. These considerations have led us to expect from the typhoid inoculation in man a similar protective effect as that observed in the inoculation against cholera, and seeing that the period of life during which the new-comers to India remain susceptible to typhoid fever extends only over a few years it seems that the application of the system, when properly organised, is likely to prove of a very high practical value.

Inoculation and General Sanitary Measures.

The anti-cholera inoculation, the inoculation against plague, and that against typhoid fever came to associate themselves to vaccination and represent attempts at dealing with epidemics on lines differing from measures of general sanitation. During the last

few years the question was therefore frequently debated as to the relation in which the two stand to each other. It is scarcely necessary to say that inoculation cannot be substituted for a good water-supply, the draining, cleansing, or improvements in the building of cities, or for the admission of a larger amount of light and air into overcrowded localities, for all those measures to which the nations owe such a wonderful improvement in health as has taken place during the present century. Only injustice would be done to the sanitarian by calling him in when a patient lies already on his sick bed, or when an epidemic actually breaks out in a community, and by asking him to stay the sickness or the epidemic, to improve the health of the population, so to say, while you wait. Epidemics, like individual diseases, require specifics, promptly administrable remedies, to be dealt with, and measures of general sanitation can be no more advised for arresting an outbreak of cholera or plague, than can an individual patient be directed to build for himself a new house, or to dry up the marshy lands, or to cut down the jungle round his habitation when he requires a dose of quinine to arrest an attack of fever. The part of vaccination and of preventive inoculation in combating epidemics stands in the same relation to general sanitary measures as therapeutics and the art of the healing physician do to domestic hygiene. It is certain that neither of these can ever be substituted for the other.

CONCLUSION; THE OFFICERS WHO ASSISTED IN THE BACTERIOLOGICAL INVESTIGATIONS IN INDIA.

My Lord and Gentlemen, permit me, before I leave this place, to pay a tribute of gratitude for assistance and coöperation in the investigation work in India to the officers of the Indian and Bombay Governments, the Director-General of the Indian Medical Service, Lieutenant-Colonel Owen, Major Bannerman, Major Lyons, Major Herbert, Captain Thorold, Captain Hare, Captain James, Captain Earle, Captain Milne, Captain Leumann, Dr. (Miss) Corthorn, Dr. Gibson, Dr. Marsh, Dr. Balfour Stewart, Dr. Ransome, as well as Dr. W. J. Simpson, Dr. Powell, Dr. Mayr, Dr. Surveyor, Dr. Paymaster, Mr. E. H. Hankin, the distinguished Bacteriologist of the North-West Provinces of India, His Highness the Aga Khan, and a number of other European as well as Indian gentlemen, much too numerous to mention them all.

Dr. ROSE BRADFORD said that, as Professor Haffkine had alluded to the inoculations against typhoid which were begun in this country by Professor Wright, it would be interesting to the meeting to hear what Professor Wright had to say on the subject.

The PRESIDENT (Lord Lister) then called upon Professor WRIGHT, who said that his position on the Indian Plague Commission bound him to silence until the publication of the Commission's report. All he would allow himself to say at present was that the experiments and results which Mr. Haffkine had brought before them in his speech were brilliant; whether the results were always as brilliant was a different matter. But he, in his position as a member of the Plague Commission, could hardly be expected to touch on that at the present moment. This much he could, however, say, that if the results were less brilliant the question would have to be considered whether Mr. Haffkine always used the best methods to secure the best results. Other important questions might also have to be dealt with. Mr. Haffkine's work in India had only a relative interest. He had undoubtedly saved a certain number of lives in India. But what was of far greater importance than the question as to whether six inmates of the Byculla Gaol had been saved or six had died was the acquisition of scientific knowledge which would render it possible to supply the inoculation method against other infectious diseases. This information had not been forthcoming. No doubt Mr. Haffkine had done a great deal of good by his inoculations, and if any harm had ever occured it had been out-balanced and overbalanced by the good. He would have liked to have learnt what was the effect of the individual constituents of the prophylactic, and whether they were all necessary. Dr. Rose Bradford had alluded to the typhoid inoculations which he (Professor Wright) had performed, and he would say that Mr. Haffkine's work in India had popularised the inoculations and rendered his task easier, because wherever he went Mr. Haffkine had been previously. But no information had been given as to the preparation of the vaccines, the dose to be used, and the methods to be employed. This was all he felt justified in permitting himself to say at present.

Surgeon-General HARVEY (Director-General of the Indian Medical Service), who was called upon by the President, said that people in England should consider that there were enormous difficulties in the way of a bacteriologist in Indfa. There was a great difference between a research in a laboratory at home and a campaign against a plague epidemic in India. Till lately there as no apparatus to sterilise corks and bottles, and with the large quantities of vaccine required there were immense difficulties in enlarging the work as rapidly as the epidemic necessitated, while at the same time ensuring in its manufacture perfect asepsis. Notwithstanding these difficulties, the results were such that the medical officer in charge of the operations at Hubli, who did nearly 80,000 inoculations, had only observed some twelve abscesses. Only twelve abscesses in 80,000 inoculations showed good work. He had himself been inoculated. The effects were extremely unpleasant, but still he was able to do his work as usual. The fever disappeared in the course of a day or two, but the arm remained painful for a week. The questions raised by Professor Wright were being attended to. They all required time. Experiments were being conducted in the Bombay laboratory to standardise the prophylactic and turn out larger quantities of uniform strength. At present the laboratory was turning out 10,000 doses a day; but what was this quantity compared with the 300,000,000 inhabitants of India? The Government of India had given Mr. Haffkine the assistance he asked for, and were prepared to give him more. The Indians were a strange people. In Calcutta the mention of inoculations drove 300,000 people from

the city. They afterwards returned and many of them got inoculated. In another part of the country he had himself seen them coming in their hundreds to be inoculated, and paying two annas each for the privilege. He felt, however, that even if the whole population of India were to make up their minds to be inoculated, neither the material nor the doctors to carry out the inoculations could be provided, and what was really necessary was sanitary improvements which prevented epidemics. As to the accuracy of Mr. Haffkine's results, he himself had investigated the experiment at Undhera, and, as far as he could judge, there was no possibility of error. The difference in the mortality was nearly ninety per cent. in favour of the inoculated. There were some interesting instances of families in whom the few that were not inoculated all died while none of the inoculated suffered. He had no doubt as to the good done by the inoculations. The difficulty chiefly lay in the application of the system; however, he was glad to say that these difficulties were being gradually overcome.

Professor W. J. SIMPSON, who was next called upon by the President, said that he gladly accepted the invitation, as it afforded him an opportunity of expressing the admiration which he felt for Mr. Haffkine and his work. For a considerable period he had the honour of being associated with him in a very secondary position in his earlier work on cholera inoculations in India, and that association made him familiar with the stupendous difficulties which Mr. Haffkine encountered, and which his untiring energy had overcome. It also gave him an insight into the caution and unbiassed character of the man. It often seemed to him that Mr. Haffkine was the greatest antagonist to his own results, criticising every fact in favour of the inoculations from the most adverse point of view. He mentioned this because, from personal experience, he could testify to the solidity of the basis of the earlier observations on cholera, and he knew from what he had seen at Kirkee, near Poona, in 1897, that the same rigidly careful methods of verification of facts were also applied to the plague inoculations. There was no question in his mind as to the solid foundation on which the results rested. For himself he confessed that facts and results were more attractive than theoretical considerations, and that he looked to the results obtained by the inoculations as more important than the questions raised by Professor Wright. As regards dosage and preparation of the vaccines, these were matters of history. The one main fact brought out by the investigation was that wherever inoculations of cholera or plague were carried out, and cholera or plague prevailed, the incidence on the inoculated was always less than on the uninoculated. That was the major fact connected with the inoculations, and it was not merely a small matter of a few lives here or there being saved. Of course, there were other facts of minor importance that needed to be worked out. None, he knew, were more conscious of this than Mr. Haffkine, and none more anxious for improvements. Given the opportunities, Mr. Haffkine would, no doubt, go on improving the system which he had introduced.

The PRESIDENT said that the information he had collected before Mr. Haffkine's return from India had convinced him of the efficacy of the inoculations, and, if anyone present had doubts before the meeting, he was sure that after listening to what had been said all their doubts must have been removed. As to further progress in this question, no doubt in this case as in others progress was possible, and some requirements, such as those mentioned by Professor Wright, might be necessary; but, whatever progress was possible, he was convinced that the best man in the world to attend to these requirements was Mr. Haffkine himself. He was also glad to hear from Professor Wright that Mr. Haffkine's work had prepared the way for the typhoid inoculations. Mr. Haffkine had mentioned that some 80,000 inoculations had been performed in the Dharwar district. If any proof were necessary that with tact and patience the inoculations could be applied on a large scale, no better instance could be adduced than this one. The people in that district not only came forward in thousands to be inoculated when the efficacy of the method was explained to them, but they paid for the inoculations. The good done by Mr. Haffkine in India reached beyond the saving of thousands of lives, for it convinced the people of India that the Government and Europeans were trying to do their best for them, and thus his work tended to break down the barrier that existed between the British and the people of India.

Mr. HAFFKINE, in reply, said he was very sensible of the kind words of the President and of other speakers about his work and himself, and of the manner in which they had been received by those present at the meeting. As to the interesting questions raised by Professor Wright, some he was able to answer, others he was not. The latter would probably provide work for Professor Wright and others. As to himself he could only take the responsibility for the little he had done, and asked to be excused for what he had not done.

TRACHOMA AND RACE.

By Dr. EDWIN VAN MILLINGEN,

Premier Oculiste aud Auriste du Palais Impérial, Professeur d'Ophthalmologie à l'Ecole Impériale de Médecine.

THE able article by Mr. M. J. Yarr on "Trachoma and Race," which appeared in the May number of the JOURNAL OF TROPICAL MEDICINE, deals with a subject which has attracted my attention for many years, and I was much pleased to see it brought forward in one of the first numbers of a paper which has such a brilliant future before it.

As the subject is likely to be discussed in your columns, I am axious to remove any misunderstanding which Mr. Yarr's statement may convey to your readers as regards my views on the subject, and I shall attempt to show that my reasoning is not so difficult to follow as Mr. Yarr supposes.

The point at stake is whether the negro is subject to trachoma or not.

That the Soudan negro is liable to contract trachoma has, I believe, been sufficiently proved by my statements in the paper to which Mr. Yarr refers.

In a subsequent paper read before the twelfth Congress I further showed that 70 per cent. of the negroes in lower Egypt suffer from trachoma. The negro in his native country does not suffer to the same extent, but he readily contracts the disease in coming to Lower Egypt. I quote the following from the papers alluded to :—

Examination of negroes in Egypt.

	Found healthy, i.e., without trachoma.
Of 100 negroes born in Egypt	30
,, 100 ,, resident in Egypt from 4 to 30 years..	50
,, 355 negro women, wives of Soudanese soldiers	219
,, 117 negro children (Soudanese) of above women, with trachoma	37
,, 390 negroes (Soudanese, all from South Soudan)	336
,, 33 negroes East Soudan	29
1095	701

The total number of trachoma cases was therefore 394.

With this evidence as a basis, it was only natural that I should be sceptical in believing that the West Coast negro is immune, and this doubt has only been encouraged by failures in the attempt to prove the immunity of certain white races (Chibret).

As regards the American negroes (originally West Coast negroes) I attributed their apparent immunity to their living in a country where trachoma is not of frequent occurrence. Any evidence showing that negroes in America have been known to suffer from trachoma must disprove their immunity, however small the number of cases.

It appears to me therefore that it is logical to take the evidence of those who *have* seen cases of trachoma on blacks, and to neglect the statements of those who *have not* seen any such cases. The evidence given in my paper in support of the possibility of trachoma affecting the American negro is not as insignificant as Mr. Yarr makes it out to be. I may quote the following from my paper.

"Prof. Gama Piuto :—'I have seen *here* (Lisbon) and at Rio Janeiro some cases of trachoma in negroes presenting all the characteristic appearances, pannus, shortening of the conjunctiva.'

"Dr. Moura, of Brazil,[1] an oculist at Rio Janeiro, assured me that he had seen many negroes suffering from trachoma (meaning in Brazil, of course).

"Charles Finlay, of Havanna :—'Trachoma is of rare occurrence amongst negroes; ten cases throughout the year in a population of 460,000 negroes, whereas the proportion among whites was 660 cases in a population of 1,010,000.'

"Professor Knapp :—'The negro is seldom subject to trachoma.'"

This, it appears to me, is evidence that the American negro may contract trachoma; there is therefore no absolute immunity.

A naval physician who had visited the West Coast, and whom I questioned on the subject, asserted that trichiasis and pannus is frequently seen amongst the Kroo boys.

Absolute immunity in the American negro being out of the question, it remains to be seen whether his apparent immunity is characteristic to his race, or dependent upon other causes, for which the investigation is well worth trying. The Italian, Polish and Irish emigrants carry trachoma with them from their country. In foreign countries they live under the same roof with their own countrymen, certainly never with blacks. Trachoma is not so highly contagious as to be transmitted by mere approach. Living *under the same roof* is most favourable and usual for its transmission.

Those who may more readily transmit it (abundant secretion) keep to their rooms, and chiefly those in immediate contact with them are most exposed to contagion.

The term "mixed communities" *in the sense of living under the same roof* can never be applied to blacks and whites in America. In the upper classes when the servants are blacks, the hygienic conditions exclude the possibility of trachoma; the same applies to the eunuch of the harem as regards opportunities of contracting trachoma.

Even the poorer white classes in America do not live under the same roof as the blacks.

In visiting Egypt for this particular study I was struck with the fact that no cases of trachoma were to be seen amongst the English soldiers of the army of occupation. I inspected a hundred men at the hospital and a hundred at the barracks; all these men had been in the country for more than five years. (The natives suffer in the proportion of 75 to 90 per cent. of the population.)

Was I to infer that it was owing to immunity that these men had been spared?

The experiences of former expeditions (1800) proved well enough what havoc trachoma could make on soldiers of the same race in times when hygiene was neglected because unknown.

From this point of view, therefore, I repeat that it is desirous to make a very full and careful investigation before "safely accepting as an established fact that the United States negro is not subject to trachoma."

[1] "Brazil" was omitted in my paper.

CLIMATIC BUBOES.*

By Dr. B. Scheube,

Sanitary Adviser, Imperial Physician and Medical Referee in Greiz.

Translated by P. Falcke.

(*Continued from page* 304.)

Probably several other observations appertain to the same illness, although these are looked upon by the doctors reporting them as a form of malaria. Thus Ludwig Martin[7] observed in Deli, in the N.E. of Sumatra, that patients who had previously suffered with malaria and had incipient or developed cachexia, frequently were affected with inflammatory swelling of the oblique inguinal glands, more rarely of the vertical (femoral) group. These glandular swellings were but slightly painful, but often attained an

* Reprint from the German *Archiv. für Klinische Medicin*, vol. lxiv.

enormous size. Fever and swelling rapidly decreased with suitable treatment, first and foremost change of climate, graduated compresses, bandages, quinine, arsenic and iron. When, however, the inflammation was neglected, suppuration set in, which required surgical assistance, otherwise it left ugly fistulous canals, which took a long time to heal. Martin concludes that the illness is of malarial character, because the disease came under observation in malarial patients or malarial cachectics. If, however, one considers that Martin's field of observation is essentially a malaria region (80-90 per cent. of his sick being malarial patients), these arguments lose their significance, especially if one takes into consideration the fact that his treatment was by no means purely anti-malarial.

The proof yielded alone by blood examinations was not given.

The same holds good in regard to the five cases of inguinal glandular inflammations observed by Lesueur-Florent,[8] in March and April, 1895, in Madagascar, on board an insanitary ship, and which affected in one case the inguinal glands, in three the crural glands, and in one case both. The illness set in suddenly, and after one or several days was associated with intermittent or remittent fever. The glandular swelling was soft, elastic, not spontaneously painful, and but little so on pressure. At first the separate glands showed swellings, but later through participation of the peri-glandular tissue a tumour was forced, more or less voluminous, sometimes attaining the size of a hen's egg; it increased with every attack of fever and became somewhat painful. The fever disappeared after a few days under anti-malarial treatment (quinine and arsenic), while the glandular swelling retreated sometimes quickly, sometimes slowly. Lesueur-Florent himself believes that malaria plays but a subordinate part in the ætiology of these cases. According to my opinion, these, as also Martin's cases, most probably come under the category of climatic buboes. Possibly also it is the same in regard to the cases of "bubo malaricas" in Hungary, published by A. Bodner[9] and J. Ruber.[10] These, however, I will not discuss here.*

We have thus to do with an illness which possesses an extensive geographical distribution; it occurs in Europe also, but seems to find its principal regions in East Africa, Madagascar, the East Indies, Sumatra and the West Indies. I am not in a position to be able to add more as to the seats hitherto known.

During my residence in Japan (Kioto) I observed, during the years 1877-1881, sixteen cases which doubtless may be classed with the same illness. The inflammation of the glands, with the exception of one case in which the crural glands were involved, affected the inguinal glands, mostly on one side only, in one case only on both sides. In four cases there was suppuration, and consequently incision was resorted to, to disperse them. The cases usually ran their course with fever; and, particularly at the commencement of the illness, they were accompanied by fever, which in one case preceded the glandular swelling ten days, and was intermittent. In several cases the illness dragged on for a few months. None of these cases exhibited anything characteristic. Most of the cases occurred in young men of from 17 to 29 years of age, who belonged to the most varied occupations. Amongst my cases there were only two persons of more mature age, a man of 46, and a woman of 49.

In the separate years and months the illnesses were divided as follows:—

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
1877..	..	..	..	..	..	..	..	..	..	...	..	1
1878..	..	..	..	1	..	..	..	..	..	1	1	..
1879..	..	..	..	..	..	..	..	..	..	..	..	1
1880..	..	..	1	1	2	..	..	..	..	1	1	..
1881..	1	..	..	..	..	..	..	..	..	1	..	..
	1	..	1	2	2	..	..	..	..	3	2	2

According to this, if one be justified to form a conclusion from so small a number, the illness in subtropical Japan sets in principally in the autumn and spring months. In the East African blockade squadron also, according to Ruge, the months September to November, and March to May, yielded most illnesses. In the other above-mentioned statements no information is given as to the time of year in which the illness occurred.

In my cases, also, I could find no connection between them and malaria. I certainly did not undertake blood-examinations, for my observations were made prior to the discovery of the malariae parasites by Laveran, and the knowledge of the value of this discovery. There is no support for the opinion of Skinner, who in his cases almost always observed intestinal disturbances, and therefore is inclined to consider the illness as a secondary infection originating from the mesenteric glands.

James Cantlie,[11] who came to the knowledge of this illness in Hong Kong, takes quite another view as to the nature of this illness. This writer brings the same into association with the plague, and identifies it with the so-called pestis minor, or abortive ambulatory plague. Under this head one mostly comprehends slight illnesses which are occasionally observed in plague-regions, months or even years before or after plague epidemics, and in which, without symptoms of severe indisposition, even without fever, the formation of buboes takes place gradually, these being mostly in the inguinal glands, and which suppurate, or, after being present a short time, may disperse.

Thus it was reported that the epidemic in Mesopotamia, 1876 to 1877, was preceded two or three months by non-febrile glandular swellings, and the same followed the epidemic for two months after. On

* According to my opinion, the cases of lymphangitis described by Roux (*Traité pratique des maladies des pays chauds*, iii., 1888, pp. 1-35), and which Ruge classes with those here described, do not belong thereto. In regard to the slight forms mostly occurring on Réunion, it is undoubtedly a question of filarial disease; whereas I will not attempt to classify the severe form which is mostly prevalent in Rio de Janeiro,

the south coast of China, idiopathic buboes prevailed several years before the outbreak of plague.

Epidemics of buboes have, however, also been observed without plague developing therefrom, and, on the other hand, real plague also comes to pass without being preceded or followed by pestis minor.

Pestis minor set in at Singapore and the Straits Settlements at the same time as it did on the Chinese coast, but the former places were not attacked by plague.

It may be added that Cunningham found bacilli in the blood of pestis minor, but these were not infectious and did not seem to be identical with plague bacilli, and simultaneously no illness of rats came under observation. Cunningham made his examinations in Calcutta, where pestis minor had been observed several years before the appearance of actual plague. The pestis minor question, therefore, according to my views, has passed into a new stage. It appears to me questionable if the cases of illness which hitherto have been designated pestis minor have anything at all to do with plague, or if rather they are not identical with the climatic buboes that resemble them symptomatically.

This question can only be decided by the examination of the contents of buboes, that is, excised lymphatic glands for plague bacilli. The simple practical serum-diagnosis can unfortunately not be applied for the solution, for, as Sticker has pointed out, the agglutinating effect of the serum in plague only appears distinctly in severe cases of illnesses; the more severe the illness, the more distinct; but it fails in abortive cases.

In no case can the stretching of the comprehension of pestis minor be extended to climatic buboes, as understood by Cantlie.

The question raised against the same by Godding, that the idiopathic buboes are most severe on the East African Coast, where they can have nothing to do with plague, certainly falls to the ground through Koch's discovery of a plague centre in Uganda.

On the other hand, the cases observed by me in Japan long before the plague was thought of in China and India denies with absolute certainty every connection with plague.

It is impossible to answer the question as to the cause of climatic buboes; the elucidation of the same must be left for future investigation. Only so much may be said, that the affection is originated by a still unknown excitant of illness or inflammation which undoubtedly stands in some relation to the climatic factors. In this sense the neutral term "climatic buboes" is a sufficient one until a better designation can take its place.

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WHITE MEN IN THE TROPICS.

Mr. Alfred Russel Wallace, in the *Independent*, characterises as a myth the current idea that white men cannot live in good health in the tropics. The trouble is not with the climate, but with diseases resulting from insanitary conditions, such as prevailed in Europe a century ago with the same result, and still prevail to a large extent in temperate zones. Mr. Wallace says:—"Commonly associated with the tropics are the various forms of malarial fevers, but these also are in no sense due to the climate, but simply to ignorant dealing with the soil. My own experience has shown me that swamps and marshes near the equator are perfectly healthy so long as they are left nearly in a state of nature—that is, covered with a dense forest or other vegetation. It is when extensive marshy areas are cleared for cultivation, and for half the year are dried up by the tropical sun, that they become deadly. I have lived for months together in or close to tropical swamps, both in the Amazon Valley, in Borneo, and in the Moluccas, without a day's illness; but when living in open, cultivated marshy districts I almost invariably had malarial fever, though I believe the worst types of these fevers are due to unwholesome food. But here, again, malaria was equally prevalent in England less than two centuries ago.

"If we take the great belt, about 2,000 miles wide, extending from 12° to 15° north and south of the equator, we have an enormous area, by far the larger part of which is not only well adapted for European colonisation in the true sense—that is, for permanent occupation by white men—but is also, with proper sanitary precautions, the most healthy and enjoyable part of the world, and that in which the labourer can obtain the maximum return with the minimum of toil.

"It is a well-known fact that in Ceylon and India the men who enjoy the best health are the enthusiastic sportsmen, who seize every opportunity of getting away from civilisation, and who often submit to much privation and fatigue, with benefit rather than injury to their health. The fact is that white men can live and work anywhere in the tropics, if they are obliged, and unless they are obliged they will not, as a rule, work even in the most temperate regions. Hence, wherever there are inferior races, the white men get these to work for them, and the kinds of work performed by these inferiors become *infra dig.* for the white man. This is the real reason why the myth as to white men not being able to work in the tropics has been spread abroad."

THE DEPOSIT OF IRON IN THE LIVER AND SPLEEN IN MALARIA.

Some observations of Dutton upon the iron content of the liver and spleen of two cases of malaria are of interest in showing the enormous increase of iron which may occur in these organs in this disease. The following are the results compared with those obtained by Quincke in the normal.

	Milligrammes iron per 100 grammes of organs.	
	Liver.	Spleen.
Healthy case 1	80-40	253
Healthy case 2	81	144
Malarial case 1	208	1,135
Malarial case 2	440	1,825

—*Boston Medical and Surgical Journal*, June.

A LINIMENT FOR HÆMORRHOIDS.

The *Gazette Hebdomadaire de Mèdecine et de Chirurgie* for April 20 credits the following formula to Adler:

℞ Fluid extract of hamamelis, Fluid extract of hydrastis, Compound Tincture of Benzoin, each, 16 parts;
Tincture of belladonna ... ... 4 „
Five per cent. solution of carbolic acid in olive oil ... ... 32 „

M. To be applied two or three times a day.—*New York Medical Journal*, June.

MOLLUSCUM FIBROSUM, CHELOID AND ELEPHANTIASIS ARABUM

Occurring together in a Native of Jamaica.

The combination of ailments shown in the accompanying illustration supplied by Dr. Izett Anderson occurred in the person of a native of Jamaica.

Molluscum fibrosum, the skin affection here so profusely present, is of course in no sense a specifically tropical disease. It is frequently designated "Molluscum Pendulum," a term by no means satisfactory, as many of the tumours are sessile. "Molluscum Simplex" and even "Fibroma" are met with in the nomenclature of disease as applied to the condition, but molluscum fibrosum is the usually accepted name and one to which fewest exceptions can be taken.

Molluscum fibrosum shows itself at an early age, so early that it is held by many that the growths in an undeveloped state are present at birth. Many of these may remain undeveloped, and although invisible can be detected as subcutaneous nodules when the hand is passed over the skin. The tumours reach all stages of development, from mere elevations to well-marked tumours, varying in size from a pea to that of a pigeon's egg. A medium size, however, is the rule, the majority attaining the dimensions of a marble. Many are sessile, but all tend in time to become filbert-shaped and pedunculated.

The tumours consist of simple fibrous tissue covered by healthy skin, which may be somewhat more vascular and congested than normal, owing, no doubt, to the pendulous nature of the growths.

As will be seen from the illustration, almost every part of the body may become the seat of molluscum fibrosum, and a back view of the same patient shows that the dorsal aspect of the scalp, trunk, and limbs are equally affected with the anterior. Instead, however, of a general crop there may be only one such tumour present, or at most a few pedunculated growths may occur on any part of the skin.

The disease is quite harmless, and gives rise to disfigurement merely. These tumours have but little tendency to undergo degenerative changes, and usually remain throughout life either of a stationary size or with but an infinitesimal tendency to increase.

A nice distinction as regards their origin and pathology has led to the discussion as to whether they are to be regarded as a mere malformation, or as true pathological excrescences. Usually the patient is of frail body and possessed of weak intellect.

When the tumours are few and pedunculated they are easily got rid of by cutting through the peduncle at a little distance from the root, so as to allow for the cutaneous elasticity and consequent retraction. When, however, the tumours are present in enormous numbers, and especially when they are sessile, operative treatment is out of the question.

Cheloid.—The presence of chelomata with molluscum fibrosum is an association which would seem at first sight to be a natural one. Chelomata are cutaneous tumours consisting of a hyperplasia of the fibrous tissue elements within the corium. The characteristic feature of the growth is the marked tendency of the tumour to throw out roots or spurs into the surrounding tissues of the skin.

A favourite site of cheloid is over the sternum, where a cluster of four or five, or more, tumours may be agglomerated so as to form a confluent mass. There is no clinical connection, however, between molluscum fibrosum and cheloid, so that their association in the present instance is noteworthy.

Elephantiasis.—The West Indies is one of the latitudes in which elephantiasis is endemic. As it is chiefly with Southern Asia and Egypt that we associate the disease, it is well to have the fact brought home to us in so graphic a manner, as the accompanying photograph shows, that in the West Indies it is thoroughly endemic.

Wherever the filaria nocturna is to be met with elephantiasis arabum prevails and *vice versa*, and although not always endemic the disease occurs sporadically in many tropical countries. Even in Europe cases are met with, notably in Spain. In Australia cases have been described in Brisbane; and from several of the islands of the Pacific filaria nocturna, and consequently elephantiasis arabum, are reported to be frequently encountered. In South Africa Dr. Ben Blaine met with two cases of elephantiasis, of which mention was made in the Journal of Tropical Medicine, March, 1899, p. 224. Since Dr. Manson's classical investigations in the relation between filaria nocturna and elephantiasis arabum no observer had set himself the task of repeating Dr. Manson's experiments until Dr. Bancroft, of Brisbane, took up the work. The results of Dr. Bancroft's labours have been to confirm in every point Dr. Manson's theory.

MOLLUSCUM FIBROSUM, CHELOID AND ELEPHANTIASIS ARABUM

occurring together in a native of Jamaica.

By Dr. IZETT ANDERSON.

Bale & Danielsson, Ltd., London.

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KALA AZAR.

THE report on the nature of kala azar which Major Ronald Ross, of the Indian Medical Service, recently presented to the Government of India is of immense value to those concerned in the study of that disease. Though the malady has not been investigated from the bacteriological side, and in consequence we are inclined to hold our judgment as to the conclusions arrived at concerning the disease, yet it must be admitted that the arguments and facts adduced in favour of kala azar being a form of malarial fever are exceedingly strong. The conclusion is all the stronger as it is in direct confirmation of that arrived at by Dr. Rogers after a prolonged and painstaking research. Major Ross differentiates the symptoms of anchylostomiasis from true kala azar, and shows that the anæmia with dropsy which characterise the former disease should not be confused with the fever and enlargement of the spleen and liver which essentially belong to kala azar. Both diseases are communicable from the sick to the healthy, but the points of community go no further, and if either were swept out of the country the other would remain. It is stated that if the anæmia were to be swept away the mortality in the localities now suffering from kala azar would be but little diminished. To judge from the symptoms, kala azar has the closest resemblance to the disease produced by the protozoal parasites of the red corpuscles, that is, to malarial disease. The sudden accession of high fever, of remittent or intermittent type; the frequent recurrence of similar attacks; the gradual tumefaction of the spleen and liver; even the occurrence of epistaxis, and the final cachexia, may be said to correspond exactly and in every way with the classical symptoms of that disease—in other words, kala azar is *prima facie* malarial fever. And this view is further subserved by the fact that it exists in a region known to be malarious. While, however, the clinical symptoms favour the malarial nature of the disease, pathological examination gives somewhat unexpected results. Thus in well marked cases of kala azar not a single malaria parasite could be discovered on repeated and careful examinations of the peripheral blood in the large majority of the cases even when fever was present. Further, examination of blood taken from the spleen, and sometimes the liver, during life, showed the presence neither of parasites nor in most cases of the black pigment. In two out of seven autopsies made on cases which had been diagnosed kala azar, neither parasites nor black pigment could be found in the spleen or liver, but yellow pigment was present in the liver in all the autopsies but one. Moreover, careful thermometric observation of the patients showed that the temperature charts gave the curve of constant low fever, and not of typical malarial fever of any kind. These results are sufficient to raise grave suspicions that kala azar, in spite of its great clinical similarity to paludism, is really something quite different. The facts in favour of the malarial theory are that the early symptoms are almost identical with those of malaria; that the disease occurs in malarious regions, and that most of the cases contain yellow pigment; while the facts against this theory are high death-rate, the intractability to quinine, the existence of a low constant fever not amenable to quinine, and not like malarial fever in the second stage of the disease, the apparent absence of the parasites, and of the melanin of paludism from many established cases

of the disease, and, lastly, the communicability of kala azar from the sick to the healthy, and its epidemicity. But Major Ross thinks these pathological differences are more apparent than real, and that they are explainable. Thus he points out that in the early cases of kala azar the malarial parasites are found with great facility; later, as the disease advances into several months, they are discovered less and less readily, though when not observed in the peripheral blood they or their immediate product, melanin, can generally be detected in the spleen or in the liver; finally, when the enlargement of the organs has become very marked and the low fever is established, the parasites or the melanin cannot be found; and he further points out that precisely similar pathological phenomena are to be noted in ordinary fever when prolonged; thus in old cases of paludism the absence of parasites, the presence of low fever, and much enlargement of the liver and spleen are to be observed.

In support of these observations of his own, Major Ross cites those of Vandyke Carter and of Kelsch and Kiener, on the occurrence of secondary fever in chronic malaria not dependent on the presence of parasites; and those of Dr. Daniel, who, in British Guiana, found malarial parasites much less readily in cases of chronic enlarged spleen than in early cases of malarial fever; who also found in cases of enlarged spleen an apparently non-malarial fever not amenable to quinine, and not accompanied by parasites; and, lastly, detected no black pigment in a very large number of enlarged spleens examined *post mortem* even of patients who suffered from this fever at the time of their death.

With these facts before him, Major Ross believes that kala azar is a form of paludism in which the infection falls with a very heavy incidence on the spleen and liver, especially the latter; and it is the enlargement of these organs, and the accompanying low fever, which are the greatest feature of this disease, and which, he thinks, are the chief cause of the exceptional mortality, and that kala azar is principally a manifestation of the secondary results of malarial fever. As regards the communicability of kala azar, Major Ross thinks that there is no reason on this ground to object to it being a malarial disease, for, as he has recently shown, malarial fever is itself a communicable disease which is, indirectly, spread through the agency of insects, and he is of opinion that isolated instances of a similar fever occur, more or less, throughout India, and that in particularly malarious localities they occur in large numbers.

Article for Discussion.

THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS.

Scarlet Fever.

We are so accustomed to dilate upon the prevalence of virulent diseases in tropical countries that it may come as a surprise, to many unacquainted with tropical ailments, that some of the severest scourges, known to medical science, diminish in frequency of occurrence and in virulence as the Equator is approached.

No disease stands out more prominently in this respect than does scarlet fever. In India, in the tropical regions, at all events, the disease is unknown, and in the countries bordering on the China Sea—South China, Tonquin, Annam, and Cochin China the same holds good. How far this extends to other countries inside the Cancer-Capricorn area is not recorded; but at the present moment there is no information to be had that scarlet fever exists within the geographical limits mentioned. As the tropic of Cancer is approached from the north the disease disappears from the nomenclature. As an example, the cases of Shanghai situated 31° N. lat., and Hong Kong 22° N. lat., may be cited; scarlet fever is met with in Shanghai, but never in Hong Kong except as an importation. I saw but one case of scarlet fever in Hong Kong; the patient was a young man who had landed two days previously from England. Dr. F. W. Clark, Medical Officer of Health for Hong Kong, in his report for 1898, writes:—"Four cases of scarlet fever were reported during the year, two on board

H.M.S. *Barfleur* and two on board H.M.S. *Powerful*. The infection appears to have been brought out from England, as this disease is practically unknown here, and does not appear to have any tendency to spread among the Chinese."

The cause of the disappearance of scarlet fever would seem to be partially climatic, continued high temperature lessening the power of the virus, and directly a high equable temperature prevails so does the scarlet fever infective power diminish. Still it does occur in the tropics, as Dr. Clark's report indicates, and cases of communication on board ship are not unknown. I am acquainted with several such cases. One example will suffice. A family of British children on the way home from Japan developed scarlet fever. Children taken on board at Hong Kong contracted the disease between Hong Kong and Singapore, and several cases occurred on the way across the Indian Ocean to Columbo. It would seem, therefore, that the factor of continued equable temperature is not the only one, as, in the tropical heat, the disease may exist and be communicated. The absence of scarlet fever amongst natives and European residents in the tropics is, however, a fact, and it would seem that environment may have to do with it.

On the other hand, is it possible that scarlet fever is present in some "altered" form? Not infrequently we hear of malarial fever being "catching," and it is just possible that some non-malarial ailment is the explanation. A child in Hong Kong developed what I diagnosed as remittent fever. The mother remarked that "whether it was malarial or not, it would go all over the house." Certainly three of the brothers and sisters of the patient developed a similar fever within a short time, in each case attended by high temperature for from seven to fourteen days. The mother had had a parallel experience previously, and based her opinion upon it. This is by no means an isolated example, and it opens up a question of an infectious non-malarial fever, although of course the factor of equal exposure to malaria must not be forgot, in this and similar occurrences.

May I look to readers of THE JOURNAL OF TROPICAL MEDICINE to record their experiences as regards this disease? The absence of a disease in any country is a scientific fact of much interest, but it is a form of negative record we are apt to neglect. The geographical distribution of scarlet fever and several other diseases is of eminent interest, and science would benefit greatly would medical practitioners in the tropics help to elucidate the subject. J. C.

Translations.

BLACKWATER FEVER (HÆMOGLOBINURIA).

By R. KOCH.

Translated by P. FALCKE.

HÆMOGLOBINURIA is an illness which occasionally comes under notice in all climates. It may be caused by organic or inorganic toxins, such as chloric acid salts, arseniurated hydrogen, sulphuric acid, muriatic acid, phenol, naphthol, aniline, chrysarobin, pyrogallic acid, toluylendiamin, glycerine; furthermore, by a few vegetable poisons, of which that contained in Helvella esculenta has been most minutely investigated.

Other causes also, such as the influence of cold or strenuous muscular exertions—especially arduous marches—may be the origin of its existence.

However, that form of hæmoglobinuria designated blackwater fever (*fièvre bilieuse hématurique*) only occurs in tropical climates; it is scarcer even in subtropical climates. The cases occurring in temperate climes always originate in tropical countries. This particular kind of hæmoglobinuria is supposed to stand in intimate relationship with malaria; this has hitherto been almost universally accepted. Generally even one goes so far as to assert that the attack of blackwater fever is nothing more nor less than an exceedingly intense attack of malarial fever; and, in truth, there is a very great similarity between the former and the latter. A severe attack of ague is followed by a stage of heat of not very long duration, then the fever decreases, and therewith the end of the actual paroxysm. The impression is formed of a somewhat severe attack of fever not differing essentially from the usual course. The appearance of considerable quantities of hæmoglobin in the urine, the icteric discolouration of the skin, the uncontrollable vomiting, &c., are looked upon as complications which are really attributable to the severity of the fever, in connection with which one is led to think of particularly virulent and numerous malaria parasites which disintegrate the red blood-corpuscles, releasing their hæmoglobin.

Nevertheless, all medical men who have had the opportunity of becoming acquainted with blackwater fever in the Tropics are not satisfied with this explanation. Again and again the opinion is vouchsafed that

blackwater fever is something peculiar, a specific disease. From this stand-point researches were made to find a specific cause of the disease in the blood and urine of the patient, the starting point of the opinion being that blackwater fever must be an infectious illness.

But all investigations with this end in view have been without result, and the assertion of Yersin, who professes to have found in a certain kind of bacillus the cause of blackwater fever, has found no confirmation.

In this direction, therefore, the existence of blackwater fever has not been elucidated, but one has succeeded in another way in showing that the fever is an independent process of disease which does not stand in close connection with malaria, but rather agrees with what we know of hæmoglobinuria in non-tropical lands.

It is to S. Tomaselli that the credit belongs to have rightly directed us in Sicily. He observed a number of cases in which hæmoglobinuria followed the administration of quinine, and he was convinced that it was not the question of a coincidence, but that the quinine was the cause of the hæmoglobinuria, so that, in a word, the latter must be a quinine poisoning. His communications were soon confirmed by Greek observers, and, in the course of time, by medical men in French and German colonies.

In regard to the position I myself took up in this question, I may say that at the beginning I was entirely opposed to it. When, quite unexpectedly, I had the opportunity to investigate tropical malaria in East Africa, hardly any literature was at my disposal, and nothing remained for me but, on the grounds of my own observations, to form an opinion as to the nature of this illness which is fraught with such significance for tropical Africa. Gradually I was forced by accumulating facts to relinquish my original opinion that blackwater fever was a certain modification of malaria, and was led to consider the illness to be a state of intoxication. It was a great satisfaction to me to learn subsequently that I by no means stood alone in my opinion.

As to the results I arrived at in my investigations, I have made previous statements in a report laid before the German Colonial Society, and also in the reports of my travels.

These short reports, really intended for the general public, called forth much controversy, especially from tropical medical men, and lately a lively discussion on blackwater fever has developed. I had really intended to gather further material and then to publish an exhaustive statement. It seems to me, however, in view of the interest aroused, that I may now, at least, present the grounds which led me to my present opinion of blackwater fever.

The question primarily is whether blackwater fever be an attack of malaria or not. It is only after this query has been decided in the negative that further investigations can be gauged as to the actual nature of the illness.

Malaria is fortunately one of those illnesses recognisable with certainty, the presence of the malaria parasite being taken as the infallible diagnostic sign of malaria. Of several hundreds of cases of malaria examined by me, I have not once failed to prove the presence of the parasites. Certainly it may occur that the parasites may be missed during a long interval between the attacks, or when quinine had been used previous to the time that the examination of the blood was made; but if one waits until the next relapse, which is hardly ever missed, the result of the examination will give positive evidence. During, or shortly after a fresh attack, the parasites are always present.

In order to decide the question if blackwater fever belongs to the group of malaria illnesses one should make an investigation to ascertain if a certain blood parasite be regularly found in this disease as is the case in malaria.

Remarkable to relate, such examinations, at all events in the tropics, have only exceptionally been made. Besides a few fragmentary reports, I am only acquainted with the methodical examinations of F. Plehn, made in the Cameroon district. He reports that during the attack of blackwater fever he had seen in the blood, and, indeed, partly in the red blood-corpuscles, and partly free between the same, mobile little bodies which did not take the methyl-blue stain. He considers these little bodies to be young malaria parasites. This explanation of his discovery, however, cannot be the right one, for the reason that it is just the youngest forms of the parasite that take the methyl-blue most intensely. What F. Plehn saw were presumably the blood-lamella or fragments of disintegrated blood corpuscles. I must take it for granted that—as in F. Plehn's cases—no parasites could be discovered in the blackwater blood through methyl-blue staining; there were none extant.

As to my own examinations, I have, for the reasons assigned, only made use of such cases of blackwater fever in which blood examinations were made, under the application of reliable methods.

Of such cases the following are at my disposal:—

(1) 19 from East Africa.
(2) 2 from South-West Africa.
(3) 15 from Cameroon.
(4) 5 from Germany.

Total 41

I myself observed those cases originating in East Africa and those occurring in Germany. As to the two cases from south-west Africa, I received the charts and blood preparations from the physician to the Imperial body-guard, Dr. Dempwolff. The Cameroon cases were collected by Staff-surgeon Dr. Dœring. The latter kindly gave me the material—which is being used for another publication—for my investigations.

In regard to the method of examination, I may remark that dry cover-glass preparations exclusively were used. If these are prepared so that the blood, immediately after its exit from a small puncture, is spread in the thinnest and most even layer possible, dried, hardened for twenty minutes in alcohol, and then stained with alkaline solution of methyl-blue or with borax methyl-blue, the results achieved are absolutely reliable.

Even Doering's prepared cover-glasses, made two years ago, revealed the parasites with a clearness

which left nothing to be desired. The parasitical conditions in my cases were as follows:—

In twenty-three cases no parasites at all were found, notwithstanding several, and in some cases very frequent, examinations. Of these twenty-three negative cases four had a fatal issue, and the remaining cases had no relapses, although in thirteen cases no quinine was administered either during or after the attack.

These two facts, the lack of parasites in more than half of all cases, and the omission of the relapses, would alone suffice to prove that blackwater fever itself cannot be malaria, but that it is pretty frequently associated with malaria.

The absence of the malaria parasites in the blackwater fever attack has already been remarked, but other investigators give this explanation for the phenomenon:—that with the disintegration of the red blood-corpuscles the parasites contained therein also perish, and thus disappear from the circulation. The following argument contradicts this purely hypothetical assertion:—

Did blackwater fever originate in the manner set forth by the foregoing hypothesis, namely, that only the red blood-corpuscles infested with parasites became disintegrated, it would follow that the parasites collectively must disappear from the blood in every case. This by no means occurs. Amongst my cases there were four in which the parasites were to be seen during and directly after the attack. In one case, indeed, they were present in great numbers. Furthermore, in nine cases relapses occurred later on, which proves that the parasites did not perish regularly in the blackwater fever attack.

Furthermore, the behaviour of Texas fever contradicts this hypothesis. Texas fever is the only illness in which malaria-like parasites are extant in the blood, and in which a hæmoglobinuria is caused by parasites. Here, then, we have an illness which in reality exhibits those conditions which have been conjecturally ascribed to blackwater fever. A comparison with Texas fever must, therefore, be peculiarly suitable to the better comprehension of blackwater fever, and it happened fortunately for me that in East Africa I was able simultaneously to observe blackwater fever and Texas fever, and thus to compare the conditions of the two illnesses. I found that in Texas fever the appearance of the hæmoglobinuria was in direct ratio to the number of the blood parasites. When the illness ran a mild course but few parasites were to be found in the blood, and the hæmoglobinuria was quite absent; it only set in when the parasites were more numerous and the course of the disease became acute. The most intense hæmoglobinuria was seen by me in Texas fever in cases with a virulent course and innumerable blood parasites. But even in these cases, and in which very many blood corpuscles had perished, I never remarked that the parasites were influenced in the least by the attack. They were present in such myriads that considerable masses of the same could not disappear. Besides, in just such cases I have seen shoals of parasites free in the serum, and I am of the opinion that these were parasites which, after the disintegration of the blood corpuscles they had formerly denizened, had become free without in any way having been damaged by the disintegration of the blood corpuscles.

I have never encountered a similar unanimity between the number of the parasites and the intensity of the hæmoglobinuria in malaria. In not one of the cases of blackwater fever in which blood parasites were found was there a considerable number. It was always a question of relatively few, sometimes, indeed, *very* few parasites. I can quote quite an opposite condition in a few cases of malaria with very numerous parasites without even a trace of hæmoglobinuria being present. In two cases with fatal terminations the number of parasites averaged respectively 30 per cent. and 80 per cent. We thus perceive that the malaria parasites do not perish in blackwater fever attacks; that during the attack they are either entirely absent or show no remarkable increase; that even if they are extant in great numbers no hæmoglobinuria need develop. Therefore, how can one still assert that blackwater fever is caused by malaria parasites?

(*To be continued*).

A STRANGE CASE OF MALARIAL MELANODERMIA.

Clinical Note by Dr. GAETANO FAZIO.

Medical Officer to the Military Hospital of Perugia.

Translated from "Giornale Medico del Regio Esercito," 1899.

BARTOLETTI GIUSEPPE, carabineer, aged 23, was treated in this hospital from February 27 to March 31, 1898.

Nothing of interest was gathered from the information given respecting heredity or antecedent malady; he had not been a drinker or smoker, and had never suffered from syphilis or venereal disease, or been treated with arsenic or salts of silver.

Three years before, being in Celli de Castro, he fell ill of malarial fever of a tertian nature, and the first attacks were serious, for he asserts that he was cured of a malignant fever. His illness had lasted for some days when a mark appeared upon the skin of the thigh, and after this the cutaneous pigmentation gradually increased after each paroxysm of the fever. This occurred in the following manner: the fever began with a long and violent shivering fit, the temperature rose rapidly and after five or six hours fell, and the crisis passed off with a profuse perspiration. About an hour after the administration of quinine the patient complained of intense irritation of some fixed spots upon his skin, at which points a red mark had formed about the size of a pea or a centime, and if any other mark had previously appeared in the same place, the red mark extended peripherally, beyond the former mark, to the sound skin, forming a circle with the former mark as its centre. The itching gradually disappeared, and in the course of a few hours the mark assumed a blackish-brown colour, which underwent no further modification.

During convalescence no further marks were formed, and the quinine administered as a prophylactic temporarily changed the colour of the marks from blackish-brown to a vivid red.

Actual condition.—The subject is of a robust physical constitution, with well-developed muscles, and is well nourished. The lymphatic glands are not enlarged. An examination of the internal organs reveals only a slightly enlarged spleen.

Upon the skin marks are observed, roundish with an irregular margin, varying in size from that of a centime to that of a crown piece; they are of a blackish-brown colour, not modified by pressure or scratching. The marks are clearly limited by the normal coloured skin, they do not itch, or cause any pain or positive disturbance; there is no desquamation, the surface is smooth, and the subcutaneous tissue is not infiltrated. They are distributed without any symmetry whatever, being irregularly scattered over the skin of the trunk and limbs; the largest are in the regions of the thighs and loins, and the most numerous on the back, only a few are to be seen on the abdomen and limbs, and there are no traces of them on the face and neck.

During a month's residence in the hospital he had no further attacks of fever. One day a grain of bi-sulphate of quinine having been administered, the marks assumed a fuscine red colour for some hours, and afterwards gradually resumed their former blackish-brown colour.

The blood extracted from the patient's finger showed no hæmatosis or granules of melanin.

An examination of the urine disclosed an abnormal quantity of organic, and a notable quantity of mineral, matter.

Histological examination.—A small piece of skin was removed from one of the marks with a curved pair of scissors, and was placed in alcohol immersed in paraffin, and cut in sections.

The microscopical examination revealed the existence of an abundant deposit of roundish granules of a reddish brown colour in the Malpighian layer and upon the outer surface of the skin. The pigment was for the most part outside the cells. These granules vary in size and are massed together in a manner which makes the small increase of size apparent. A section of the skin, after being treated for half an hour with sulphide of ammonia, presented a greenish colour; the microscope showed that the rete mucosa had taken on a dark green colour at certain superficial points, and the pigment had turned completely black. This reaction showed the presence of iron in the pigment. The sulphide of ammonia did not completely dissolve all the granules.

Diagnosis.—It is easy to exclude from this case congenital or idiopathic pigmentation of the skin, or that following chronic inflammatory processes, traumatic lesions and external causes. We must therefore turn our attention to symptomatic pigmentation arising from an internal malady, or following upon the ingestion of some medicament.

It is not due to the taking of salts of silver, for the patient has never made use of them, and the pigmentation is not uniform and does not extend to the mucous membrane of the mouth. We cannot admit that it is due to quinine, though clinical history shows that it is often caused by the administration of that drug, and though there appears some evident relation of cause and effect. In fact the nutritive and circulatory alterations of the skin due to quinine observed in other diseases, eczema, roseola, urticaria, and desquamative exanthema, disappear rapidly, as soon as the remedy is discontinued, and have never caused a permanent anatomical alteration.

It is further asserted by the patient that no marks appeared during convalescence, in spite of the internal use of quinine.

Among symptomatic cutaneous pigmentations arising from constitutional causes we need study only pigmentary syphilis, and paludal infection.

The patient never suffered from venereal contagion, nor were there any specific secondary manifestations thereof; the lymphatic glands are actually impalpable, nor are any other concomitant symptoms to be seen; there are no cicatrices on the skin or mucous membrane which can give rise to a suspicion of syphilitic infection.

Pigmentary syphiloderma does not commence with itching, and its most frequent seat is the side of the neck where Fournier observed it twenty-nine times in thirty.

Neither can the therapeutic results be taken as a criterion for diagnosis, for it is universally admitted that mercurial treatment has no influence upon syphilitic pigmentation (Bumstead).

This melanodermia cannot therefore be considered as a manifestation of the secondary or gummous period, nor as a pigmentary dischromia following syphilodermic progress.

On the contrary, the characteristic course of the paroxsysms of fever, their intermittence, the appearance of the marks constantly coinciding with the fever, the enlarged spleen, the therapeutic and prophylactic action of the quinine upon the disease, and indirectly upon the pigmentation, evidently point to a malarial origin. These marks, from the development of the muscles and well nourished condition of the patient, and the rapidity and suddenness of their formation, cannot be due to chloasmic cachexia.

The negative results of the examination of the blood as regards hæmatosis, is of very small value, considering that the blood was not extracted from the spleen, that the patient had been ill for a long time, and that from a long and correct treatment infection might be extinct. The said case, besides the origin and formation of the pigmentation, offers an occasion of considering the method of formation and the action of the quinine upon the colouration.

The colouration of the skin in malaria is anything but rare, and I will only mention here the case of acute melanemia reported by Professor Cardarelli which resembled Addison's disease; and the other, published by Morcata, which showed a symmetrical pigmentation of the lower limbs.

The first colouration came on all at once during the first attack of fever, and the cause was set down to be due to the enormous quantity of melanin found circulating in the blood.

In the second case trophic lesions in the region of the splanchnic nerves were found, so that the malaria would be the cause of a nervous lesion, and only the indirect cause of the melanodermia.

In the clinical case in question we may follow the formation of the pigmentation and its gradual increase

in the different paroxsysms of fever and we find a repetition of what only occurred once in the case of Professor Cardarelli.

In order to explain this fact I think it would be well to recall briefly certain researches concerning the origin of malarial pigment.

In the first place I will state that the formation of pigment in the skin is a physiological function. According to some, the pigment is of hematic origin (Virchow, Kölliker, Aebry, Ehrmann, Riehl, Rénant) and is carried to the skin by the migrating elements by means of the leucocytes and of those dermic cells called chromoblasts and chromataphores. According to others it is of automatic origin, and is a product of the nucleus or protoplasm of the epidermic cells. (Averbeck, Mertsching, Krammer, Caspary, Port, Delepine.)

In malaria it is admitted by all that the melanosis is derived from the colouring matter in the blood, following an abnormally abundant disintegration of the red corpuscles, and Sakaroff admits that the malarial pigment is really formed from the melanosed nucleus produced at the expense of the paranucleus.

This pigment, according to Lavèran, presents a characteristic chemical reaction. Last year he communicated to the Societé de Biologie, that two pigments are found in those dying of malaria; the yellow pigment, and the black pigment. The latter is proper to malarial infection, the former is found in many other pathological conditions. The first is yellowish, and gives the reaction of iron, the second resists acids and alkalies and does not give the reaction of iron, but dissolves rapidly in sulphide of ammonia. The two pigments are found differently distributed in the tissues.

Analysing these reactions it is recognised that they are not sufficient to prove or annul the malarial origin of a pigmentation. In fact there are other pigments which do not contain ferro-hematoid, such as the pigment of the human hair, of the choroid, of Addison's disease and of certain melanotic sarcomata.

Since in a section of the organs of a malarial subject the yellow and black pigment may both be found, the reaction of the one may conceal that of the other. Therefore the reaction of iron discovered by the microscopic examination with sulphide of ammonia is of a very relative value.

It remains to inquire where the formation of the pigment takes place, and how it reaches the skin.

It would be out of place to quote here the hypotheses of Virchow and Frerichs, or that of Kühne, Arnstein, and Welsch upon the pathogenesis of melanemia, and to-day it may be admitted that the malarial pigment is formed in the circulating blood.

Marchiafava and Celli, before the discovery of the malarial parasite, had observed that in the substance of the red corpuscles dark granulations were formed, and supposed to be microparasitic. After the discovery of Laveran, it was demonstrated that the malarial parasite accomplished its biological cycle in the red corpuscles; and a number thereof, first studied in Italy by Golgi, contained granulations of pigment which were afterwards deposited, principally in the spleen, by the action of the melanitic leucocytes, which ingest the pigment which remains after sporulisation, or by the plasma forming phagocytes, which contain dead parasites or those in the course of disintegration. Thus if it is proper to this malarial parasite to live in the red corpuscle and to form a pigment of the hæmoglobin, it is clear that no discoloration should be found in other infectious diseases.

The cutaneous pigmentation in this case should be considered like the melanosis of the other organs, and since it is formed from four to five hours after the commencement of the fever, the period when it is proved that the largest number of melanific leucocytes are present in the blood—it was the white corpuscles which carried the pigment to the skin. The rapidity of the formation of the pigmentation, and the intense irritation which accompanied it in the beginning, excludes the notion that it was produced by a slow elaboration of the epidermic cells by a functional modification thereof; and it should be attributed to a species of tatooing, both of the exterior and interior of the vessels. Finally, seeing that such a form of cutaneous melanosis is rare, while malaria is a very common disease, it must be admitted that special conditions existed to determine it.

These conditions, in my opinion, were due to the variety of the parasite causing the malaria and the patient's idiosyncrasy for quinine. It was a case of somewhat serious illness, which during the paroxsyms of fever produced an enormous destruction of red corpuscles, which were deposited in the various tissues and in the skin, and these organs, not being adapted to purify the blood sufficiently, gave rise to these ulterior modifications; in confirmation of which I note a fact observed by the patient, that in that malarial country some others bear the same marks, though much less numerous.

The other factor which contributed to produce the melanodermia is represented by the quinine, which is occasionally the determining cause. This was due to the individual idiosnycrasy for this drug, which is evident from the vaso-motor phenomena which it always produced upon the marks during convalescence, and from the fact that during the fever the marks always appeared after it had been administered.

To explain its action it may be admitted that a stimulant to the nerves or vaso-motor centres exercised a paralysing action upon the amæboid movements and vital activity of the melanific leucocytes (Geltovski), causing thereby an abnormal deposit in the various tissues at the time when the pigment was circulating in the blood.

The cutaneous pigmentation depending upon these two conditions, *i.e.*, discoloration and idiosyncrasy for quinine, it is not surprising that it should be found in such close relation to the various attacks of fever.

The treatment followed was principally directed to combating the malarial infection, and after a month's residence in the hospital Bartoletti resumed his service in good health. The pigmentation showed no increase or modification.

Reprints.

ON TRANSMISSION OF PROTEOSOMA TO BIRDS BY THE MOSQUITO: A REPORT TO THE MALARIA COMMITTEE OF THE ROYAL SOCIETY.[1]

By Dr. C. W. DANIELS.

From the *Proceedings of the Royal Society*, vol. 64.

I HAVE the honour to report the results of my observations since my arrival here (Calcutta) on December 21, 1898.

(2) Major Ronald Ross, I.M.S., after demonstrating and explaining to me his method of dissecting the mosquito, showed me in prepared specimens the pigmented bodies met with in the stomach walls of mosquitoes fed on birds infected with proteosoma, and also the changes which these bodies undergo day by day. Finally he demonstrated to me the "germinal threads" in cysts in the stomach wall, in the fluids of the body, and in the cells of the veneno-salivary glands.

(3) On my arrival there were in the laboratory, in test-tubes, several series of mosquitoes which had fed on birds infected with proteosoma on the nights of November 30, December 10, December 12, December 15, and December 20.

Of each of these series Major Ross dissected specimens, and demonstrated in them the same bodies that he had already shown me in prepared specimens. He pointed out that in the older mosquitoes it was possible to predict from an examination of the fluid, obtained on cutting the thorax, the nature of the contents both of the "coccidia" (the term employed by Ross)[2] in the stomach, and of the cells of the veneno-salivary glands.

These points I readily observed.

(4) Of the mosquitoes referred to I day by day examined those which died, and others which I killed. In these I was able to repeat the observations and, in insects belonging to the earlier series, to trace the changes in the size and in the nature of the contents of the "coccidia."

I also examined a large number of mosquitoes caught about the laboratory, and others which had been raised from larvæ. In none of these did I find either "coccidia" in the stomach wall, germinal threads in the body fluids, or germinal threads in the cells in the salivary glands; nor did I find "black spores" (Ross).

(5) Major Ross informed me that his published results were based on observations made in the hot season, when the temperature was 80° F., or over; and that now, as it was the cool season, I should find the changes progress more slowly, although the sequence of events was the same. My observations on the mosquitoes fed on December 20 and December 15 showed that this was the case. Major Ross also informed me that, with lowered temperature, mosquitoes fed less readily, and that more difficulty was experienced in rearing them to a spore-bearing age.

These difficulties the use of the incubator was only partially successful in overcoming.

(6) On the evening of January 1, following exactly in Major Ross's lines, I commenced a repetition of his main experiment:—

A large number of grey mosquitoes, reared from larvæ, were released in two mosquito nets.

In net No. 1 four birds were placed. On December 31 I had already found proteosomata in large numbers in three of these birds, and in the fourth in moderate numbers.

In net No. 2 two birds, in whose blood no proteosomata had been found, were placed. These two birds died two and three weeks later; on dissection no black pigment was found in their organs. Repeated examinations of their blood had failed to discover proteosomata.

On January 2 none of the mosquitoes had fed, and on January 3 only two in net No. 1, and eight in net No. 2. On January 4, which was a warm night with a minimum temperature of 59·2° F., sixty-three mosquitoes were found in the morning gorged with blood in net No. 1, and were caught in separate test-tubes which were then plugged with wool and placed in the incubator. Of the control series in net No. 2, where the non-infected birds had been placed, eighteen were caught and treated in the same way.

On the following two evenings, with minimum temperatures of 60·7° and 63·2°, sixty-three and forty-six mosquitoes were fed on the infected birds, net No. 1, and were kept for the preparation of specimens; and twelve mosquitoes were fed on the non-infected birds, net No. 2, bringing the number of the control series up to thirty-eight. At a later date eighteen mosquitoes were fed on a blue jay with numerous halteridia.

On the third day the sixty-three mosquitoes, from net No. 1 (with exception of those previously killed for examination, or which had died) were released inside a clean net free from other mosquitoes. Birds free from proteosoma were also placed in this net.[3]

In the morning all mosquitoes found inside were collected. Most of them had fed well. The minimum temperature during the night had been 63·2° F.

The mosquitoes were not fed on the following night, as they were full of blood, which most of them voided during the night. Many died next day.

The remainder were given the opportunity of re-feeding every night after this; but as a spell of cold weather set in with minimum temperature of 44-49°F.

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[1] Communicated by Dr. M. Foster, Sec. R.S., by direction of the Malaria Committee. Received February 13,—Read March 16, 1899.

[2] See note by the Malarial Committee appended to this Report.

[3] This is the method Ross employs to re-feed mosquitoes. If infected birds are employed to re-feed the insects, a younger generation of coccidia is produced; I therefore used sterile birds for this purpose.

The method works fairly well in warm weather, but there is always some loss, as the full number is never collected again in the morning. As the process is repeated over and over again, this loss becomes serious, the more so the longer the period required for maturation of the coccidia. Moreover, in a frequently repeated process of this kind there is always the possibility of an outside mosquito getting inside the net, and to that extent vitiating the experiment.

(only on one night did it exceed 50° F.), few fed well or at all, and there was a consequent continued heavy mortality. Only one insect, which subsequently escaped in the night, being alive on the tenth day.

This method of feeding was very unsatisfactory in exceptionally cold weather. During the day the mosquitoes being kept warm in the incubator rapidly digested their food, whilst at night the cold rendered them torpid, and they did not feed.

The control mosquitoes, of net No. 2, were treated in exactly the same manner, being fed on birds free from proteosoma. The last died on the thirteenth day.

7. The results of the two series are as follows:—

Of sixty-three mosquitoes fed on proteosomal birds, forty-nine were examined, three were reserved for sections, one was too much decomposed for satisfactory examination, ten were not accounted for, having been lost in the nets.

Of the forty-nine examined two were killed on the first day—that is, under twenty-four hours, and possibly under twelve hours after they had fed; no coccidia were found in these. Two more were examined the following morning, that is, under thirty-six and possibly under twenty-four hours after they had fed; no coccidia were found in these.

In two examined about 4 p.m. of the same day, that is, under forty-six and possibly not more than thirty-four hours after they had fed on the infected birds, minute pigmented coccidia were found.

The remainder were examined on the following days. The largest numbers (eighteen) were examined on the fourth and (twelve) on the seventh days, as on these two days the mortality amounted to this.

In all these mosquitoes, with one exception, coccidia were found—usually in numbers; in one there was only one coccidium.

The exception occurred on the ninth day; but as by that time the insects had been re-fed several times, the mosquito in question may have been an outside one which had effected an entrance.

Of forty-five mosquitoes fed on the infected birds and examined, more than thirty-four hours after, forty-four contained coccidia.

This I may say is a more successful result than in the other series I have seen.

The other two series of mosquitoes were used by all of us for the preparation of specimens, and no record was kept of the number of non-infected insects. Judging from my own examination, only about three-quarters of them developed coccidia. Their treatment had been somewhat different, as for several days half of them were not incubated.

Of the controls fed on birds free from proteosoma, thirty-eight in number, and treated in the same manner, twenty-nine were examined and nine are unaccounted for — lost in the nets. None of the twenty-nine were examined on the first day, but one was on the afternoon of the second day. The largest number, seven and five, were examined on what would correspond to the fourth and seventh days, four were examined on the fifth and four on the sixth days.[4] In none of these twenty-nine were coccidia found.

Of the eighteen fed on the blue jay with halteridia, twelve were examined from two to six days after feeding; none contained coccidia.

(8) The coccidia (pigmented bodies) found on the second day measured 6-7 μ, some of them a little more. They were oval bodies containing scattered granules of black pigment, and had a sharp, clear outline.

(To be continued.)

[4] It will be observed that these control mosquitoes were not, as the other series, collected on one, but on three nights. A very slight difference in breeze and light seems to affect the numbers that feed; any extra restlessness on the part of the birds has the same result.

Recent Literature on Tropical Medicine.

TROPICAL OPHTHALMOLOGY.

Several papers of interest to ophthalmic surgeons practising in the tropics were read and discussed at the recent Congress of the Ophthalmological Society of France. Prominent among these were Dr. Trantas' "Observations on Fundus Lesions in Leprosy"; Dr. Péchin's "Case of Malarial Iritis"; and Dr. Zanotti's "Case of Quinine Amaurosis."

Fundus Lesions in Leprosy.—M. Trantas, of Constantinople, whose work on the ocular lesions of leprosy I have more than once had occasion to refer to in these columns, read an interesting communication on the ophthalmoscopic examination of lepers. In the course of a systematic examination of a large number of lepers he found distinct fundus lesions in several; in the majority of the cases these consisted of white or black spots beneath the retinal vessels, without any surrounding area of pigment disturbance, and apparently producing no visual defect. One case, however, presented in addition to these spots a large patch of atrophy in the macular region three times the size of the disc; in another a white process projected into the vitreous from the retina, probably retinitis proliferans following a large hæmorrhage; and in some were posterior staphylomata although the eyes were not myopic. One leper subsequently developed a capsulo-lenticular cataract which was successfully removed, shewing that cataract extraction is not contra-indicated. Of other ocular lesions in leprosy M. Trantas specially alludes to hyphæma, corneo-scleral leproma, iritis with occlusion of the pupil, and keratitis. Fundus lesions, in his opinion, are by no means uncommon and should be always looked for where ophthalmoscopic examination is possible.

In the subsequent discussion M. Antonalli confirmed Trantas' observations and described a case of ocular leprosy which had been under his care for a lengthened period. At an early stage this patient presented atrophic changes round the disc, diffuse pigment disturbance, and a few foci of choroido-retinitis; soon, however, keratitis and lenticular changes appeared and ophthalmoscopic examination became impossible. He also advocated the systematic examination of the eye in all lepers. M. Morax (whose work on leprosy written in collaboration with M. Jeanseline I have had the honour of introducing to English readers) stated that he had never been able to detect fundus lesions during life and believed them to be rare; he also laid stress on what he considers an invariable rule, viz., that the anterior segment of the eye is always the first affected. M. Koenig mentioned the case of a leper who developed a very peculiar form of corneal infiltration with spots of pigment between the laminæ.

In replying, M. Trantas found himself unable to agree with M. Morax that the anterior segment of the eye was invariably the first affected; he thought the order of ap-

pearance of the ocular lesions was not always the same and that cases occurred in which changes in the fundus preceded changes in the anterior portion of the eye.

Double Iritis of Malarial Origin.—Dr. Péchin read notes of a case of malarial iritis. Patient, a woman, aged 48, suffered from tertian ague in 1870; the fever lasted in all several months, but eventually yielded to quinine. During one of the paroxysms her right eye became injected and painful with discolouration of the iris and immobile pupil; eventually the iritis subsided, leaving a posterior annular synechia. Five years later she suffered again from a severe attack of malarial fever; this time the left eye became affected in one of the paroxysms, and several synechiæ formed; she has had no recurrence of either malaria or iritis since. Dr. Péchin goes carefully into the history and antecedents of the patient and into the differential diagnosis of malarial iritis, and comes to the conclusion that the disease in this instance was undoubtedly directly due to paludism. He notes the extreme rarity of malarial affections of the iris while dwelling on the comparative frequency of optic neuritis, retinitis and choroido-retinitis.

In a similar case I would venture to recommend a small iridectomy; the operation would be quite justifiable and the microscopic examination of the excised portion of iris would be of extreme interest.

Quinine Amaurosis and Malarial Choroido-Retinitis.—Dr. Zanotti reported a case of quinine poisoning. Patient, a man, aged 38, swallowed 12 grains of quinine for a wager. Within half an-hour he lost consciousness and became violently convulsed. On recovering consciousness some hours later he complained of flashes before the eyes and loud noises like thunder in the ears; people in the room appeared like moving shadows. Medical aid was not sought until forty hours later; amaurosis was then complete, pupils widely dilated and immobile, hearing quite gone on the right side, much diminished on left, taste and smell unaffected, patient in a state of hebetude, which persisted for six or seven days. The amaurosis remained complete for ten days and then vision gradually returned by almost imperceptible gradations. A year afterwards his visual condition was as follows: Visual acuity $=\frac{5}{7}$ normal, fields greatly contracted, achromatopsia for green, dyschromatopsia for the other colours, night-blindness; ophthalmoscopic examination showed white discs, filiform vessels, little hazy white spots around discs and maculæ—probably choroido-retinal changes following hæmorrhages.

Incidentally Zanotti mentioned the case of a young girl, aged 20, suffering from malarial choroido-retinitis for which quinine was administered. After each dose (·75 gramme) dimness of vision was complained of, and on examination pallor of the discs and contraction of the vessels could be seen.

This case forms a valuable contribution to the literature of quinine amaurosis. It differs in some important respects from the case I reported in the Journal of September 15 and November 15 last, and is, I believe, the first in which microscopic changes in the choroid and retina have been observed.

FRANCE.

An Epidemic of Psittacosis.

Nicolle, writing in the January number of the *Archives Provinciales de Médecine*, describes a small epidemic of psittacosis which occurred among the inhabitants of one household in Bernay. It occurred subsequently to the arrival of a parroquet which had been bought in Paris. The bird, when it arrived fourteen days before the beginning of the epidemic, was distinctly out of sorts, and was suffering from diarrhœa. During meal-times it was allowed out of its cage, and was fed by the different members of the family, either from the hand or the mouth. Shortly after its arrival it bit three of these persons deeply enough to draw blood. The parrot recovered, but a fortnight after its arrival one member after another of the household took ill. Seven persons in all were attacked within a fortnight, and four of these died.

The features of the disease were either pulmonary symptoms in the form of localised pneumonia, *with marked absence of expectoration*, or intestinal symptoms, or more commonly both combined. All the cases showed the typhoid state.

Of the seven cases, six were apparently the result of direct infection from the bird. The seventh case was that of an old sick-nurse who attended one of the patients, and who had not been in contact with the parrot, which had been dispatched to the laboratory of Rouen.

A dog in the house also became ill and suffered from diarrhœa.

The bacteriological investigations were carried on in the laboratory of Rouen. In their researches for the purposes of isolating the bacillus the investigators made use of expectoration, blood, and fecal matter of the patients, but they were unable to isolate the bacillus described by Nocard. They found, however, that Nocard's bacilli were agglutinated by the blood serum of some of the patients, but as they had serum from only three of the cases, the investigation was not a completely thorough one. The blood serum of one patient also agglutinated typhoid bacilli, though not so rapidly as those of psittacosis. The writer remarks that the blood serum of animals inoculated with cultures of the bacillus psittacosis usually shows itself very active towards typhoid bacilli.—*Scottish Medical and Surgical Journal*, June.

INDIA.

Plague in Monkeys and Squirrels. By Dr. Alice M. Corthorn, M.B.

On Plague Duty, Bombay.

My attention having been called by F. K. Vincent, Esq., Chief Plague Authority in Gadag, to the fact that several monkeys had been observed to drop dead from the trees, I asked him to try and procure for me some specimens for examination. This he kindly did, and on December 10, 1898, I examined the bodies of two monkeys picked up that morning and in both of which *rigor mortis* was well marked.

Monkey I.—In the first there was a bubo the size of a large cherry in the left groin, which on dissection consisted of a group of four glands, hard and purplish in colour with a good deal of œdema of the surrounding tissue. From this I made a smear preparation, which showed an enormous number of organisms resembling the plague bacillus; and I also inoculated a tube of agar, on which I found within twenty-four hours plague-like colonies.

The spleen was much enlarged, and from it I made both microscopical and culture preparations with the same result as from the bubo.

Monkey II.—In the second monkey there was a small bubo about the size of a bean with some fulness in the right axilla. From it I obtained a successful smear preparation, but the culture was a failure. The spleen resembled that in the first monkey, and gave both a good smear preparation, and a growth of plague-like colonies within twenty-four hours.

The three tubes were sent to Dr. C. J. R. Milne, I.M.S., of the Plague Research Laboratory, Bombay, for further examination.

In both monkeys the general appearance was similar to that observed in human subjects dead from plague. There were numerous subserous and submucous ecchymoses and the blood was dark and unduly fluid.

Squirrels.—On December 17 the body of a grey striped squirrel was brought to me. I found no bubo, but the spleen was nearly 3 inches long, about 1 inch wide and $\frac{3}{4}$ inch deep. A smear preparation from it showed typical bacilli in large quantities, and a growth was obtained

on agar which was sent to the laboratory for further examination.

NOTE BY C. J. R. MILNE, M.B.
Captain, I.M.S.

(*a*) *Monkeys.*—Miss A. M. Corthorn, M.D., on plague duty at Gadag in the Dharwar District (Bombay), sent me on December 12, three sub-cultures from the spleens, &c., of dead monkeys. She says in her letter: "Both monkeys were observed to drop dead from the trees, and about seven monkeys have been observed to do the same thing within the last few weeks." The tubes were contaminated with various organisms, which is not surprising when one considers the conditions under which Miss Corthorn was working. Several plague-like colonies were noted in each, and these were transferred to and spread over fresh agar slants. By successive sub-cultures a pure growth of plague was obtained, and this was finally transferred to cocoanut-oil bouillon in which the characteristic stalactite growth was observed in about three days. On January 2, ·5 c.c. of a five days' old culture in bouillon was injected into a healthy rat. The rat died sixty-seven hours after inoculation and the tissues were found to be swarming with plague bacilli. Sub-cultures were made from the heart's blood, liver, &c., and these all exhibit pure plague growths. Miss Corthorn, I may add, notes that the monkeys examined by her had buboes, and one of her cultures was made from a bubo.

(*b*) *Squirrels.*—On December 17, 1898, Miss Corthorn examined a grey striped squirrel's body and made cultures from its spleen. A suspicious growth occurred in twenty-four hours, and she sent on a culture to the laboratory, which reached me on December 22. The appearances were exceedingly plague-like and sub-cultures were at once made. Pure cultures of plague were obtained on agar and in bouillon, similar to that noted in the case of the monkey. A rat inoculated with ·5 c.c. of a 5 days' old culture in bouillon died of plague in seventy-nine hours.

The importance of the fact that animals such as monkeys and squirrels are also susceptible to natural plague infection in addition to rats cannot be over-estimated, especially in their relation to the carrying of infection from one district village to another. These squirrels and monkeys are so common in India that they might well be considered as pests.

These investigations are being still further pursued, but Mons. Haffkine considers that the results of the investigation so far are so important as to be worthy of immediate publication.

Mr. H. M. Phipson informs me that the monkeys referred to by Miss Corthorn are probably the "Bonnet monkey" (*Macacus Sinicus*), while the squirrel is the "Palm Squirrel" (*Sciurus palmaeum*).—*Indian Medical Gazette*, March, 1899.

AMERICA.

DYSENTERY.

Johnston prescribes magnesium sulphate two drachms, with aromatic sulphuric acid, five minims, every four hours, until plenty of bile appears in the stools. He then gives ox gall in water. Dysentery is said to disappear in two or three days under this treatment.—*Medical Brief.*

TREATMENT OF CEREBRO-SPINAL FEVER.

In Professor William Osler's Cavendish Lecture on the Etiology and Diagnosis of Cerebro-Spinal Fever, delivered at the West London Medico-Chirurgical Society, the following remarks were made on the treatment of the disease. In our series of cases we have used no special drugs. Morphia has been freely given to control the pain. Ice sponging has been employed whenever the temperature reached above 102·5° Our mortality, considering that the cases were, as a rule, quite severe, has been low, only eight of eighteen cases in the Hospital, and nine among the twenty-one cases which I have seen.

I have already spoken of the possible benefits in certain cases of the relief of pressure by the withdrawal of cerebro-spinal fluid.

In two of our cases the spinal canal has been opened, drained and irrigated. Winter (*Lancet*, 1891), in tuberculous meningitis, removed the spinal process of the second lumbar vertebra and drained the spinal canal. Von Ziemssen first attempted to use local therapeutics to the membranes of the cord by injecting a weak solution of iodine in a case of meningitis; and later Sahli practised permanent drainage through a canula and catheter. So far as I know, an extensive laminectomy had not been done for acute spinal meningitis until our first case, November 6, 1898, in which the operation was suggested and performed by Dr. Harvey W. Cushing, the first assistant in the surgical clinic of my colleague, Professor Halsted. The case has already been given in abstract under the section on bacteriology as the one in which the staphylococci only were present at the time of operation. The spinal canal was thoroughly irrigated with salt solution and a quantity of purulent exudate washed out. No change followed in the existing paraplegia. The bladder and kidneys became infected, and he died about two months after the operation. The paraplegia persisted. At autopsy the spinal meninges were smooth and looked perfectly normal. It was impossible to say where the dura mater had been incised, and there were neither adhesions nor areas of the thickening on the pia-arachnoid. There were extensive changes in the cord itself and in nerve roots.

In case 12 laminectomy was performed on the fourth day by Dr. Cushing. The patient was very ill, and the spinal symptoms were especially pronounced. A catheter was passed beneath the dura mater, and the membranes drained and irrigated with salt solution. For several days he seemed much better. He developed a hæmorrhagic cystitis and pyelonephritis, and died on the sixth day after operation.

Dr. Musser, of Philadelphia, has also had an unsuccessful case. Dr. Rolleston and Mr. Herbert Allingham have reported a case of sporadic cerebro-spinal meningitis, in which the patient recovered after laminectomy and drainage. On the principle of a desperate remedy for a desperate disease, the operation (which has been criticised adversely in an editorial note in the *Philadelphia Medical Journal*), seems justifiable in certain severe cases, in which, as in our first case, the spinal symptoms are very marked.

GERMANY.

UROTROPINE IN THE TREATMENT OF BACTERIAL DISEASES OF THE URINARY TRACT.

Dr. S. Ehrmann, Vienna, writing in the *Therapist* of May 15, 1899, says:

"In 1894, Nicolaier discovered that, on the addition of formalin to urine, which otherwise deposited abundant crystals of uric acids and urates, these conditions were prevented. As formalin, owing to its being too poisonous, could not be administered, Nicolaier prescribed, internally, hexamethylentetramine, formed by the action of formaldehyde on ammonia, and which he called urotropine, and thereby obtained the same results, so that the internal therapeutic administration of formalin in uric acid diathesis (see below) could be entertained.

"As a dermatologist, I had, of course, no opportunity of testing urotropine in uric acid diathesis, but I have employed the remedy in the treatment of some bacterial diseases of the urinary tract for about two and a half years, and think I may be allowed to give an opinion as to its value.

"From my experience, it is undoubted that its sphere of influence in this respect is a very extended one. Nicolaier has already found that by the addition of formalin to urine its ammoniacal fermentation is prevented, and it suggested itself, in cases of cystitis with ammoniacal fermentation of the urine, to administer urotropine internally, in order to prevent the latter. Loebisch has clearly detected for-

maldehyde in the blood after the administration of urotropine, and also, as could be expected, that it was excreted in the urine; in which it has also been detected by Casper. I have in five out of twelve cases detected formaldehyde in the urine after giving urotropine; in three I found it once, but not a second time. It seems, therefore, that its excretion by the urine is not an even one, which explains the contradiction which has been met with in literature regarding Casper's statement. When examining, I distilled the urine, and detected formaldehyde in the distillate by means of fuchsin, discoloured by sulphurous acid, being again turned red; or by the reduction of $AgNO_3$ (as silver mirror). In most cases, however, I tried both ways.

"For the first time I gave the remedy to a person suffering from peri-urethral abscess and cystitis, following gonorrhœal infection. The urine was alkaline, and the sediment contained phosphates of ammonia and magnesia, urate of ammonia, gonococci, and other bacilli, such as Coli bacillus vibrionen. After the abscess had burst and healed, I commenced washing out the bladder, but for two months it was impossible to get the urine clear and a disappearance of the bacteria; but after giving 7½ grains of urotropine three times a day the urine was completely clear within three weeks. The bacteria rapidly decreased in number until they absolutely disappeared.

"Urotropine was continued for another month after the local treatment had been discontinued, and now, after two years, the patient has clear urine.

"I treated eight other very similar cases of cystitis with a similar result. I also treated six cases of bacteriuria with urotropine, and had the following results:—In five of the cases, in which no change of the prostate or seminal vesicle could be discovered, the bacteriuria was permanently cured with urotropine. In three casee 22½ grains of urotropine were given daily, in three doses of 7½ grains each, and for a further month 7½ grains twice a day. In two cases 22½ grains of urotropine were given daily for six weeks, 15 grains daily for four weeks, and, as, after discontinuing, a slight cloudiness reappeared, we again continued giving 22½ grains daily in one case for three weeks, and in another for four weeks, after which the bacteria and cloudiness disappeared from the urine. In one case, in spite of urotropine, bacteriuria continually reappeared. This was a case of a patient who had suffered from prostatitis and spermatocystitis gonorrhoica along with cystitis, whilst the other five cases suffering from bacteriuria had only chronic gonorrhœa, which for a long time had been treated by means of sounds and deep injections with Guyon's or Ultzmann's syringe. One patient had, also, a stricture, which he had himself been in the habit of treating with a gum-elastic catheter. There can be no doubt that in these five cases bacteria got into the bladder by means of the instruments. In the first case, which had continued relapses, however, it was a question of a fine communication with the rectum, by means of which the bacteria re-entered, although those in the bladder were completely destroyed."

LOCAL TREATMENT OF DIPHTHERIA BY LOEFFLER'S SOLUTION.

In addition to the injection of antidiphtheritic serum, or when it is not employed, Professor Loeffler (*Deutsches Medicinische Zeitung*) makes use of the following applications with most beneficial results. When the disease is still localised to the uvula or tonsils, treatment by this method almost certainly checks the further spread of the disease. The investigator found that carbolic acid dissolved in alcohol, when applied to the diphtheritic patches, destroyed the germs in twenty seconds. More rapid results were, however, obtained when such substances as benzol or toluol were added. The addition of menthol is advocated when young children or nervous patients have to be dealt with. The mode of application is as follows:—The mucus is first removed from the affected portions with a cotton mop. Another swab saturated with the following mixture is then applied and kept in contact with the diphtheritic patch for ten seconds:—

℞	Menthol	10 grm.
	Toluol	ad 86 c.c.
Then add—	Solve. Misce.	
	Creolin	2 c.c.
	Sesquichloride of iron solution ...	4 c.c.
	Absolute alcohol	ad 100 c.c.

After the first, another application must be immediately made. The treatment should be kept up for four or five days at intervals of three hours. At the end of this time the disease will probably have disappeared. In cases of mixed infection, as where staphylococci or streptococci are present, this treatment is not so effectual as it is with pure growths of the Loeffler bacillus.—*The Scottish Medical and Surgical Journal*, June.

THE INFLUENCE OF SOIL IN EARTH BURIAL. — The suitability or otherwise of a soil for the site of a cemetery depends on the same conditions—viz., the presence or absence of nitrifying bacteria, its permeability to air, and its humidity and drainage—that determine its fitness or unfitness for the treatment of sewage. In the one class of soils bodies undergo desiccation, and the soft parts "return to dust" in the course of a few years; in the other they putrefy, and instead of decomposition and disintegration they remain for years—in fact for indefinite periods—festering masses of corruption. An official inquiry in Germany some years ago showed that in the "loess" four years sufficed for the disappearance of everything but the bones and hair, whereas in stiff wet clays the bodies still presented stinking putrid masses; and in excavating the churchyard of St. Andrew's, Holborn, pre-eminently a "dead" earth, there was little difference to be seen between the interments of twenty and of two hundred years previously. Yet with few exceptions the situation of the cemeteries around London makes it clear that, whatever considerations determined the choice of the localities, the nature of the soil was not taken into account. That of the Necropolis at Woking is indeed an ideal one, but the district ought rather to have been developed into a health resort and been appropriated to sanatoria than converted into a city of the dead. These reflections have been suggested by reading the graphic description by Dr. A. Riedel in the *Münch. Med. Woch.*, June 6, 1889, of two exhumations, at which he was present, carried out by order of the judicial authorities, the one a year after burial in a stiff wet clay, and the other after eighteen months in a loose and well-drained sandy soil. In the latter, the body was dry and little changed in appearance. On opening the abdomen the odour emitted was not unpleasantly aromatic from the abundant formation of adipocere, and the internal organs were in such preservation that the uterus could be unhesitatingly pronounced that of a virgin, the allegation of pregnancy disproved, and the charge against a suspected party dismissed. The other case was that of a woman who had been buried twelve months in a stiff wet clay. It being mid-winter, and the snow lying deep, it had been arranged to conduct the examination of the body in a room at the workhouse; but this was found utterly impossible. The appearance revealed on opening the coffin was, says Dr. Riedel, more horrible than can be described or conceived by anyone who had not seen the slimy, gruelly, stinking mass of corruption, retaining litttle resemblance to the human form. The stench was such that the air for yards around was almost irrespirable. The gaping crowd assembled on a neighbouring hillside soon dispersed, and the few whom duty compelled to remain in the churchyard suffered from the poisonous emanations. He and his medical colleagues were for more than a week unable to get rid of the smell by repeated baths, and were with difficulty able to enter the houses of their patients. In compliance with the order of the court, the stomach and viscera were sent to the University for examination, but, as might have been expected, the results were wholly negative. Such an object lesson, he adds, would have converted the most bigoted opponent of cremation.—*Public Health, July*, 1899.

News and Notes.

BRITISH MEDICAL ASSOCIATION.

Meeting to be held at Portsmouth, on August 1, 2, 3 and 4, 1899.

L.—Tropical Diseases.

Committee Room, No. 1, Town Hall.

President: George Thin, M.D. *Vice-Presidents:* Colonel Kenneth McLeod, M.D.; James Watson, M.D. *Honorary Secretaries:* Fleet-Surgeon John Tyndall, R.N., Tresco, St. Ronan's Road, Southsea; John Robert Stevenson Robertson, M.B., 5, Western Parade, Southsea.

The subjects arranged for discussion are as follows:—

1. Psilosis or Sprue; its Relations (Etiological and Pathological) to Other Forms of Tropical Diarrhœa, and its Treatment.
2. On Thermic Fever (so-called Siriasis), with Special Reference to its alleged Microbic Causation.
3. The Agency of Insects in Spreading Infections.

Amongst those who have consented to take part in these discussions are Dr. Thin, who will open the first subject, and Colonel Kenneth McLeod, I.M.S., the second; Major Ross, I.M.S., will open the discussion on the third. Major John Ritchie, R.A.M.C., will take part in the discussion.

The following also intend to take part in the above discussions or send papers: Dr. Patrick Manson, LL.D., F.R.C.P.; Inspector-General A. Turnbull, M.D. R.N.; Dr. W. J. R. Simpson, Mr. James Cantlie, F.R.C.S., Colonel Crombie, I.M.S.; Fleet-Surgeon H. M. Ellis, Royal Marine Artillery; Dr. James Watson, Surgeon Andrews, M.B., R.N., Captain Hughes, R.A.M.C., and Surgeon Stalkart, M.D., R.N.

The following papers have been offered:—

Andrews, Surgeon, O. W., R.N. Antivenene.
Cantlie, J., F.R.C.S. "Supra-hepatic Abscess."
Childe, C. P., F.R.C.S. A Case of Bilharzia Hæmatobia with Specimens.
Hughes, Captain, M. L., R.A.M.C. The Geographical Distribution of Undulant (Malta) Fever.
Manson, Patrick, M.D. Filaria Periodicity.
Stalkartt, Dr. Blackwater Fever.
Yarr, M. T., Major, R.A.M.C. A Further Contribution to the Study of Malarial Eye Affections.

The Opium Habit in the East.—The extent to which the opium habit prevails amongst Europeans in the East is only properly appreciated by chemists, as they are brought into contact with the victims of the vice even more than the medical men themselves; they will, therefore, I am sure, be greatly interested in the announcement made by Dr. Neil McLeod, of Shanghai, that it is possible to cure the habit by the administration of sodium bromide. He gives the drug "in two doses of two drachms, in solution, every two hours for the first two days, and one drachm on the third day," and adds that "three ounces of the drug in all will probably suffice in most cases." The treatment is quite safe, and well worth trying.

Report of the Peking Hospital for the Year 1898.—The thirty-seventh annual report of the Peking Hospital of the London Missionary Society is interesting in every way.

The medical work is, of course, of most direct interest to us, and in the wards of the hospital and in the outpatient departments an immense amount of excellent work has been done. Under the direction of Dr. Eliot Curwen and his Chinese assistants, some 32,000 attendances were made.

Some fifty-one patients were admitted for the cure of the opium habit. In several instances a cure was effected after keeping a careful watch on the sufferer for three weeks.

Sandrons (Limited) inform us that they have decided to adopt the title "Liquor Ferri Assim. (Sandrons)" for "Sandron's Assimilable Iron Tonic." This they do on the suggestion of several leading London consultants who prescribe it. The company also inform us that the tonic is now obtainable in 12 oz. and 18 oz. bottles, unstamped for dispensing, as well as in the usual stamped retail form. Both kinds can be obtained direct from Sandrons (Limited), 34, Devonshire Street, Portland Place, W., or from any of the wholesale houses.

Epidemic Jaundice.—During the autumn of 1898, Peking and Tientsin were visited by an epidemic of jaundice.

The first case seen was on October 22, and the last just before Christmas-day, and few days passed during these two months that one, two, or three cases did not come to our dispensaries for treatment. Their ages varied from 17 to 60, and seven times more men applied for treatment than women. The attack from which one of my students suffered was typical of the series. On October 27 and 28 he told me he felt weak and lethargic; on the evening of the second day he returned to his home feeling sleepy, ate two pears, had pain in the abdomen and felt sick; at night he was very hot, complained of severe pain in his temples and says his conjunctivæ were suffused. During the next two days his temperature kept up between 102° and 102·4,° he felt very ill and the pain in his temples continued; calomel, grains three, resulted in five dark-green motions. On November 1, temperature was down to 96° in the morning; the conjunctivæ were slightly yellow for the first time; pulse 60; no tenderness over the liver or abdominal discomfort; still very weak. Tongue slightly furred. Urine gave Martin's and Gmelin's reactions. The colour of the conjunctivæ became more marked by the 4th, when he began to complain of itching of the skin. From this date his symptoms gradually improved; ten days later, however, the conjunctivæ were still pale-lemon coloured. Some of my patients were severely ill for some days, while a few others were affected so slightly that they did not know there was anything the matter with them till I gave them a looking-glass and told them to look at their eyes. Most of them, however, complained of great weakness

and some of shortness of breath; there was generally a feeling of abdominal distension and nausea, and constipation was the rule. More than two-thirds of the patients told me that for the first few days, varying from one to ten, they suffered from some degree of fever; in five cases only were the conjunctivæ colour noted as deep, in the great majority of cases the colour being a pale straw or lemon colour and not deep enough to be noticed in the yellow skins of the Chinese; only three men told me their skins were irritable. Another prominent symptom was pain in the temples, and this in some cases was described as very severe; it was noted in two-thirds of the cases. The liver was noted as slightly enlarged and tender in two patients only. Either Martin's or Gmelin's test reacted in each case tried. The pulse was rapid, from 80 to 128, in each case with the exception of the one narrated above; giddiness was only complained of twice, and the only other nervous symptom noted was in the case of a man of 60, whose fever lasted ten days, who told me that about the time his eyes became yellow he began to suffer from pains in the muscles of the right arm and in his two ulnar bones, the muscles and bones were still tender on deep pressure when I saw him. Only one patient told me he had neighbours suffering from this complaint, but many attributed their attack to the common cause of all illness in China, namely, "loss of temper." The treatment adopted was, I fear, as unsatisfactory as the results of my enquiry into the etiology of the disease; fat, sugar and spirits were interdicted, and the patient put on a course of sulphate of magnesia, or, when diarrhœa existed, of creasote; nitrohydrochloric acid was exhibited when needed in the third week.

MARRIAGE.

CANTLIE—FARQUHAR.—On June 28, at the Union Church, Hong Kong, by the Rev. S. H. Wainwright, M.D., of Kobe, Japan, Frank Hay Cantlie, M.B. C.M., Swatow, China, youngest son of the late William Cantlie, Keithmore, Dufftown, Banffshire, to Isabella Ann (Annie), third surviving daughter of Robert Farquhar, Esq., 455, Great Western Road, Aberdeen, Scotland. (By cable.) American and Canadian papers please copy.

Letters, Communications, &c., have been received from:—

B.—Dr. R. C. Bennett (W. Kensington).
F.—Staff-Surg. R. A. Fitch (Cape of Good Hope).
M.—Dr. R. U. Moffat (P.M.O. Uganda), Knochholt Mr. H. A. Moffat (Buluwayo); Surg. W. B. Maurice (Mediterranean).
N.—Dr. F. Neal (Demerara).
P.—Dr. Albert Plehn (Driezmin).
S.—Rev. James Sandilands (New Hebrides); Dr. R. A. Shekleton (Dublin).
W.—Dr. John Weddick (Hawaiian Islands).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Clinical Journal.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
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2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."