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DENGUE or "THREE DAY FEVER".

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The nomenclature of certain pyrexial conditions still remains a matter of considerable doubt and confusion, a state of affairs which will continue until more precise knowledge of their etiology is forth-coming. The acceptance of such terms as "THREE_DAY FEVER" and "SEVEN_DAY FEVER"as adopted by Major Rogers can hardly be universal until their individuality is unquestionably established by the discovery of a definite path--ogenic agent. In a recent paper in the Indian Medical Gazette Capt Megaw I.M.S. has demonstrated the close connction between Dengue and "Three-Day fever", expressing the view that they are one and the same disease.

During the early months of this year, there occured an epidemic of fever cases on this Island closely res--embling in its symptomatology, that described as THREE DAY FEVER. It seemed therefore that a short account might prove interest, though nothing new regarding etiology can be advanced. To this series of cases it did not at first seem justifiable to give the diagnosis of Dengue, lacking as it did several of the most recog-nized characteristics of that complaint; as by doing so the term would have to cover several minor conditions and lose in definition what it gained in elasticity.

HISTORY. On April 14th a cargo vessel arrived from Calcutta, the crew was a European one and the Captain reported, that they had had a few cases of Fever since leaving Port, but that they had been of a very trivial nature.

At this time the weather on the Island was hot and damp, the Meteor-
-ological Report shewed a constant rainfall with a high degree
of atmospheric humidity. Two days after the arrival of the vessel
5 coolies came out to Hospital with Fever, they had all been
working on board -trimming; from that date onwards there were
were constant daily admissions to Hospital of similar cases;
so that almost all those at work on the wharf suffered; while
it is of interest to note that practically no cases were admitted
to the Hill Hospital at a distance of 1½ miles from the wharf.
It was not found possible to admit all cases to Hospital, but
all with a temperature of 102 F. or more and those whose temper-
-atures had not returned to normal by the same evening received
admission, a total of 165 cases being entered in the books.

SYMPTOMATOLOGY. — From first to last the patients
presented themselves with ~~almost~~ identically the same ~~symptoms~~ complaints,
so that the reiteration of them became almost monotonous -FEVER-
-HEADACHE- -PAIN in the BACK- . The fever was of sudden onset
and varied between 102°F and 104°F, there was no initiatory rigor.
The face was slightly flushed in about 50% of cases and there
always intense frontal headache; the coolie invariably put his
finger and thumb to the temples, or came decorated with Chinese
plasters. There was never any Rash, though it was carefully search-
-ed for. The pain in the back was limited to the Lumbar Region
and often very acute, the limbs were quite free from all discom-
-fort and stiffness. The tongue was almost always furred in the

centre and red at the margins, but beyond a general feeling of malaise there were no gastric symptoms and there was no vomiting. The bowels were constipated. The Pulse-Temperature Ratio did not correspond, for with the high fever there was little correspond--ing acceleration of the cardiac action, the Pulse Rate being seldom above 85; while the Maximum Systolic Pressure registered between 115 and 125 m. m. of Hg. In 55% of the cases soreness of the throat was admitted on inquiry, but had not been complain--ed of and on examining these cases, there was congestion of the Uvula and Fauces, but no membrane formation and no glandular enlargement. In only 10% was there any coryza present and it was ~~only~~ but slight in degree. In 32% cough was complained of and a scanty muco-purulent sputum was expectorated.

The temperature fell in one or two days and in almost every case the decline took the step-like appearance, noted by Major Rogers and well seen in the accompanying charts. With the defervesence of the temperature the headache and lumbar pain vanished without any epistaxsis or diarrhoea and the patients appetite returned; only in a few cases was there any feeling of debility, giddiness and lack of energy and then ~~only~~ of short duration. The cases were carefully watched for a terminal rise of temperature, but it was never apparent. There was never any subsequent stiffness or rheumatic pain in the joints, and though constantly looked out for no rubeolar or other rash showed itself. There was also never any desquamation nor any of the sequelae sometimes seen af

after Dengue namely- sleeplessness, irritability, furunculosis and the like. In no case did a recurence at a later date occur one attack apparently provided immunity.

EXAMINATION OF THE BLOOD. The leucocytes were always diminished and in some cases a marked leucopenia was found, being as low as - 3,700, but the average was 5.800. The polymorpho-nuclear leucocytes were slightly decreased averaging 65%; while the large mononuclears were rather increased averaging 15%, the lymphocytes 18•5%, and the eosinophils 1•5%. Reduction of the number of Red Blood Corpuscles was noted in a few cases with a corresponding fall of the Hoemoglobin percentage. The blood was examined in almost every case, but only after the onset of fever, as the cases came under observation when they felt ill; were not seen before; the above averages are based on the results obtained from a 100 cases. Examination of Blood Films was always negative both to any Malarial Plasmodium and to any Spirillum. In three cases two red blood corpuscles were seen to contain a quantity of deep staining intra-corpuscular bodies somewhat like gonococci, staining dark blue by Leishmans method, arranged more or less in pairs and apparently unpigmented possibly the same condition as described by Grahame in relation to an outbreak of Dengue. This was not likely to be a Malarial manifestation for the coolies in whose blood the appearance was noted, had been from 4 to 6 years on the Island, where, owing without doubt to the absence of the Anophelinae, cases of Malaria do not occur. Cul-

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Cultures on Agar-Agar and Blood Agar made from the blood of these cases were negative and so as the appearance was only noted in 3 cases little importance can be attached to it, as the causal agent.

EXAMINATION OF THE URINE. —There was little alteration; during the fever there was diminished excretionof urine,high coloured, and with a raised Sp. Gr. but only occasional a trace of Albumen. The Chlorides were diminished in one case amounting to ~~only~~ .6%, as estimated by Mohrs method. No abnormality was seen microscop-ically and cultures inoculated proved quite negative.

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EXAMINATION OF THE SPUTUM. —In only a limited number of cases was there any sputum present, but in these it was frequently examined and it is of interest to note, during the height of the fever , a spirochaete was invariably found, but often ~~only after~~ requiring prolonged search; on the other hand after the subsidence of the fever it was not found. In character it was very similar to the ~~Spir:~~ Pallida, and stained with Geimsa and dilute Carbol Fuchsin, but not with Gram. It was of delicate construction and had occasionally a bifed terminal and in one case was distinctly nodular or striated in appearance. Having no Mic-rometer these were not measured, but to the eye it seemed to be finer and more delicate than S. Dentium of the mouth and as noted above was not found after defervesence. Swabs from

the throat were taken and cultures made in Broth and on Agar-agar and Blood Agar, but with negative results, only B. Buccalis and and Lept: Buccalis ~~being found.~~ & other organisms common to examination of the saliva being identified.

TREATMENT. — Very little was required; isolation of cases as far as possible was attempted. The patient was put on a milk diet, but as far as the administration of drugs was concerned, the short duration of the fever rendered it hardly possible to decide, which had really a beneficial effect. A brisk purgative was given at the outset and if necessary a Belladona plaster applied to the back. Quinine, Asperin and Sodium Salicylate were adminsitered, the latter seemed if anything the most satisfactory, followed by a simple tonic. The patient left fit for his usual work at once.

DIAGNOSIS. — When these cases presented themselves Malaria was eliminated by the blo od examination. Influenza was negatived by the absence of coryza and of serious respiratory complications, by the character of the temperature curve and the examination of the sputum. The diagnosis of Dengue seemed hardly justifiable, considering that the initial stage of skin congestion, so characteristic of this complaint, was practically never present, also there was complete absence of joint pain and stiffness and of the terminal temperature and rubeloid eruption. It was therefore considered as probably similar to "THREE DAY FEVER", but on 20th July a ship arrived from Bombay with fever on board,

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and a precisely similar outbreak to the above occured, but more limited in the extent. In this case, however, there was typical dengue on board the boat. One is forced therefore to believe, that probably both outbreaks were due to the same origin;-infection by the causal agent of dengue in an attenuated form. The notes on this epidemic thought obivously lacking in any finality are of, some interest for the question of the differentiation of "THREE DAY FEVER" from Dengue as a disease per se is still an undecided question. It is recognized that the similarity on broad lines is very close and from the above epidemic, one is inclined to believe, that Dengue maybe represented in such a mild and attenuated form as to have lost what are usually considered to be its most characteristic symptoms and thus apparently to be strictly analogous to what has been described as "THREE DAY FEVER".

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The Water Supply of Towns in the Tropics, chiefly from the Bacteriological Standpoint, as illustrated by the Water Supply of Khartoum

by

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As events are trending there may yet be some justification for a lack of faith in the value of Bacillus Coli communis as an indicator of pollution of a potable waters. You are aware that this organism has been put forward by Houston, Savage, Jordan & others as, with certain restrictions and reservations, a reliable test of the fitness or otherwise of

a water-supply. This view has recently received confirma-
-tion at the hands of Portuguese investigators of the Guirand and Mandoul in France, and has also
been upheld by Fromme in a lengthy paper in the Zeitschrift
fur Hygiene. On the other hand there seems to be a growing
tendency to regard it as mistaken and I need merely mention
recent expressions of opinion by Starkey and Russel McLean
in this connection. I have neither the time nor the desire
to plunge into this vexed question so far as water-supplies
generally are concerned but I would direct your attention to
it as regards water-supplies in the Tropics. Until recently
records of bacteriological investigations of water supplies
in tropical countries have been few and far between. This
is natural enough, as, hitherto, facilities have not existed
for work on these lines, but with the establishment of well-
equiped laboratories in many parts of the tropical world it
is probable that ere long abundant statistics will be available
and that we will be in a better position to appreciate the
differences which undoubtedly occur in the bacterial content
of tropical waters as compared with those in temperate
climates and the possible necessity for modifications of
existing standards of bacterial impurity.

In the Tropics the water-supply of a town or village is
usually derived from one of two sources - from wells, usually
shallow wells, and from rivers or streams.

In Khartoum, for I intend to take the problem of water-
-supply there as an illustration of the whole question, the
supply used to be obtained from both these sources, the river
being the Blue Nile - a mighty stream springing from Lake
Tana in the Abyssinian highlands and fed for the most part
by the torrential rains which fall at one period of the year

on the mountains and tableland of the ancient Kingdom of Ethiopia.

Before, however, entering more minutely into the condition of affairs which obtained and now obtains at Khartoum let us see if there is any recent work to guide us in our consideration of water-supplies in the Tropics, both from a general and from a bacteriological stand-point. As stated, this is not easy to find but an excellent article on drinking water in India and its connection with the subsoil water is that by Lt-Colonel Dawson[6] of the Indian Medical Service. He advocates the preparation of contour maps of the subsoil water and deals with the filtering capacity of soil. One point, he specially brings out and it is one with which I must deal later and which I think has received but scanty notice and that is that the subsoil contains much clay, especially black clay, in which there is a large per-centage of organic matter, a process of purification by natural filtration is impossible and there exists indeed, a culture medium. "Caution must be exercised", he says, "when reliance is placed upon the purifying property of the soil, especially if its characteristics be unknown, for the soil is after all a filter, and all filters, whether artifical or natural, are treacherous, and are a source of danger if not constantly attended to by some competent person." He also comments upon the presence of disused wells in India and their use by natives for insanitary purposes whereby the subsoil water is apt to be contaminated.

Turning now to papers* dealing more exclusively with the bacteriological aspect of the question we find Daniels[9] and Finlayson reporting upon the natural waters of the Federated

* The work of Edwards[7] on the water derived from areas of the deltas of Philippine rivers scarcely merits consideration. He merely says that waters should be condemned (a) for the presence of animal parasites (b) for the presence of pathogenic bacteria + (c) when the colony count is above 200 per c.c. A paper by D. Kabrak[8] in the Trans. of the B. Inst. Chem. is more speculative than helpful.

Malay States. These are jungle streams and Daniels remarks that the organisms present in such water are abundant and differ in type from those met with in temperate climates or in tropical climates where the rainfall is less uniform and vegetation not so rank. It is therefore, evident that no general rule can be applied to water-supplies in tropical countries as a whole. Each must be judged on its merits and it will be a long time before sufficient facts will have accumulated to enable us to compare the waters of hot, dry, desert countries like the northern Sudan with those of steamy, humid regions where vegetation is rife and the rainfall is heavy.

As regards samples of water taken from such jungle streams Daniels considered it advisable to consider as a basis:-

1. The number of organisms exclusive of known, easily recoghised, non-pathogenic organisms e.g. B.subtilis, B. megaterium, B. prodigiosus, B. violaceus, and some of the organisms which form characteristic yellow colonies which have been proved to be non-pathogenic to lower animals.
2. The amount of the water required to react, forming acid and gas, with MacConkey's medium, from .2 c.c. to 2 c.c. being employed in the tests.
3. Indol formation in 48 hours in peptone water with 10 c.c 5 c.c. and 1 c.c. of the water to be tested.

As a standard the two observers suggested that:-

1. Not more than 100 organisms exclusive of those mentioned should be present in 1 c.c. of the water.
2. That no acid and gas should be formed in 24 hours in MacConkey's medium at 37° C with 2 c.c. of the water.

3. That no indol should be formed in peptone water in 48 hours with 5 c.c. of the water added.

They remark that the standard is not a high one but that it is exceptional to find a natural water that will pass all three tests. In unprotected shallow wells liable to be polluted by surface washings they found the average bacterial count to vary between 300 and 900 per 1 c.c. while in 23 out of 27 wells examined acid and gas-forming organisms were present in 2 c.c. and usually in 4 c.c. of the water, and indol formers in 5 c.c. in the case of ten of the wells. B. pyocyaneus was found in one instance and in two cases organisms indistinguishable from B. coli communis.

However useful then this work was for local purposes it does not help us much in considering the question as a whole and until recently there were, so far as I know, no statistics of value in this direction. Happily the first step has now been taken by Major Clemesha and his assistants who have inaugurated in India a series of observations which may yet prove to be epoch-making and are already highly suggestive.

~~Let us consider very briefly what Clemesha set himself to do and at what conclusions he has arrived.~~ Major Clemesha ~~He~~ stated in the first instance, that there must be considerable doubt as to whether the standards of purity in common use amongst sanitarians in England were suitable to India. The simpler tests when applied to Indian water samples showed that most of the latter are loaded with faecal contamination and that no analyst in England would dream of passing them as fit for human consumption. And yet it is remarkable that the evil results of using such sewage as drinking water are not always apparent even on enquiry. True there are outbursts of epidemic cholera, dysentery, ~~like the poor, is always present~~

to some extent and there is a terrible infantile mortality but all things considered the results are not in proportion to the cause at all times. Again a great deal of the pollu-tion of surface waters in the East is caused by the excrement of animals, chiefly cattle and goats, whereas in England the pollution of rivers at least has its origin largely in the sewage from towns upon their banks. After these preliminary remarks and others which need not be quoted Clemesha proceeds to give an account of the bacteriological methods he and his assistants followed in their efforts to arrive at a suitable standard of purity for the drinking water in the Madras Presidency.

With these I need not trouble you beyond saying that MacConkey's procedure, somewhat modified, and including the use of special sugars was found of great value, that stress was laid on the value of determining the total colonies on agar, that the sporogenes milk test was adopted as was the reaction of Voges and Proskauer, while, as regards indol, the benzaldehyde reaction was employed. Further the short cuts which have been devised by Houston in his work for the Metro-politan Water Board were found to be of the greatest service. Having had the privilege of seeing Houston's methods in operation I can well believe this to be the case, especially in a hot country where every labour-saving device is to be commended.

An important chapter in Clemesha's report is that in which he considers the "coli" standard as applied to India. He points out how bacteriologists differ in their definition of B. coli communis and he gives the interpretations put upon it by four authorities, namely the English Committee, the American Committee, Houston & Savage. There are two main

schools of thought he says:-

1. "The position taken up by Houston, Savage, and nearly all other bacteriologists in England, who apply the term "coli" in an elastic sense to an organism defined as above, but, who recognize a large number of ill-defined varieties, termed by them "atypical coli" so that the term is in reality made use of to describe a group of varieties, if not different species.

2. That taken up by MacConkey, Drs. Bergey and Deehan, of the Pennsylvania University, Dr. Orr (in his recent work on the contamination of milk) and ourselves, who apply the name bacillus coli communis to Escherichs organism only, calling all bacilli that differ from it in any "permanent" test, different species, to which different names or numbers are applied."

"We in India" he continues "consider the ~~position~~ position maintained by the former authorities is open to two objections: (1) That it is inadvisable to apply the term 'coli communis' to a group of organisms, or even so-called varieties, and (2) The tests laid down as essential according to the definitions quoted, do not go far enough; that the list contains too many that are of doubtful significance and utility, and too few that are admittedly of greater importance in the identification of a bacillus. To discuss this last objection first: Houston, Savage and all experienced bacteriologists admit, that the tests given above are not of equal value in the identification of what they term 'coli'. Thus to quote from Savage, 'Differ--ent characters have not an equal value. Some, such as the fermentation of sugars, liquefaction of gelatine, and acid production in milk are of so permanent a type that for an isolated organism to show divergence from a typical bacillus coli in any one of these particulars would be to throw grave

doubt on its being excretal at all, while others, such as the gelatine surface colonies, and the exhibition of motility, are so subject to variation that comparatively little sign--ificance can be attached to their, perhaps temporary, absence or modification.' And again in another place he says: 'Organisms that differ from the typical excretal type (bacillus coli communis) only in the fact that they show no motility and that their gelatine colonies are atypical may be regardedas not having their significance diminished on these grounds. Similarly the loss of Indol-production power would not reduce their significance very markedly. On the other hand the absence of glucose or lactose fermentation would at once, in the writer's opinion, exclude the organism isolated from being bacillus coli at all."

The obvious inference from these opinions,is, why not do away with some of these doubtful tests and substitute for them the more important fermentation reactions in sugars such as dulcite, adonite and inulin? These are entirely on a parallel with the glucose and lactose fermentation referred to as being "of a permanent character." This is practically the position taken up by MacConkey, in his paper on the Bacteriology of Milk (Journal of Hygiene, Vol.IV, No.3, July 1906), and, following his recommendations, by ourselves in the work under report".

He proceeds to illustrate his argument by the results obtained by the application of the proposed methods to Indian waters. It is found that Houston's true B. coli can be split up into ten different bacilli while if his non-typical coli is also considered more are added to the list, a list which includes B. Gr[illegible]nthal, B. oxytocus perniciosus, B. coscoroba and others, distinguished so far only by numbers. According to

Clemesha the definitions of Houston and Savage err in omission as well as in commission. He says: "Take the organism bacillus cloacae as an example; it will be observed that this organism does not come within Houston's definition of "true coli"; because it ferments saccharose, nor within Savage's group that indicates foecal contamination, because it liquefies gelatine, yet it is undoubtedly foecal in origin. Why should the mere fact that this organism liquefies gelatine mean that it does not indicate foecal contamination to an equal extent as any non-liquefier? Surely considering it originally came from the foeces of man or some other animal, the coincidence that it liquefies gelatine, cannot put it beyond the range of indicators of foecal contamination. As a matter of fact B. cloacae is not an indicator of dangerous and recent pollution, because it is a resistant organism, w with a fondness for the bottom of lakes, and river sand; but the liquefaction of gelatine has nothing to do with these facts." and continues:

"The weak point of the above method lies in the statement that a large number of what we believe to be entirely, differ--ent species of bacilli equally represent dangerous foecal contamination. If this be so, it should be demonstrable that these different species are equally resistant to such forces of nature as the action of sunlight and the action of storage. Otherwise if it can be proved that one organism is particular--ly resistant, and another particularly susceptible to these natural forces, it is obvious that the resistant organisms cannot be considered, on all occasions, of equal value, as an index of recent and dangerous pollution, as the susceptible ones. Examining the ten organisms, which fall under Houston's definition of "coli", and which represent his worst type of

pollution, we are prepared to state from actual experiments, that, the greatest possible variation exists in these ten organisms in their power of resistance to the action of natural forces. To take two extreme examples B. Grunthal is particularly resistant to the action of sunlight. It is found in waters that have been exposed to the tropical sun for weeks; it is nearly always one of the last bacilli to dis--appear from a well-sunned and stored water, and also from a mixture of cowdung and water exposed to the sun. On the other hand, true B. coli communis of Escherich (the organism that ferments dulcit and does not ferment saccharose, adonit, inulin, etc.), will disappear with great rapidity from a water exposed to the action of tropical sun. It is seldom found to survive longer than three days in a clear water of considerable depth. It is reasonable to include both these bacilli under the same term of "true coli", and to state that both equally represent objectionable pollution? It is possible to elaborate this still further. In the course of our work we have found that under very exceptional circumstances, a bacillus known as Oxytocus perniciosus is occasionally found in a water-supply. There is good reason to believe that this organism is extremely susceptible to natural conditions, such as sunlight, etc., for although it is present in cowdung, and probably in human foeces, it has only been found in water supplies after heavy floods, and it disappears with extra--ordinary rapidity. Thus it is probable that this organism represents a more recent, and therefore a more objectionable contamination, than any other organism met with up to now. Yet it will be observed that this particular bacillus does not fall within Houston's definition of "true coli", nor within the category of organisms in which, according to Savage, are 'equally of excretal origin'."

Such being the case why, asks Clemesha, do the methods employed in England yield, as they undoubtedly do, such satisfactory results? He answers this as follows:-

"Firstly, the majority of the contamination in surface and river waters is due to human excrement in the form of sewage from towns.

Secondly, the guiding principles that assist a water analyst in arriving at a conclusion are based on the study of organisms present in sewage, that are human in origin. Thus, the reason Houston gives for saying that saccharose fermenters do not represent so objectionable a form of pollution as the non-saccharose fermenters, is, because the non-saccharose fermenters greatly preponderate in sewage. It should be noted that this kind of reasoning would obviously lead us into serious error in India, where much of our pollution is due to animals.

Thirdly, there is very little doubt that coli communis of Escherich (dulcit +, saccharose, adonit, inulin, Voges and Proskauer -) is probably the commonest organism found in human foeces, and in rivers polluted with town sewage. The work of MacConkey supports this view.

Fourthly, B. coli communis of Escherich is a very susceptible organism to the forces of nature, that are inimical to bacilli generally, and its presence therefore does actually represent a recent and dangerous contamination. Evidence in support of this statement can be found in Houston's annual reports, where the ratio between his "true coli", "confirmatory" and "presumptive" tests varies with the time of the year. Thus in summer the actual number of "true coli" is much less than in winter, due undoubtedly

to the action of the sunlight in killing off these susceptible organisms.

Consequently when an analyst in England finding "true coli" in 1 c.c. condemns a water, because it shows recent pollution, he is right in a great majority of the cases, and in the remainder the worst that can be said of his opinion, is, that he has slightly erred on the side of overcaution, for it must be remembered that the whole of the 10 organisms comprising his "true coli" are originally of foecal origin. Now in India things are entirely different. The coli communis of Escherich is a very rare organism in the waters of this country, whereas the other organisms that are included in Houston's definition are extraordinarily common".*

Considering these facts and also that the bacteriological flora of the faeces of man and animals varies at different times of the year he came to the following conclusions:-

1. That standards in use in cold climates are useless and worse than useless in tropical countries.
2. That it is necessary to separate the individual species of bacilli by well-established tests and to study their characteristics and their position in nature.
3. That it was advisable to classify all lactosefermenting organisms according to their ability to resist the action of sunlight and on this to base the standard of bacterial purity.

In the first place the bacteriology of earth known to be heavily polluted with human excrement was studied. The results are summed up as follows:-

(1) Bacillus cloacae, bacillus, grunthal, bacillus No 75 and to a less extent bacillus coscoroba are capable, under favourable conditions, of remaining alive in the soil for a considerable length of time, probably up to 3 or 4 years.

* In this connection one may note that research work by Castellani" goes to show that the typical B. coli of Escherich is rare in the intestines of persons living in Ceylon.

(2) ~~That~~ Faecal organisms of any kind do not appear to exist in large numbers after they have been in the earth for longer than 1-2 years; night-soil buried in the ground, even in large quantities loses most of its organisms in this period.

(3) Sporogenes enteritidis spores are capable of remaining alive in the ground for a period of something between 3 and 4 years.

(4) Bacillus coli communis has never been isolated from the trenching ground samples; hence it is probable, though not certain, that this organism does not remain alive in the ground as long as the more resistant organisms like cloacae, etc. It has been proved in one experiment to exist in the ground for a period of 97 days."

Next the change in the bacteriological flora of water and faeces during monsoon weather was made the subject of an investigation ~~which showed:-~~

"(I) That the conditions obtaining during a time of heavy monsoon weather when fresh faeces may find their way easily into water, the whole country being submerged, is in some way connected with the appearance of a set of rare micro-organisms in all the water-supplies over a wide area. This fact has not been noticed with previous and subsequent heavy rain.

(2) That these and allied bacteria may suddenly become extremely common in the faeces of man and animals. The cause of this increased prevalence is unknown.

(3) That flood conditions having passed away the bacilli very rapidly disappears from all waters. Consequently this supports the suggestion, that these organisms are especially susceptible to natural forces inimical to bacteria."

Dr Balfour then gives an account of Major Clemesha investigations.

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~~Following this~~ a systematic analysis of the water of a shallow well at the King Institute, ~~was undertaken.~~ By a shallow well Major Clemesha means one which does not pass through an impermeable stratum of soil or rock and is thus liable to contamination from surface and soil washings. ~~It was found that -~~

"(1) In common with most other water-supplies, including rivers, lakes, springs, etc., bacillus cloacae would appear to be the predominating organism in a well after prolonged drought.

(2) Even a small downpour of rain, after a prolonged spell of dry weather, has a very marked influence in increasing the number of foecal bacilli in wells situated in porous soils.

(3) That early showers following on a long period of hot weather, have caused an increase in the number of coli communis in the well of the King Institute. Further investigation is necessary before it can be stated that this is true of all wells. It is not by any means certain where these coli communis come from, but it is unlikely that they come from the surface of the ground, and probable that they remained alive in the lower layers of the soil.

(4) That after heavy downpours of rain, in consequence of which it is certain that surface contamination has penetrated deep into the layers of soil, a mixture of foecal organisms is usually found in the water obtained from wells. Coli communis appear to be present after early rains, but disappear as the rains become more plentiful.

(5) Plentiful rains improve the quality of sub-soil water after the contaminated surface water has run off the land.

(6) Shallow well are a most unsatisfactory source of water--supply, especially if they are situated in a highly porous soil, and unless the greatest possible care is exercised in

protecting a large space of ground in the immediate vicinity from all chances of pollution."

(2) Thereafter the bacteriological flora of the faeces of man and other animals was studied and the following conclusions reached:-

"(1) That, the flora of the intestinal tracts of men and animals are subject to very considerable changes due to influence which are at present unknown. These influences have been proved to operate over very wide areas.

(2) That, within certain very wide limits, these forces appear to affect man and animals equally, both as regards number and kind of micro-organisms.

(3) That, under well defined conditions, such as heavy rainfall, the water-supplies contain the same organisms as the faeces of man and animals at that particular time; but that this similarity of bacteriological flora is also noticed occasionally when rain is absent and there is now apparent cause for it. The explanation of this occurrence is at present unknown.

(4) That, having regard to the variation in the bacteria in faeces, both in quantity and kind, no constant approximate composition can be aarived at. Even in the large groups, suggested by MacConkey, variation in percentage composition in the same animal is considerable.

(5) No lactose fermenting organism has been isolated by us that has been proved to be the inhabitant of the intestinal tract of xx cattle or man only.

(6) That the numerical relation of the organisms constitut-ing MacConkey's groups in the intestines of cattle in India is entirely different from that in England; while xhx in the

intestines of man it appears to be very similar in the two countries.

(7) That, a study of the organisms present in faeces at different times of the year is necessary for the proper inter--pretation of the results obtained from water analyses."

(3) In accordance with his scheme of work Major Clemesha then undertook by Ingenious and laborious methods to determine the effect of sunlight on faecal oragnisms. From Laboratory experiments it appeared that:-

(1) that the sun has has a very powerful action in destroy--ing all faecal organisms in water, particularly when they are "naked" and not surrounded by mucus derived from the intestine;

(2) that all faecal organisms do not possess the power of resisting the action of sunlight to an equal degree;

(3) that it is possible to divide faecal organisms, with a reasonable degree of accuracy, into the following classes:-

(i) The delicate organisms or those that are very suscept--ible to the action of the sunlight;

(ii) An intermediate class containing a very large number of organisms which occupy an intermediate position between the two extremes; and

(iii) The resistant organisms or those capable of resist--ing the action of sunlight for a considerable length of time.

While from weekly analysis of water from a neighbouring lake it was found that:-

(1) That the action of the sun is powerful in destroying the faecal organisms and that the surface layers of any large volume of water are in consequence purer than the deeper ones.

(2) xThxtxthxxxxxxxxxtyxxx

(2) A practical point, following on this statement, is that the outlet from a reservoir should be arranged as near the surface as is possible.

(3) That these analyses show that, in the Red Hills lake, bacillus cloacae is by far the commonest foecal organism isolated from the bottom.

(4) That coli communis even in large numbers, in a huge volume of water, disappear with great rapidity. In the results obtained from the Red Hills lake the organisms never survived longer than six days.

(5) There is some evidence in these results, though as yet it cannot be taken as thoroughly established, that, under certain conditions, the middle layers of a lake of over 12 feet deep, contain more water organisms and more foecal bacilli than the bottom or the surface.

(6) That, when taking samples of lakes, ponds, etc., the depth from which the sample is taken should be carefully stated.

Further investigations on these lines apparently proved:-

1. That the bacillus cloacae and grunthal will persist in water exposed to the action of sun and storage for many months, and may therefore be looked upon as resistant to these forces.

2. That the rareness of such organisms as coli communis in the samples analysed demonstrates the fact that organisms, very susceptible to the action of sunlight, do exist; coli communis being the commonest example of these.

3. That there exists a large class of organisms, which in point of power of resistance to the action of sunlight and storage, come between the very resistant and the susceptible organisms. This class is made up of many species. Further

investigation may make it possible to sub-divide this large group.

4. That storage, an important factor in which in the tropics is the action of sunlight, is capable of rendering a highly contaminated water extremely pure, and of killing a very great percentage of the foecal organisms present after gross pollu-tion.

As a result of all this work it was evident that delicate organisms such as B. coli communis, and oxytocus perniciosus exist in nature along with resistant forms of which B. cloacae and Ounthal may be taken as examples while between the two groups come intermediate species such as B. lactis aerogenes. These then are the three classes into which Clemesha divides organisms which are of importance in the bacteriological examination of water and many of which, let it be remembered, are included in Houston's elastic term true B. coli.

Let us see how he applies his classification to practical purposes i.e. to establishing bacteriological standards for drinking waters in India.

"All we claim to have established," he says, "is, that, in any naturally occurring mixture of micro-organisms, a certain proportion, (how great a proportion, we have at present not much idea), of one or more species will be found to survive the others when the mixture is exposed to prolonged sunlight. But it is with this resistant residum that the sanitarian in India is most frequently called upon to deal. Hence the great importance and utility of this classification." He believes, that in a river in the Tropics the susceptible individuals among organisms will die off with great rapidity and that the bacilli, which the analyst finds, can be compared with those whose powers of resistance have been

studied in the experiments quoted. It is, therefore, necessary that the analyst, besides making himself fully acquainted with local conditions, should always ask himself " what chances has this sample had of being exposed to the action of sunlight"? As regards the waters themselves Clemesha considers the three groups as follows:-

(1) <u>Those containing class I</u>. - The samples which contain coli communis and the members of the susceptible group, present very little difficulty in arriving at a conclusion, as regards the undesirable nature of the pollution. In such samples, derived from river or lake, there can be no doubt we are dealing with recent, and therefore objectionable contamination. At the same time if foecal contamination has been recent, susceptible organisms (those in group I) <u>will be mixed with those of groups 2 and 3, roughly in the proportion in which they occur in foeces</u>. Hence we have the double reason for suspecting the water; namely, (1) the presence of susceptible organisms, which shows that the contamination must have been recent and (2) the confirmatory evidence of getting the unaltered proportions of foecal organisms. In actual practice coli communis is so seldom met with, that the latter condition of affairs is often all we have to guide us. In rivers, organisms in class I are more commonly met with, because pollution is constantly being added all along the banks, and when found certainly indicate pollution of a most serious and dangerous nature.

(2) <u>Those containing class III</u>. - It is probably not realised how extremely often, in a tropical country, organisms belonging to class III alone are found in water samples. Personally we doubt whether such a condition of affairs is ever recorded in Europe. But a glance at the analyses of the several supplies,

during the quarter of July, August and September, is absolutely convincing on this point. It is quite common to obtain samples of water which contain nothing else but bacillus grunthal or bacillus cloacae or a mixture of the two. When this occurs, one may be practically sure that we are dealing with a water from a tank or river that is getting low, and also that these organisms represent the residue of a pollution months old.

(3) <u>Those containing mixtures of two or more classes</u>. - The chief difficulty that will be met with by the analyst using this method, will be to arrive at an opinion from samples show--ing a mixture of organisms in classII, or a mixture of class II and class III. It is in dealing with such samples that it is absolutely necessary to know the particulars concerning rain--fall, etc., that have been enumerated in the early part of this chapter. Mixtures of organisms are to be expected in rainy weather and flooded rivers. In attempting to arrive at a conclusion one has to be guided by (1) the number of foecal organisms present in the sample; (2) the number of different species present in the 10 colonies isolated: (3) whether the organisms show a tendency to belong to those at the top or at the bottom of the list given in class II; and (4) whether class III are present in considerable quantity. Thus, if in a water containing a hundred foecal organisms per cc., known to have been taken from a lake or river within two or three days of a heavy downpour of rain, we were to find four or five different varieties of organisms all belonging to group 2, we should be justified in considering, that, although no organisms belonging to class I happened to be isolated, that the circumstances were highly suggestive of recent foecal contamination. In such a sample as this, one would be guided by the relative number of such organisms as lactis aerogenes. It has been shown that, in

the ordinary fresh mixtures of foeces and water, lactis aerogenes is not particularly common. Therefore a water showing a mixt--ure, rich in varieties,and yet containing few or no lactis aerogenes, would be judged to have undergone very little settlement or purification.

If, on the other hand, one found the sample containing foecal organisms in 1 cc., showing only 2 or 3 varieties, one of which belonged to class III, and if the predominating organisms happened to be lactis aerogenes, this would constit--ute fairly satisfactory evidence, that a considerable amount of purification had gone on, since the foecal matter was actually added to the water. The work given in chapters IX and X shows conclusively that, in all waters stored for some time, lactis aerogenes is a common organism, and that it is not so, in newly contaminated waters, or in waters stored for many months.

This may be taken as the rough idea as to how conclusions can be arrived at in the types of results commonly met with. On the subject of numerical standards, no hard and fast rules can be laid down, and a considerable amount of latitude must always be left to the discretion of the analyst. In the above remarks it is presumed that besides the actual organisms found in each sample, the number of lactose-fermenting organ--isms per cc., and the total colonies in 1 cc., are taken into consideration. The results obtained in the bile salt broth tubes are also very important. Thus, practically any water in India that contains no foecal bacilli in 20 cc. is probably a good water (porvided of course none of classI are obtained in a large amount).

In summing up he says of river waters:-

"Good river water should not contain more than 100 colonies (on agar at 37° C.)

Foecal organisms should not exceed 1 in 10 cc.

No organisms of class I should be present in 50 cc.

Foecal organisms present should belong to either class III or the more resistant group of class II."

"Fair or usable river water should not contain more than 300 colonies (on agar at 37° C.).

Should not contain more foecal organisms than 1 in a cc., and no organisms of class I in less than 20 cc.

The foecal organisms present should consist mainly of mixtures of class III and class II, and there should be a tendency for one organism to preponderate.

"River waters should be condemned if total colonies are more than 800 (on agar at 37° C.).

If lactose fermenters are present in number of 10 - 100 per cc.

If organisms belonging to class I exceed 1 in 5 cc.

Or if the foecal organisms isolated (class I being absent) are rich in varieties such as occur in an emulsion of foeces."

and then turning to well and spring waters remarks:-

"A good water should contain no foecal bacilli in 20 cc.

No class I in 100 cc.

Total colonies under 50 per cc.

We are for the present unable to give any other standards than this."

Now it may be thought I have dealt at too great length with Clemesha's work but it was essential I should do so as I am anxious, if possible, to obtain expert opinion on it. It has been no easy task to keep the résumé even within these limits but I trust I have touched upon all salient matters and made his arguments clear. We have now reached the point at which I was

~~aiming, namely the fact that~~ Clemesha confesses that he cannot tell how far his method is applicable to the waters of springs and wells which are not exposed to the direct action of sun--light. He admits that in these cases the question is much more complicated than when one is dealing only with surface waters, the difficulties being (1) to estimate the importance and significance of organisms that remain alive in the soil for long periods and (ii) to be quite certain when a pollution is caused by surface contamination washed down through cracks or through a porous soil.

The quantity of faecal pollution, he regards, as of paramount importance in the case of well water and the stand--ard he adopts has been given. This of course applies only to surface wells. He tells us nothing regarding deep wells and here it is that I hope to make good a deficiency and to show that in Khartoum, at least, the standard of Houston and Savage has xxxxxxxx proved itself a reliable guide in an interesting and difficult problem. I hold no brief for Major Clemesha and his work. It requires confirmation and may be refuted. At present Captain Archibald is working on similar lines with river and shallow well water in Khartoum where, however, conditions differ considerably from those in Madras. But whatever may eventually be proved there can be no doubt that the Indian work is in the right direction and that it has been carried out with much care and devotion, and in an eminently sensible and scientific manner.

Faecal notes over page.

at Khartoum

To revert to Khartoum. At the time when it became necessary to embark upon water work there, Clemesha's researches had not appeared and one had to proceed more or less on the old familiar lines.

I have mentioned the Blue Nile, a river clear when low, with very palatable water of an agreeable softness and colour and comparatively pure save where fouled from the bank at a town or village. I need not enter here into the chemical composition of its water. In flood time it is, of course, muddy, laden with silt and washings from the Abyssinian mountains. Some records of bacteriological observations are available though several were destroyed in a fire. Those existing show that in January 1907 at low Nile the number of organisms in 1 c.c. of the water taken close to the bank above the town was between 300 and 400 (agar count at 37°C, 48 hours incubation) while in a sample taken from the centre of the stream at Burrê in February, 77 organisms were found per 1 c.c. In May, with the river rising, a sample taken from mid-stream opposite the Gordon College gave 84 organisms. Other observations show that the counts vary considerably and close to the bank as many as 700 colonies per 1 c.c. have been found while B. coli of an excretal and flaginac type is commonly present in from .5 to 1 c.c. of water taken close to the bank but is not found in less than 5 c.c. of water from mid-stream. Formerly the Nile served as the chief source of supply, so far as drinking water is concerned, and so long as the water was not stagnant did not appear to be a cause of communicable disease for reasons I have stated elsewhere. (Third Report Wellcome Research Laboratories. 1908, p.64.)

Water for washing, cooking, irrigating gardens and watering animals was in the main derived from the numerous shallow wells with which the site of the town is honeycombed. Such water is always impure from a chemical standpoint and also from what we may call the English bacteriological standpoint. It is hard and often contains B. coli of a "flaginae" and excretal type in so small a quantity as 0.02 c.c. I have known B. pyocyaneus to be present in such water and have also seen its use result in dysentery and systematic pinking of such infected water lead to a cessation of the cases it had caused. In this connection I show a drawing of an excellent type of apparatus for taking water samples from wells and rivers, of a type not figured in the text books. When the question of an improved water supply came up from consideration it was evident that only two sources were available.

Comment

1. The river; the supply to be taken above the town.
2. Deep wells, if such could be obtained.

The advantages of the river supply were that the water was known to be palatable, that there was an unlimited quantity, that it would be popular with the natives and that the works required were not likely to be expensive. Its disadvantages were the necessity for the construction of settling tanks unless the water could be obtained through pipes sunk in the sand banks or by means of galleries beneath the river bed. Moreover as the river water was certainly liable to pollution from the banks and from boats and steamers it could not be relied upon as a permanent supply without filtration, and filters, even mechanical filters, are to be looked upon with suspicion especially in a country where native labour has to be employed.

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The advantages of a deep well supply were that in all probability a clear and pure water would be forthcoming and that such water would be safe from pollution and would not require filtration. Its disadvantages consisted in possible heavy expense and, in the chance that the supply might be inadequate or the quality unsuitable. It might be excessively hard or contain iron in undesirable quantities or be of such a nature as to require special treatment. In short one knew where one was with the river supply, one was totally in the dark as regards the deep wells for nothing was definitely known regarding the geological formation and there was nothing to guide one in a new country like the Sudan.

After some discussion it was decided to have a trial boring sunk which would both furnish information regarding the strata and enable samples of water to be obtained for chemical and bacteriological examination. At the same time I set on foot some enquiries into the flow of the sub-soil water and its relation to the Blue Nile. A site was selected above the town and close to the southern bank of the Blue Nile and operations were commenced with great energy. Indeed so great was the energy displayed that instead of a single trial bore the whole of an elaborate scheme of well-sinking was carried through, nor could it be checked despite protests and representations from those responsible for the public health. Proper samples for analysis could not be obtained for months and when at last the engineers declared themselves ready to submit such samples it was found that six wells had been sunk but as a seventh was afterwards drilled it will be convenient to describe the installation at a period just prior to that when the final tests were made.

The site has already been stated. It suffices to say that no great distance away lies the village of Burri which is served by shallow wells liable to pollution.

The water works, as will be seen, consisted of seven wells and a pumping plant. Since then a large, covered reservoir has also been provided. The wells lie along two lines, one at right angles to the river the other nearly parallel to it. Well No. 5 in the first line is 30 yards from the river bank. The wells are about 60 metres apart, are drilled and at that time were of the following depths i.e. 1, 2, 3, 4, 5 & 6 of 75 metres and No. 7 of 176 metres, these depths being to all intents and purposes from ground level.

Wells No. 1 to 6 were alike in being fitted down to a depth of 22.40 metres with a casing pipe of 9½ in. diameter. Below this point, the bore-hole, 8 inch^{es} in diameter, extended unguarded to the full depth of 75 metres.

Well No. 7 was cased with a 6 5/8 in. pipe down to 54 metres while the unguarded bore-hole of 6½ ~~metres~~ inches diameter extended below this to the full depth of 176 metres.

As has been said there was originally no reliable information regarding the geological structure of the site. Some details had been gathered and sections prepared when No. 1 well was drilled but as there was no core-boring and the material was washed out in a finely comminuted state the information furnished could not be regarded as very reliable. Indeed subsequent observations proved it to be incorrect in certain particulars.

So far as could be told the upper strata down to a depth of 23 metres consisted of layers of sand, clay, mud and gravel. Immediately below came rock seamed, so it was

said, by beds of clay. This rock, there was reason to believe, was a porous limestone, a "Travertine" as it is called but at the time the first tests were made no information was forthcoming regarding its nature. It was understood, however, that the casing pipes ended in it and not, be it noted, in any impermeable bed of clay. At first indeed this rock was said to be sandstone with a layer of clay marl overlying it and it was assumed that a more or less similar formation existed in the case of each of the first six wells. Anyhow below this doubtful stratum was the water-bearing sandstone which was shown as coarse and containing gravel. The earlier sections showed the rock to be seamed at different levels with clay marl.

There had apparently been a fault between Well No. 7 and the other wells for, in its case, below 23 metres the strata differed in position and thickness. One need not, however, go into details nor is it necessary here to enter into the question of the quantity of water yielded at first by these wells as our time is so limited but a word or two may be devoted to the air-lift pump, the method of water-lifting eventually employed. The air-lift which is not strictly a pumping machine was, I believe, originally an American invention which under certain conditions renders excellent service. Its principle of action is the lessened specific gravity of water in which air is contained in considerable quantity, or through which it is rising in bubbles. There is an air-pump or compressor and from it an air-pipe passes to the bottom of a water tube, into which tube air is discharged. The air rising causes the surface of the water to rise, since the atmospheric or other pressure at the base remains the same. The output from deep wells can thus be increased although the total mechanical efficiency of such a plant is said to be less than that of a good pumping installation. One mentions it here as, naturally, its influence

on the bacterial content of the water required consideration.

As may be surmised the very scanty information obtainable regarding these wells did not place a bacteriologist in a very favourable position for coming to definite conclusions regarding the results he might obtain on analysis. The responsibility was great, the problems novel and there was determined opposition to sanitary interference. One could not tell if heavy rains falling on a cracked and gaping surface soil might not wash impurities to such a depth that they could enter the well water by the unguarded bore holes, one could not tell if, under the influence of powerful pumping with great depression of the water table, river water or water from pools in the dried river bed might not similarly gain entrance. From the river bed to the top of the unprotected bore hole of well No. 5 there was only 13 feet of sand in a vertical direction and 90 feet of presumably porous material in a horizontal direction to act as a "natural" filtering medium. Again there were the shallow native wells in the neighbourhood which certainly merited some consideration. One felt doubtful about the whole scheme. There might be little evidence of pollution and yet the water might, under certain conditions, be a source of danger. One had been confidently assured that the water was the finest in the world and had replied with the expressive Arabic word "Yumkin" which being interpreted signifieth perhaps. If, at first, the results were good it might be difficult to prevent the water being rushed upon the town although it was evident that the tests would have to be prolonged and careful. The influence, if any, of the falling and rising Nile upon the wells certainly called for study, as did the effect of severe and exhaustive pumping.

It was, I confess, an anxious time and one was scarcely prepared for the results which were obtained. Before mentioning these one must outline the methods of examination employed.

Water was collected from the wells in special sterilised glass-stoppered bottles. The discharge was as a rule from a special wrought-iron pipe of small diameter leading from the large delivery-pipe directly connected with the well. The small pipe was passed through the furnace every morning of the days on which samples were taken. Its faucet was also flamed prior to collection of the samples. As a rule three samples were taken from each well and at least two of them were examined.

The bottles after their necks and stoppers had been flamed were filled, packed in ice and transported by motor or launch, to the laboratories where the examination was commenced as soon as possible, nearly always within half-an-hour of the time of taking the samples.

Quantitative Examination.

The samples were plated on the usual nutrient agar (1% to phenol-phthalein) and the quantities usually employed were .2 c.c., .3 c.c., .5 c.c. and 1 cc. Occasionally 2 c.c. plates were also poured. The plates were made in duplicate and often in triplicate. As a control one set of plates was poured by me from one water sample and another set by my assistant from another sample. Now and then the third sample was also utilised for making plates. In addition control plates were made with agar alone and with agar to which sterile water was added. The plates were incubated for 48 hours at a uniform temperature of 37°C. and then counted. Occasionally there was trouble with the incubator, and when a temperature of 37°C. could not be maintained the plates

were allowed to remain for a longer time in the incubator.

As a rule the technique proved satisfactory but on one occasion it was found that the stock agar had an inhibitive action on bacterial growth, owing probably to faulty preparation or standardisation and the counts made on this agar were discarded.

Further, on several occasions the presence of haboubs, or high winds with dust, resulted in air colonies getting access to the plates and ruining the work. It was found impossible to prevent this contamination.

Qualitative Examination.

The presance or absence of B. coli was determined at every examination. For this purpose the following standard media were employed:-

Glucose bile-salt neutral red broth with Durham's tube for gas formation, MacConkey's glucose, peptone, bile-salt medium with Durham's tube ~~xhix~~ and latterly the new lactose-bile medium of Jackson with Durham's tube which constitutes the most delicate presumptive test for the presance of B. coli.

The quantities of water examined were as a rule 5 c.c., 10 c.c. 25 c.c., 50 c.c., and 100 c.c. When any suspicious appearances were observed confirmatory tests were employed; the organisms present being plated out on the Endo or Drigalski-Conradi media or on lactose bile salt neutral-red agar and then examined for indol and subcultured into the usual milk, sugar and litmus media as well as examined microscopically.

I may say that, on the whole, the Endo-fuchsin agar yielded the best results. When considered necessary the B. enteritidis sporogenes test was applied and a search made for streptococci. When essential the pathogenic effect of a suspicious microbe was tested on animals - gerbils, jerboas, fowls and guinea-pigs.

In short the method favoured by Savage was closely followed.

Table 104-4

The condensation water in the air-pipe line serving the air-lifts was also examined bacteriologically.

Routine observations, as regards the temperature of the water for different wells tested and as regards the level of the ground water in the wells were made while the tests were being conducted. The data as regards levels were furnished by Mr. Williams - the Engineer of the Water Works.

I have said that the results of the first examina--tions were surprising. They were such as to lead to an immediate condemnation of the water both on bacteric--logical and chemical grounds. Samples were found to be faintly opalescent, becoming more so on standing and depositing a very small amount of sediment. The taste was faintly chalybeate. The colony count varied between 1000 and 2000 per 1 cc. B. coli, as a rule of a flaginae and excretal type, was present in 1 cc. and larger quantities, the free ammonia figure .5 to .6 parts per million was excessively high and iron and manganese were present in objectionable quantities.* 1.33 parts of iron per million and .17 of manganese clearly indicated that there would be trouble with crenothrix growth in pipes to which such water was admitted

* Much free ammonia unaccompanied by albuminoid, however as is well known is frequently found in waters containing iron + is derived from the reduction of nitrates.

and this proved to be the case C. polyspora speedily making its appearance.

According to Hazen ~~.005~~ .5 parts of iron per ~~10~~ 1,000,000 is about the permissible limit while, in the Rod-El-Farag well water in Cairo, trouble had ensued with .35 parts per 1,000,000 iron and .77 parts manganese.

Dr. Beam, our Chemist, and I reported that as a result of the examination and a study of the topography of the well district we were led to believe

1. That the iron and manganese are derived from strata and will persist in the water.
2. That we are dealing with a mixture of deep well water whose source is the Blue Nile and of superficial or ground water highly charged with micro-organisms which is gaining access to the wells either through defective joints in the pipes or, what is more likely, through cracks and fissures in the strata or through porous beds.

Naturally this adverse report did not please those who had forced on the water scheme without due precaution, deliberation and the necessary enquiries and tests. Only one well had been tested, and the conditions of sampling were said to be unsatisfactory.

Proper pumping trials with ordinary pumping apparatus were now commenced, the mixed water from 5 wells was examined with every precaution and the results, as will be seen from the conclusions to our report, were much the same. We stated that

"1. From a physical standpoint it is apparent that the water is quite unsuitable for a town supply. It is not clear when freshly pumped and becomes distinctly turbid on standing. Moreover, as predicted, the presence of iron and manganese in the water has resulted in the rapid growth of the Crenothrix

fungus and the development of an odour distinctly disagreeable. The taste is also somewhat unpleasant.

Such a water would infallibly cause serious trouble by leading to a blocking up of the service pipes especially those of small calibre. The greatest trouble would result when the iron-stained filaments of the fungus break off and become diffused throughout the water. This renders the water quite unfit for laundry use owing to the unsightly and destructive deposit of iron mould on linen and other fabrics. Moreover it is not to be expected that people would pay water rates for water of this character.

In this connection we may cite the so-called "water calamity" of Berlin, and the similar calamities recorded from Rotterdam, Charlottenburg and many other places, including, to come nearer home, the recent revelations in Cairo. It is true that aeration combined with filtration will, in most cases, remedy this evil but the filtration must be efficient as there is already a case on record of an incompletely filtered water, from which the iron was not wholly removed, resulting in the growth of crenothrix.

2. From a chemical standpoint the water still remains much harder than that of the Blue Nile and a soft water is always preferable for a town supply from an economical standpoint owing to the action of hard water on boilers and soap. The presence of iron is of course objectionable chiefly for the reasons already detailed.

In our former report we mentioned that the quantity of iron is far in excess of what is permissible in a town supply. The slight reduction in total solids and carbonates indicates that the forced pumping has resulted in the drawing in of a larger amount of the water to which the contamination of the deep well water is due.

3. From a bacteriological standpoint the water is in much the same

condition as it was at the time of the former examinations, so far as the presence of B. coli, the standard bacterial indicator of impurity goes.

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The number of organisms present in 1 c.c. is greater than that present in the last sample taken from well No. 3 on January 19th. The results of the examination of this sample have not been submitted to the Board as the analysis was still proceeding at the time of the last meeting. 218 micro-organisms were then found per 1 c.c. and true "Flaginac" B. coli was present in 1 c.c. of the water.

Hence there has been very little change and what change there is has occurred is in the wrong direction. In a pure, deep well water the bacterial count at blood heat should not exceed 5 to 10 micro-organisms per 1 c.c.

As stated B. coli is taken as the standard of pollution and the presence of B. enteritidis sporogenes merely serves to add confirmation to the evidence of pollution.

This organism should not be present in less than 1000 c.c. of a deep well water. Here it has been found in 500 c.c. In shallow well waters it should not occur in 100 c.c. hence it was not necessary to test for it in lesser quantities than half-a-litre.

As regards streptococci they have much the same significance as B. coli; in other words, when present in large quantity they indicate faecal contamination, but their position as precise indicators is not yet assured. It will be noted that they were present in 0.1 c.c. of the water. As compared with Blue Mile water it is evident that bacteriologically the samples of well water examined were just about four times as bad." I was now somewhat in the position of

Pooh-Bah in the Mikado. As Government Bacteriologist I condemned the water and as M.O.H. I refused to accept it - Personally I was inclined to make a stand for river water and filtration or at least to insist on filtration of the well water.

With the scanty information available it was impossible to come to any definite conclusion regarding the source of pollution. Noting, however, a certain ratio between the numbers of micro-organisms present in the well water and those in the Blue Nile water I concluded that the river water was finding its way into the wells and, considering Dawson's views, which I have already cited, I hazarded the opinion that one or other of the soil layers was acting as a culture medium and that, under conditions of increased temperature and favourable nidus, the organisms in the river water were multiplying in situ. The type of organism found on the plates favoured this hypothesis. That ~~it~~ this view was in all probability correct I may at once declare ~~xxxx~~ for further investigations proved the existence of a bed of bluish shale.

As there was also a doubt regarding the quantity of water available it was decided to obtain the advice of Mr. Abel of Cairo, a well known hydraulic engineer. He was accompanied by Dr. Todd of the Egyptian Public Health Department who acted as Mr. Abel's personal adviser on the bacteriological aspect of the question. Their visit unfortunately took place at a time when the laboratories were partly gutted by a disastrous fire and Dr. Todd's work had to be limited but they had the advantage of being given every facility and full information as it was hoped their investigations would put a different complexion on the case, especially as the air-lift plant had been installed and was in use. So far as the chemical and bacteriological analysis went this was not the case. The work done in the laboratories was amply confirmed and their report only differed from ours in declaring

that any true surface contamination was impossible. In this, I believe, they were perfectly correct. Further acquaintance with soil conditions has assured me on this point though my chief fear was that water from polluted pools in the river bed at low Nile might find its way into the unprotected bore holes.

One cannot enter into their interesting enquiries and elaborate tests in detail but it is useful to tabulate their results. These are as follows:-

"The results of the bacteriological tests show that at the time of the trials the water was far from satisfactory, from a hygienic point of view.

The two wells which were subjected to a more or less detailed examination gave evidence of a somewhat intense and irregular pollution.

For reasons given above, it was not possible to definitely locate the source of the pollution; but from the results of certain tests, we were forced to the conclusion that the water which enters the wells from some of the upper layers tapped must be highly contaminated.

How this contamination arises is a question which we are unable to settle, from want of sufficient data; but we are convinced that any true surface contamination in the immediate vicinity of the wells is out of the question.

Concerning the chemical constitution of the water, we are not inclined to lay much stress on the results of the analysis available, as these are hardly sufficiently numerous, nor do they cover a long enough period.

They, however, appear to show that, apart from the presence of iron and manganese, no serious objection can be raised from a purely chemical point of view.

Should iron and manganese ultimately prove to be present in quantities large enough to create difficulties, these would in no way be insurmountable, and the question would be merely one of expense".

Their recommendations are of special interest. They advised

(1) The cutting off of the upper strata in the wells down to a certain depth.

(2) The improval of the compressed-air plant, so as to guarantee continuous working and avoid contamination of the wells from the air-lift.

(3) A continuous pumping trial, extending over a period of at least a fortnight, with regular measurements of the output and depression.

(4) A series of bacteriological and chemical tests made at suitable intervals throughout the above trials.

If these tests give satisfactory results, so that regular pumping from the wells is begun, the following might be proceeded to:-

(5) A periodical bacteriological control.

(6) A regular chemical examination of the well-water with a parallel examination of the Nile.

(7) The deepening of wells Nos. V and VI to about 200 metres.

(8) The boring of a new well at a point suggested and taking advantage of this boring in order to obtain accurate information both as regards the various strata and as regards the water carried by these".

I may say that they were led to believe that the sandstone layers belong to the Nubian sandstone - which is said to reach from Uganda to Assouan, and that they are therefore extensive water-bearing strata fed mainly by seepage water from the Nile. Further chemical examinations have led Dr. Beam and myself to regard this opinion as correct. The deep water is probably derived from the White and not the Blue Nile and it is likely that it has travelled a long

way underground.

Messrs. Abel and Todd who were put in possession of information regarding the "Travertine" bed of which we were ignorant, made the interesting discovery that strata of similar material crop out on the banks of the river east and south-east of the town - It is therefore possible that this layer places the wells in direct communication with the river, the porous bed extending some two miles or so from the site of the wells to the river outcrops. This, and the presence of the bed of blueish shale, which was found to exist immediately beneath the 'Travertine' and to be about 6 metres in depth would, in my opinion, adequately explain the results at first obtained i.e. the evidence of a mixture of pure deep well water and of water which, from the English bacteriological standpoint at least, was grossly polluted. Granting these conditions exist could such water travelling into the wells from the river cause disease under any known conditions? I believe it might. There are native villages above the outcrops and there is a certain amount of boat and steamer traffic. Given enteric fever, dysentery or cholera I do not see why specifically contaminated river water might not reach the wells. It is true that in the bed of blueish shale saprophytic organisms might successfully combat the pathogenic but we know that B. coli communis, at least in some of its forms, is not a very resistant organism and yet it was present in very small quantities of the water. In the case of Nile water also one has to consider such diseases as bilharziosis and dracontiasis. At any rate, as will be shown, B. coli gave clear indication of the contamination or, if you will, admixture of the true deep water with superficial water. Even supposing the B. coli found were to fall wholly into

Clemesha's third group. I think it would have been the height of absurdity to pass wells which at very heavy expense presented the town with water - one half of which at least, let us say, was derived from the very river which flowed past 30 yards away from one of them. This, too, apart from the important question of the contained iron and manganese.

Happily the laboratory representations prevented this water being supplied to the town and in my Public Health Report for 1908 I wrote "one can only hope that some means will be devised whereby the water may be obtained free from chemical and organismal impurities.

If this is found impossible then filtration must be employed or, if necessary, the Blue Nile water utilised and passed through bacteriological filters. The threatened invasion of cholera last year renders this water supply question most urgent and important". Fortunately the question was solved and satisfactorily solved along the lines suggested by Messrs. Abel and Todd. The upper strata were cut off with cement and as a result the colony count fell well nigh to zero, B. coli vanished and the quantity of iron and manganese greatly diminished. As this engineering device may not be generally known to medical men it may be well to indicate how it was carried out. I am indebted for particulars to Mr. J.E. Williams, the engineer in charge of the works who, at all times, was most courteous and obliging. The process is simple enough. As noted (with the exception of No 7 which was of smaller bore) the wells down to a certain depth were lined with a casing tube of 9¾ in. diameter. A second tube of 6 5/8 in. diameter was passed down each well to the depth where it was desired to cut off the upper strata i.e. to

one or other of the deep beds of impermeable clay. This inner tube therefore descended in the open bore hole below the level of the end of the outer tube. In order to facilitate operations the well was first of all filled with sand which provided the necessary cushion. Liquid cement was then poured between the two tubes down to the depth desired and in cases where it did not settle properly it was blown down and packed by means of compressed air. It set for the most part under water and formed a hollow column or ring, the lower portion of which, confined between the inner tube and the wall of the bore-hole, in some degree soaked into the porous sandstone with which, as stated, it was in contact and rendered the latter absolutely impermeable. Although transverse fissures may occur in such a column, it is scarcely conceivable than any lengthy vertical crack can be produced in it. Hence the cutting off is regarded as efficient. Finally the cushion of sand was blown out of the well which, if the clay bed is both extensive and impermeable, is thus wholly protected from any local influx of water from upper and possibly contaminated strata.

I will now conclude what, despite many omissions, has, I fear, proved a lengthy paper by quoting from portions of my final report to the Sudan Government on the examination of the well waters before and during special and continuous trial pumpings of twenty-one days duration in the months of March and April 1909.

" The wells tested were Nos. 1,5,6 & 7 and the first three at the time of the special pumping trials had undergone very extensive alterations since the examinations conducted in 1907 - 08. For one thing the air-lift pumps and air compressors had been introduced, while well No. 5 had been deepened and, in all, the upper strata had been cut off

by a special process. No. 7 was a new well to us. It had been bored since the last examinations. As a result of these changes we soon found that we were dealing with quite a different water from that which had led to a condemnation of the original wells. Nos. 2, 3 and 4 have been discarded and filled up with sand.

As, from previous experience, confirmed by the results obtained by Messrs. Abel and Todd, we were aware that the old wells had been contaminated by the influx of polluted water from upper strata, it was of the highest importance to ascertain

1. If the beds of clay, separating the polluted water in these strata from the deeper water, were or were not impermeable.

2. If the methods of cutting off the upper strata by means of iron tubes and cement rings were or were not adequate.

For this purpose one maintained that continuous pumping trials extending over a period of 21 consecutive days were essential so that a maximum depression might be obtained in the wells, which in this way would be subjected to a trial more severe than any they were likely to undergo when being pumped on for the town supply.

The condition of the wells which were tested in groups of two was as follows:-

No. 1 Depth 100 metres. Upper strata cut off to a depth of 65¾ metres.

No. 5 Depth 112 metres. Upper strata cut off to a depth of 69⅜ metres. Combined yield 18,000 gallons per hour.

Brandy 33×8

No. 6 Depth 75 metres. Upper strata cut off to a depth of 53.82 metres.

No. 7 Depth 176 metres. Upper strata cut off to a depth of 72⅔ metres. Combined yield 21,000 gallons per hour."

It would take far too long to give the results in detail. Their general consideration as stated in the report will suffice.

" So far as Wells No. 1 and No. 7 go, these do not call for any longthy discussion. A glance at the attached table will show that in both cases, and especially in No. 7, the colony count has almost throughout been uniformly low and compatible with that obtainable from true deep wells containing water free from bacterial impurity. On three occasions, March 18th., 21st. and 22nd. on which the count has exceeded 50 colonies per 1 c.c., the results can probably be adequately explained by the hypothesis that some of the foul condensation water from the air-pipe line was forced into the well. Personally I am almost certain that this did occur on several occasions and accounted for the greater number of colonies.* Excluding these abnormal counts the average colony count for both wells works out at 5.8 per 1 c.c.

Further, and this is of greater, indeed of paramount importance, B. coli has not once been found in either of these waters in the quantities (100 c.c. and under) examined, nor has any other objectionable organism been found.

Hence <u>at the present time</u> the water of these two wells may be passed as a good drinking water, the only possible objection to it being that it is not well aerated and that it is warm, both faults which can be readily remedied.

* It seemed curious that the air blown into the water did not in any way seem to affect the colony counts. At first it was carefully filtered, but afterwards this precaution was abandoned for observations made by Mr Williams showed that the 500 cubic feet of free air passing per minute through the cylinder was probably subjected to a temperature of 220° F. This, though but momentary, would, I think, serve effectually to sterilize it.

The slight odour of sulphuretted hydrogen in No. 7 well water does not call for any special criticism.

Taking next Well No. 6 we find that its water closely resembles that found in No. 1 with a single exception. For the greater part of the trials there was present in it an organism closely allied to, if not identical with, B. proteus fluorescens.* It is necessary to consider the precise significance of an organism of this type in a deep well water. Its morphology and cultural characteristics have been discussed on p.9 and, as the same organism was present in No. 5 the remarks here made will apply, so far as this matter is concerned, equally to that well water. As has been pointed out B. proteus fluorescens is an organism capable of developing and multiplying in bile-salt media and it is well known that the majority of organisms which do so are to be regarded with suspicion. Moreover this is the very bacillus which Jäger and others have claimed, apparently with some reason, as the cause of infective jaundice (Weil's Disease) a condition not uncommon in Egypt and elsewhere in tropical or sub-tropical countries. It was, however, in Germany and the United States that the illness was found associated with the presence of this organism. It has, however, to be noted that in these cases the organism was isolated from <u>impure</u> water which had been drunk by the persons infected or in which they had bathed.

Now there is no evidence that the water in No. 6 well

* This bacillus was present as a rule in 5 c.c. and larger quantities but was not infrequently found in 1 c.c. of the water.

is in any way contaminated by excretal products. The reverse is the case, for the standard indicater of impurity B. coli communis has never been found in it. While there are limits to the value to be attached to the chemical and bacteriological analyses of water, still a vast amount of most reliable evidence goes to show that the absence of B. coli from the water is the best known criterion of safety as regards water-borne diseases. This is the view I was inclined to adopt in the present instance but as the matter was one of great interest and importance and as the organism has been proved pathogenic to man and was found to be highly virulent to certain rodents I thought it well to lay the case before Dr. A.C. Houston, Director of Water Examinations to the Metropolitan Water Board, London, and probably the leading water bacteriologist of the present day. He has confirmed this view and states definitely that, although he has encountered chromogenic glucose (and sometimes lactose) fermenting microbes in well water, he does not condemn the supply, other things being satisfactory and B. coli being absent. Acting on this principle, which appears to me eminently sound, I have passed No. 6 well, more especially as the B. proteus fluorescens had disappeared from the water towards the end of the trials.

Summary 30 Passing now to the consideration of Well No. 5 we find the problem is somewhat different. The water is clearly not derived from precisely the same source as in the case of the other wells. This is evident from its uniformly lower temperature which no consideration of the pipe arrangements &c. will explain. Moreover the bacteriological content differs to some extent. The colony count was more variable

and not quite so satisfactory. Chemically also there are slight, but possibly significant differences (Vide Dr. Beam's report attached). Thus although B. coli has never been found in this water, the facts stated, together with the constant presence of B. proteus fluorescens lead one to regard this source of supply with some suspicion. I think it would be advisable not to pass No. 5 until we have learned more about it. The water would appear to come from nearer the surface. It does not seem to have any immediate connection with the river and as the yield is small, would appear to be derived from some bed of limited extent which perhaps trends towards the surface. This is not an easy question to settle and of equal difficulty is the determination of the source of B. proteus fluorescens both in Well No. 5 and Well No. 6. It appears that adjacent to the water works, there is a bed of decomposing vegetable material close to the southern river bank and about a metre under the river bed. This was encountered when the pipe trenches for the condensation water from the Blue Nile were dug but no information was furnished me on this point and I only learned it recently from one of the officials. Considering, however, that the cutting-off by cement is satisfactory and that the deep clay beds appear to be quite impermeable it would not seem that any contami-nation could be derived from this source and in any case such contamination might ~~xxx~~ not be of a dangerous nature. The suggestion has been made that the cement used in the cutting-off process might have become contaminated from the feet of the native workpeople, and that the water-bearing beds had thus been rendered temporarily impure. If this were so, however, one would have expected a much higher colony count and a much greater variety of microbes while some type of B. coli would almost certainly have been present, unless indeed other organisms, apart

from B. proteus fluorescens, had died out in the pure deep water. This is not very likely as B. coli is known to be a fairly resistent bacillus. The organism does not appear to have come from the air-pipe line.

Table 32

It would serve no purpose to discuss this matter at greater length but there is one point on which special stress must be laid. The results obtained can only be considered applicable to the wells under existing conditions. True the tests have been fairly exhaustive and severe and personally I think it likely that the water in Wells. 1, 6 & 7 will continue to be a safe source of supply but one cannot be absolutely certain, for it is possible, though not probable, that changes in the river may induce changes in the deep water. Hence the necessity for routine bacteriological tests which indeed are now-a-days carried out in connection with all well-conducted town supplies. These will be combined with occasional chemical examinations and this leads one to draw attention to the attached report from Dr. Beam. On this occasion chemical tests are of subsidiary importance but they possess considerable interest and, so far as they go, they confirm the bacterioscopic analyses.

There is nothing in them to lead to a condemnation of any of the well waters. The quantities of iron and manganese present are too low to be likely to cause any trouble with Crenothrix growth provided the large storage tank now in course of construction is in use. At the same time they are undesirable ingredients though it will be noted that with the exception of No. 5 well they tended to diminish as pumping proceeded. It will be also noted that the plumbo-solvent power of the water was tested although very little lead piping will be used in Khartoum.

The following conclusions therefore, appear justifiable:-

1. From a chemical standpoint the four wells tested Nos 1,5,6 and 7 have yielded a satisfactory water.
2. Bacteriologically, Wells Nos. 1, 6 & 7 have yielded a satisfactory supply.
3. Bacteriologically Well No 5 has not proved so satisfactory as the others and is not passed at present. As a matter of fact I understand that as there is a low yield from this well the Director of Works has no particular objection to its being closed and this may possibly be done.
4. The examinations made refer only to the water under existing conditions though it is believed that the tests have been sufficiently prolonged and severe to make it probable that the supply will remain satisfactory as the source itself seems to be above suspicion.
5. It will be necessary to conduct routine bacteriological examinations and occasional chemical tests while the conditions of storage will require careful consideration.
6. The question of quantity is one for engineers to settle but, from the data furnished, there would appear to be an ample supply at least for the present.

I may say these conclusions have so far been amply justified. For a year and more routine tests have been made, the Nile has risen, fallen and risen again, the well water has been in general use, its quality has remained good, and apart from a little trouble in the pipes owing to Crenothrix the supply has been quite satisfactory. A large covered reservoir has now been constructed and this will tend to improve matters so far as iron is concerned.

in the Tropics but this paper, despite every effort at compression, has assumed alarming dimensions and so I would again lay stress on the demonstration which has been afforded us of the great value of B. coli, even Houston's old, ~~as~~ only partially qualified, elastic B. coli, as a standard of pollution under certain conditions in the Tropics. It would have been most interesting to apply Clemesha's tests to the B. coli which was found in the deep well water but his paper had not been published and in any case time would not have admitted of such a research. I wonder, however, into which of Clemesha's groups the B. coli recently found by Thresh in deep well waters in England would go! In Khartoum, Clemesha's distinctions were, for several reasons, unnecessary but it is possible they may be of value even for deep well waters, and not only in the Tropics, although, considering the basis on which they rest, this is, at least, doubtful. In any case I trust some interest has been excited in the subject while, apart from the vexed question of B. coli, I think some of the problems mentioned, some of the results recorded, and some of the conclusions reached may be of service to those who have the responsibility of examining and reporting upon town water supplies in tropical countries.

Brewer

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As the syllabus shows I had intended to say something as regards distribution, storage and methods of water-sterilisation

48a Brander 27

With accompanying photomicrograph.

From Dr. Andrew Balfour
Khartoum
27-2-10.

3486

O.C
Sparus
103

A Microfilaria in the blood of a horse at Khartoum.

Filaria sanguinis equi africani (Martini)

by

ANDREW BALFOUR, M.D., B.Sc., F.R.C.P. Edin., D.P.H. Camb.

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On February 5th. Captain F.U. Carr, Principal Veterinary Officer sent me a pony which was suspected to be suffering from trypanosomiasis. Such was found to be the case, a trypanosome being present in fair numbers in the peripheral blood which, so far as can be told from morphology alone, is probably T. dimorphon. The pony was then treated with arsenic and was sent for re-examination on Feb. 12th. when trypanosomes were found to be very scarce but filarial embryos were discovered in the blood. It is this micro-filaria which forms the subject of the present short paper.

History:- The owner of the horse Mr. L. Landon of the Sudan Irrigation Service very kindly furnished me with the following particulars regarding his pony. It is an Abyssinian and was purchased in 1906 when three years old. Towards the end of 1908 it was taken to Uganda, reaching Gondokoro in the Nile Province about December 10th. and proceeding thereafter as far as Kobe at the north end of Lake Albert. At that time the pony was perfectly well but was sent back to Nimule on the Nile and was there found to be slightly sick in March, but not to such an extent as to prevent its being ridden back to Gondokoro, brought down the Nile and sent to Dongola. There it developed pneumonia in June or July and since that time has been more or less ill.

Present condition. The pony has no marked signs of illness

and is well nourished. The blood coagulates rather more quickly than usual and is a little greasy but not specially so. There are no skin lesions of any kind. As stated, filarial embryos were present in it. The blood in which they were found was derived from one of the branch veins of the ear and about one micro-filaria was present in each blood film - fresh or stained examined, i.e. in about every small drop of blood.

The Parasite.

In the fresh blood the parasite is found to be exceedingly active, rapidly traversing and leaving the field of the microscope, twisting, coiling and lashing about incessantly so much so that in a thickish film it causes a certain amount of destruction of the red cells. It possesses a blunt cephalic and very sharp caudal end. No evidence of a sheath can be seen. In cover glass preparations it remains alive for at least 6 hours though its movements become sluggish and it rarely coils itself up as it does at first.

In ordinary preparations stained by the Leishman method and carefully measured by means of the ocular micrometer it is found to vary in length from 115 μ to 180 μ. In citrated blood and in dehaemoglobinised films this measurement becomes considerably less i.e. 80 μ to 95 μ or thereby. Doubtless the medium or the process of staining causes some alteration. The average width is 4 μ but this also varies a little in stained films.

The cylindrical body, especially in smears fixed in alcohol, is seen to be coarsely granular and it presents as a rule four or five clear areas or spots. There is one at the head end, possibly of the nature of a cephalic prepuce as it varies in size and is not always visible. It is rounded, exhibits no

hook or spicule, stains a faint blue, measures about 1.5 μ in length and is continued into the central column of cells as a narrow but very distinct slit about 3 μ in length by .5 μ broad. This does not show in all specimens, and would seem to depend on what aspect of the parasite happens to be presented to view. In a parasite measuring 115 μ in length the fllowing spots were made out, apart from the clear area at the cephalic end and the accompanying slit:-

1. A well marked somewhat oval lateral bay with centre 34.5 μ from the anterior extremity and measuring 4.5 μ X 1.5 μ.
2. A small indefinite central spot with centre 73.5 μ from the anterior extremity of the worm.
3. A large clear area with centre 90 μ from the anterior extremity and measuring 6 μ X 3 μ but occupied in part by cell nuclei. This is the most characteristic and constant of the spots and the cell nuclei always seem to encroach upon it.
4. A small but distinct triangular caudal end, including the tip of the tail, which does not take on the stain and which is not seen in all specimens.

In other examples one has observed a small lateral bay between No. 3 and the tail tip and also a larger lateral bay, two-thirds of the way between the anterior extremity and No. 1 (Fig. 1).

The spots are not so evident in parasites observed in dehaemoglobinized films or in smears of citrated blood.

Examination with high powers reaveals nothing of further note.

The only other filaria so far met with in horses in the Anglo-Egyptian Sudan is *F. irritans*, the reputed cause of Bursati or summer sores. Its larvae, as has been shown by Lewis and by Lingard, may occur in the blood although there is no skin lesion. The parasite just described, however, is not

the embryo of F. irritans, the latter being much larger. It is indeed visible to the naked eye and can be dissected out of Bursati tissue under a dissecting microscope. A filaria has been found in donkey's blood in the Bahr-El-Ghazal but has not been fully studied and in looking through the available literature one found that references to micro-filariae in the blood of equines were rare. Mention is made of a Filaria sanguinis equi described, it is said, in Egypt by Sonino but I could find no description of this parasite. The same applies to the embryos of F. equi or equina which are said to occur in the blood. Curiously enough Hutyra and Marek do not even mention this filaria. Happily, however, one came across an interesting and illustrated paper by Martini[1] which left little doubt but that the filaria in question was no other than Filaria sanguinis equi africani which he discovered in a Barbary mare from Togo in the Berlin Zoological Gardens in 1902. His photomicrographs of this filaria show that it is practically identical with that under discussion. Indeed his Fig. 20 might almost have been taken from one of my slides. In his paper he points out that the presence of the filaria in his case was unassociated with illness or loss of weight, that the parasite measured between 100 and 150 μ in length by 4 μ in breadth and that it possessed thick and thin ends. He describes its great activity and its mechanical destruction of red blood corpuscles, its longevity in vitro and its pale unstained areas which closely correspond to those observed in the parasites of the pony from Uganda. He regards these spots as remnants of an embryonal membrane. He ~~points out~~ how his filaria differs from a

Lingard's papers on blood filaria in equines in India are not available/

indicates/

[1] "Ueber eine Filaria sanguinis equi" By E Martini, Zeitschr. f. Hyg. und Infectionskrank, Vol XLII 1903 pp 351, 352.

pathogenic form previously found by Lange in the skin of the ear muscle of a horse and he points out its resemblance to F. perstans so far as size, activity and general appearance are concerned.

He did not find the parasite in blood from the smaller peripheral vessels but only in defribinated blood taken from the jugular vein. It occurred in comparatively small numbers, about one to every five drops of blood taken.

This, however, and the fact that he described the body as finely granular, are the only points of difference I can perceive between his parasite and that under discussion and, considering that the animal in which he discovered it was living under very dissimilar conditions to those obtaining when it became infected, one cannot lay any stress on such slight variations.

I think that this parasite is Filaria or better Microfilaria equi sanguinis africani and, so far as I can find out, it is the second occasion on which it has been observed.

The infection was probably acquired in Uganda and is not of a pathogenic nature, the chief interest attaching to it being possibly the somewhat close resemblance of this embryo blood worm to the larval F. perstans which is such a common human parasite in Uganda. That it is not F. perstans is evident enough. It possesses no retractile spicule, it is smaller, its caudal end is not truncated and its spots do not correspond to those of the perstans embryo. Still it would appear to be a parasite of the perstans class and doubtless the adult forms exist in situations similar to those in which the male and female perstans were found by Daniels.

One may conclude by noting that on both occasions when the blood was found to contain the parasites the examination was made about mid-day and that gerbils, a jerboa and a young

rabbit inoculated with blood from the pony in connection with work on the trypanosome have failed to exhibit the filaria.

Reference:-

1 Martini, E. (1903) "Ueber eine Filaria sanguinis equi", Zeitschr. f. Hyg. und Infectionskrank. Vol. XLII. p.p. 351, 352.

One photomicrograph sent, x 950 diam. (Fig 1) taken by Mr. George Buchanan, from a slide stained by the Leishman method.

W. R. L.
Khartoum
27.2.10

23.3.11.

Editor,

Journal of Tropical Medicine
& Hygiene.

Dear Sirs,

I send you a further short contribution on the subject of Spirochaetosis. It deals with recent work which, I think you will agree, is of special interest, throws fresh light on the mechanism of the crisis & has, I have little

doubt a decision bearing on African Tick Fever & possibly also on other forms of acute & chronic human trypanosomiasis. I have sent a note also to the B. M. J. but this differs from that in certain particulars & I trust may be found suitable for your columns. The work bears out the importance of the Infective Granule to which I drew attention at the B. M. A. meeting last summer when discussing Sir David Bruce's paper on Sleeping Sickness.

Yrs faithfully

Andrew Balfour.

The Rôle of the Infective Granule in certain Protozoal Infections as illustrated by the Spirochaetosis of Sudanese Fowls.

8186

Preliminary Note.

by Andrew Balfour M.D. &c.
Director, Wellcome Tropical Research Laboratories
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91 x 3 Granules

On several occasions I have contributed papers on the "Spirochaetosis of Sudanese Fowls" to the Journal of Tropical Medicine & Hygiene, the last contribution being on October 1st. 1909. I there stated "It is easy to formulate theories but hard to prove them correct; and owing to the difficulty of observing these small bodies (the intra-corpuscular forms) and granules (set free from the red cells) in the fresh blood more work is required upon them and special methods may have to be adopted." It is by the use of these latter, and more especially the dark-field method of illumination and the employment of the Levaditi-Yamamato method for staining sections of tissues and a slight modification of it for blood films, that the interesting problem presented by this disease of fowls has, I believe, at length been cleared up. Not only so, but the more recent investigations may possibly throw light on what is at present obscure in the case of other spirochaetal infections.

The full account of these later researches will be presented in the Fourth Report of these Laboratories which is now in the Press and is due to appear in the autumn of the present year and here I wish merely to place on record a few of the more salient features of the work.

It will perhaps be remembered that one found intra-corpuscular forms in this fowl spirochaetosis and that, following Sambon, one had come to the conclusion that these endoglobular bodies represented a stage in the life-cycle of the spirochaete, constituted in short its stage of schizogony in the fowl. Sambon, however, who expressed this view from the study of a few slides I gave him, did not indicate how this red cell invasion occurred. For a long time

I believed the spirochaetes themselves entered the red cells and broke up, or coiled up, within them to form these remarkable bodies. As the parasites can and do enter and leave the erythroblasts of the fowl there was good ground for this supposition. Now, however, I know better. By the use of the dark-field method and more especial-ly by practising liver puncture on chicks at the crisis or on chicks which have been given a sufficiently large dose of "606" I have found that in the liver in particular, also in the spleen and lung, the spirochaetes undergo an astonishing change. They discharge from their periplastic sheaths spherical granules and it is apparent-ly these granules which enter the red cells, develop in them and complete a cycle of schizogony. The appearance is very remarkable. If a well infected chick be given a dose of "606" the peripheral blood is soon cleared or nearly cleared, of spirochaetes. If then a drop of liver juice be examined by the dark-field method it will be found swarming with spirochaetes and with highly refractile granules. The source of the latter is soon apparent for attention will be directed to spirochaetes which are not moving in the usual way but are in a state of violent contortion or are, so to speak, shak-ing themselves to and fro. Indeed I cannot give a more apt comparison than by likening their movements to those of dogs which have been in water and are shaking themselves vigorously to dry their coats. The object of the spirochaetes, however, is to rid themselves of the bright, spherical granules which can be seen with-in them and which may or may not be aggregations of the so-called chromatin core. These are forced along the periplastic sheath and suddenly discharged from one or other end of the parasite, so that they become free in the medium and dance hither and thither as tiny, solid, spherical, brilliantly white particles. In process of time the spirochaete loses its activity, becomes difficult to see and eventually all that is left of it is the limp and lifeless sheath drifting aimlessly in the fluid and liable to be caught up and swept

away by some still vigorous parasite. Such a sheath may still retain one or two of the granules which it has been unable to discharge. As may be imagined the process is most fascinating to watch and my observations have been confirmed by Captain Fry and Mr. Buchanan of these laboratories and by Major Ensor and Captain O'Farrell, R.A.M.C. I may also say that the first-named had previously seen a shedding off of granules by trypanosomes in the peripheral blood of experiment-al animals, a phenomenon which he is now studying.

It is these spirochaete granules in the liver, spleen and lung and possibly also in other internal organs, which I believe, invade the red cells. I think I have seen the penetration occur but require to make further observations in order to be certain as to the mode of entry. Such a chain of events fully explains all the puzzling features which this intra-corpuscular infection has hitherto presented and moreover brings it into line with the infective granules found in the ticks for these very closely resemble those seen in liver juice films both when examined by the dark-field method and when stained by the Levaditi-Yamamato process.

Until my laboratory assistant, Mr. Buchanan, modified this method one had considerable difficulty owing to the deposit which formed on films. Now, however, one can obtain a clean film which shows the granules both in the spirochaetes and lying free. The process is as follows:- Smears are fixed in absolute alcohol for 10 minutes and washed thoroughly in distilled water after which each slide is placed in an oblong glass trough containing about 10 c.c. of a filtered solution of 5% silver nitrate. The film side faces downwards and the slide rests on small glass rods in order to prevent contact with the bottom of the trough. The troughs are placed on a glass slab and over them is put a suitable glass cover the edges of which where it is in contact with the slab being sealed by vaseline. The whole is then placed in the incubator at 37°C for two days at the end of which time the films are very thoroughly washed in running tap water for about 10 minutes. They are then treated with filtered reducing solution (pyrogallic acid 2%, tannic acid 1%) under the same conditions as above and incubated at

37°C for an hour or so. They are then again washed very thoroughly in running water and transferred to fresh reducing solution in which they are left in the incubator for two days. They are finally very thoroughly washed and dried.

As will be seen this method does not differ from the Yamamato modification of the Levaditi process save as regards the very thorough washing and the more prolonged treatment with the reducing solution.

By its use I have recently been able to confirm observations made with the dark-field upon the peripheral blood of chicks at the crisis and to show that this granule-shedding occurs not only in the internal organs but in the circulating blood stream. Moreover the same thing happens to spirochaetes ingested by ticks and it is no doubt in this way that the granules of Leishman are formed. It is indeed remarkable that while the latter take on the Romanowsky stain the granules shed in the blood of fowls and chicks cannot apparently be coloured by this method, whilst they have endowed the red cells, a curious fact which may explain why they have not been hitherto observed in other spirochaetal infections. It is, however, worth noting that so-called "coccoid" bodies have been seen in the blood in human relapsing fever. Were these free spirochaete granules?

I see that Jowett in South Africa has recently discovered what appears to be an identical form of fowl spirochaetosis and I trust he will employ the dark-field method and endeavour by liver puncture and the use of "606", for the purpose of creating an artificial crisis, to follow out the curious cycle I have indicated. From these observations and others which will be fully detailed at a later date I have come to the conclusion that this fowl spirochaete must be classed as a specific entity and I am proposing for it the name Spirochaeta granulosa penetrans Nov.Sp. which, though lengthy, suit-ably indicates its more important peculiarities. At the same time it is quite possible, nay even probable, that other pathogenic

spirochaetes behave in a similar manner. I have found these granules to be resistant forms and their presence in countless numbers in the tissues might explain part of the mechanism of relapse and the difficulty of curing completely some of the more chronic spirochaetal infections as, for example, syphilis and yaws.

In conclusion I must thank Professor Ehrlich for most kindly placing at my disposal an ample supply of his new and valuable remedy.

Khartoum
22.6.911.

NOTES ON THE HUMAN TRYPANOSOME OF NORTHERN RHODESIA.

By L.E.W.Bevan .M.R.C.V.S. Southern Rhodesia.
Post graduate of the Royal Veterinary College , London, and Pasteur Institute, Paris.

Source of Virus. The virus used to originate the strain of human trypanosomiasis at this laboratory was obtained from an European who , arriving in Southern Rhodesia from the North in November last , was found to be suffering from trypanosomiasis. This case was of considerable interest inasmuch as there appeared grave reason to suppose that the patient had contracted the disease in Northern Rhodesia in an area thought to be free from Glossina palpalis.In these circumstances it became a matter of grave importance to determine the identity of the trypanosome with which he was infected and the manner of its transmission in nature.

Transmission through animals. A supply of blood having been obtained by the courtesy of Dr Heygate Ellis of the Mediacl Department, a number of laboratory animals were inoculated, and among others,sheep and a mule; the facility with which these latter became infected opened up the question as to the part played by the lower animals in connection with human trypanosomiasis in nature. Shortly after these experiments commenced it was announced that Bruce and his colleagues working in Uganda , had found that cattle in the 'fly area'(G palpalis) did naturally harbour T gambiense, - a discovery which rendered the study of Sleeping Sickness a legitimate branch of work in a veterinary laboratory.

The results obtained here have been of some interest and have shown that not only are some of the domestic animals readily susceptible to this human trypanosome but that artificial inoculation gives rise in them to symptoms no less severe than those caused by infection

with the animal trypanosomes.

It has been possible to keep under observation at the same time sheep inoculated with the so-called T dimorphon of North Western Rhodesia, and the animal trypanosome of these territories , and it has been found that the disease arising in sheep inoculated with the human trypanosome has been more acute and characterised by more severe symptoms.

Similarly a mule offered no resistance to a single inoculation with the human parasite which produced a far more severe reaction than the animal trypanosome of Southern Rhodesia to which equines appeared to possess a marked resistance.

Virulence of strain. From an examination of charts of sub-inoculated animals it would appear that the strain is of quite exceptional virulence. - an observation which coincides with the experience of those Medical Officers who have studied the disease in human subjects in the field.

Compared with the recorded results of previous experiments with T gambiense , the period of incubation and the duration of the disease appears to be unusually short, as indicated by the following averages:-

Animal	Average period of incubation	Average duration of disease.
Rabbit	7 days	24 days
Guinea-pig	8 "	38 "
Rat	5½ "	36 "
Mule	6 "	about 100 days
Sheep	6 "	47 "

The above figures are only approximate as in some animals

the course of the disease has been modified by treatment and the intensity of the virus has been altered from time to time by passage through various species of host, In sheep, severe infection is not always associated with the appearance of trypanosomes in the peripheral blood, and the period of incubation can then only be based upon the first definite elevation of temperature.

Symptoms. In most cases the disease runs an acute course, or, after a preliminary stage when symptoms have not been well marked, has terminated by crisis. In rabbits and sheep there is a remarkable oedema of the face especially that part situated between the eyes and nostrils. The photographs give a ~~goddidea~~ good idea of the characteristic appearance presented. In other trypanosomiases oedema at the base of the ears and around the nose occur, but the extent and situation of the swelling in these cases appears to be exceptional. In those sheep which have shewn no other sympton save the irregularly elevated temperature, and where trypanosomes have not been found in the peripheral blood, the swelling of the head has enabled one to recognise infection. If this sympton holds good in natural circumstances it should assist in the detection of 'reservoirs' in those areas where prophylactic measures are being adopted. An elevated temperature has also been present in all cases, but examination of a large number of charts does not reveal any characteristic thermal wave, nor has it been possible to recognise any relation between temperature and the appearance of trypanosomes in the peripheral blood. In sheep for example the disease may run its course without any parasite being detected in the blood, although a small quantity used for the subinoculation of rabbits will give rise to an infection characterised by an abundance of trypanosomes in the blood.

HUMAN TRYPANOSOMIASIS.

SUBJECT:- Black sheep with lamb at heel.

VIRUS:- 3 c.c. warm citrated blood from brown buck rabbit.

DATE:- August 2nd., 1910.

Date.	Temp.	Remarks.
August 3	102'4	
" 4	103	
" 5	104'5	
" 6	105'2	
" 8	104	
" 9	102'6	
" 10	104'8	
" 11	104'8	
" 12	104'4	Rabbit inoculated with 2 c.c. blood.
" 13	104'8	
" 15	105'8	Above rabbit shews trypanosomes.
" 16	105'8	
" 17	106	
" 18	106	
" 19	105'4	
" 20	105'8	
" 22	105'6	
" 23	106'2	Above rabbit dead.
" 24	105	Oedema of throat. Typical swelling of head.
" 25	105'8	
" 26	106	
" 27	106	
" 29	105	
" 30	105	Very ill. Oedema of head and neck.
" 31	104'8	
September 1	102'4	
" 2	...	Dead.

Trypanosomes never found in peripheral blood.

In one or two rabbits somnolence has occurred, and in some of the sheep brain symptoms have predominated during the last few days.

In other respects the symptoms are those common to animal trypanosomiases.

Identity of the Trypanosome. It has been suggested that the human trypanosome of Northern Rhodesia may not be T. gambiense but some animal trypanosome (e.g. T. brucei) habituated by the method of tramsmission or passage, to the human host. This possibility has been borne in mind since the commencement of experiments at this laboratory, and from time to time material has been sent to experts in Europe with the view to establishing the identity of the parasite.

Morphology. The endeavour has been made to classify the parasite by careful study of its morphology, but up to the present no features have been detected which would justify one in differentiating it from T. gambiense; moreover it has been felt that the utmost caution should be taken in this respect inasmuch as the too hasty anouncement of the discovery of a new trypanosome pathogenic to man would naturally cause considerable alarm and economic loss in those countries threatened by its invasion.

Minchin in a "Note on the polymorphism of Trypanosoma Gambiense" (Parisotology Vol.1. No 3,P. 326) recognises three typical forms of this trypanosome when a blood smear is fixed with osmic ~~paper~~ vapour, stained with Giemsa's stain, and mounted with Canada balsam. He claims that "the difference between the three forms in by no means "one merely of size" and recognises:-

1. A slender form of great length and having a very long free flagellum.
2. A stumpy form which is short, the flagellum of which is also very short, especially the free portion.
3. The ordinary form more or less intermediate between the two extreme forms.

The human trypanosome which, although passed through animals .....

animals in this laboratory has retained its morphological characteristics, exhibits in most cases specimens of the three types described by Minchin.

The prevailing type , -of which the long and short forms appear to be derivatives - , may be described as follows:-

Average total length 26-28 m.

Average width I.7 to 2.25 m .

Micro-nucleus about I.75 m from posterior extremity.

Posterior extremity a truncated cone , the apex lying to the side of the median line.

Macro-nucleus longtitudinal ovoid, 4 to 6 m long, posterior edge about 7 m from the posterior end of the parasite.

Undulating membrane well developed and highly festooned (five or more folds)

Flagellum fine with free portion about 7 m long.

Some specimens shew granules and in some there is an area which stains only faintly anterior to the micro-nucleus.

The long form has its posterior extremity drawn out into a'beak' , its body is narrower, the macro-nucleus is elongated and the free flagellum may measure up to I2 m or longer.

In the short forms the posterior extremity is shorter and the micro-nucleus may be terminal. The macro-nucleus is round or slightly ovaland is situated centrally or slightly posterior to centre.The parasite is broader than the other types. The flagellum in shows has little or no free portion.

Another type is frequently met with especially in animals about to die.It is of the long or medium type but stains faintly, the undulating membrane and flagellum being very inconspicuous.

Parasites undergoing degeneration or division , or ~~become~~ altered or distorted in the preparation or staining of the smear, frequently present unusual appearances and may be siezed upon as evidence of a new species.Too much importance should not be attached to such irregularities until various methods of technique have been employed to determine whether they are proper to the parasite or merely artifects.

Atoxyl resistance . It has been stated that the trypanosome of Northern Rhodesia and Nyasaland is remarkably resistant to Atoxyl,a contention which is brought out by the following experiment:-

---

Subject:- Ewe (large brown fat-tail.

Date		
31/10/10	10 a m	Received intravenously I gm Atoxyl in aqueous solution.
"	3,30p m	Received subcutaneously 10 c c citrated blood of lamb No 3 suffering from human trypanosomiasis.
1/11/10	12 a m	Received intravenously ½ gm Atoxyl.
2/11/10		Received subcutaneously 5 c c citrated blood.of lamb No 3.
10/11/10		I c c of this ewe taken and injected at once into rabbit.
		Later. Received I gm Atoxyl.
16/11/10		Above rabbit shewing trypanosomes.
18/11/10	5 p m	Above rabbit died .Blood containing average 25 trypanosomes to the field.
21/11/10		Sheep shewing marked clinical symptoms.

Note:-The strain recovered from lamb No 3 has not been rendered atoxyl-resistant by artificial means but has been shewn by other experiments to be temporarily affected by the exhibition of the drug.

The experiment would appear to indicate that Atoxyl is useless as a preventive against this trypanosome which can establish itself in a host previously saturated with the drug. Further, the parasite recovered from the treated animal appears to have increased in virulence.

Trypan-blue . The trypanosome also appears highly resistant to trypan-blue as is shewn by the following experiment:-

---

Subject :- Ewe No 5.

Virus :- 3 c c warm citrated blood from black ewe.

Date :- 29/8/10

Result :- Temperature and clinical symptoms shewed infection. Trypanosomes also found in blood I6/9/10, 28/9/10, 7/10/10.

October I7th Animal apparently dying.
Blood taken and inoculated into Control rabbit No I .

Later. Received intravenously I00 c c of I½% solution Trypan-blue.

I8th Animal still very sick Intensely stained.

I c c blood taken from Ewe and inoculated into Control rabbit No 2.

Later. Received I0 c.c. of I0% solution of Atoxyl intravenously.

2Ist. 4 c c blood taken (76 hours after injection of Atoxyl and injected into Control rabbitNo3

29th. Ewe died.

Control Rabbit No I	Control Rabbit No 2	Control Rabbit No 3
Oct 22 Trypanosomes seen.	Oct 22 Trypanosomes seen.	NovI6 Still alive
Oct 26 Died after bleeding.	Nov I Died.	Trypanosomes never seen.

Post mortem examination of ewe shewed well marked staining of the tissues with blue. The meninges were highly coloured but the brain itself was not stained.

Various methods of treatment, both with drugs and sera have been tried but up to the present no successful results can be recorded. No case of natural recovery or immunity has been encountered: those animals which have appeared resistant or tolerant have eventually succumbed.

It has frequently happened that the exhibition of an agent of low parasitotropic but high organotropic properties, to an animal in which the disease is running a normal course , has brought about crisis. The use of drugs in unsuitable doses has produced similar results.

It may be mentioned that experiments have been conducted which have proved that the tissues of a foetus of a highly infected mother are not infective; also that the milk of an infected ewe does not convey infection to the lamb feeding upon it, or to animals artificially inoculated with it. Lambs feeding upon such milk derive no immunity therefrom.

Remarks on some Cultural Characters
of the Fungi of Tinea Imbricata.

by

Dr Aldo Castellani M. D.
Director, Clinic for Tropical Diseases,
Colombo.

The description I have given of Endodermophyton Concentricum and Endodermophyton Indicum (Journal of Tropical Medicine of March 15, 1911) is based on the appearances of cultures kept in the dark, at the temperature of the room (80 to 90 Far) and using tubes closed in the usual manner with cotton wool plugs, without rubber caps. If any of these conditions is altered the cultural characters may be greatly changed. For instance, when rubber-caps are used both Endodermophyton Indicum and Endodermophyton Concentricum take often a beautiful bright -red colour. If however, subcultures are made from these , using tubes closed in the ordinary way with cotton wool plugs, without rubber caps, the fungi show again after a time the same appearances I have described in my papers. Various changes in the cultural character of the fungi take place, as it was to be expected, also when they are exposed for a long time to strong light, lower or higher temperature than usual &c.

I may take this opportunity to state that further inoculation experiments have amply confirmed that the malady can be easily reproduced in human beings by inoculation of pure cultures of the fungi, as described in my previous publications.

Reference December 15, 1910 and
Journal of Tropical Medicine March 15, 1911

Pemphigus Contagiosus

Scott. 55

Bursa.

June 19th, 1911

Dear Sir,

As neither Manson nor Castellain mention Bursa in the geographical distribution of this disease, I wish to point out that it is fairly common in the hot weather in this country. I recently came across 6 cases in one family with the following history:

Case I - Mrs. H,, aged 30, first noticed the eruption on the 28th April, 1911 on the back of her right forearm and about the middle. When I saw it the vesicle had burst and was drying up.

Case II - E.H. , aged 10 months, male. Two days later the vesicles appeared on her child; about the forehead there were several of them. They then extended to the face, upper part of chest, and the front ofthe right forearm.

Case III - E.H., aged 6 years, male, was attacked 3 days later and they first appeared on the abdomen; then a few showed themselves at the root of the neck, and only one vesicle appeared on the right cheek.

Case IV - C.H., aged 3 years, female,. In this patient the

eruption appeared practically on the same day as Case III, and first shewed itself on the lower part of the chin; then a few appeared on the forehead and forearms.

Case V F.H., aged 12 years, female. In this case the eruption appeared on the chin, and then extended to the forearms. It appeared two days later than Cases IV and V.

Case VI - G.H., aged 6 years, male. It appeared on the same day as Case V, The disease first shewed itself on the chin, and there was only one other vesicle on the wrist.

Remarks

(a) It will be seen that in three of these cases the eruption first appeared on the chin; in one it appeared on the forearm, in another it appeared on the abdomen, and in the infant on the forehead. The usual places like the crutch, axilla etc were not attacked.

(b) The course of the eruptions followed was the usual one. The sizes of the vesicles varied from a pea to about those covering the area of an inch. They did not affect the general health of the children.

(c) In case VI there were only 2 vesicles, in case I there were four. Case III shewed 10 vesicles and in the others

there were several. The most diffused cases were in the children aged 10 months and 3 years respectively, and in these two cases they gave rise to much discomfort and irritation.

(d) The cases lasted from one to three weeks.

(e) The treatment was cleanliness, frequent use of Mercury lotion and antiseptic powder dusting.

R.H. Castor,

Lt,Col. I.M.S.

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IS BLACKWATER FEVER THE EXPRESSION OF ANAPHYLAXIS TO A MALARIAL PLASMODIUM?

By J. Burton Cleland, M.D., Ch.M. Principal Assistant Microbiologist, Bureau of Microbiology, Sydney.

The study of precipitins and allied bodies has led to the discovery of that remarkable condition, known as anaphylaxis, a hypersensitiveness to the introduction into the circulation of small quantities of the antigen (known as anaphylaxis). Naturally this has resulted in directing the attention of pathologists to the question of the possible rôle anaphylaxis may play in setting up certain diseased processes whose etiology is still obscure. For instance, the onset of puerperal eclampsia can be more satisfactory explained on this basis than on any other, a conclusion I had come to independently and had discussed with others before finding, in the Medical Record of April 3rd, 1909, that others had forestalled me. For several years I had held the view that this condition must almost certainly be due to the setting free in the maternal circulation of small portions of the trophoblast of the foetus and to the probable solution of these in the blood. That such foetal elements do frequently enter the mother's blood vessels and pass on as emboli is very reasonable considering the way the syncytium eats into the maternal sinuses, and is proved by the frequent finding of small syncytial elements in the lung capillaries, etc. quite apart from true pulmonary embolism. Such a solution of foetal cells in the maternal blood might be quite well supposed to produce toxic effects on the kidney, liver and brain cells, etc., with the onset of symptoms of eclampsia. This view was not quite satisfactory, however, since, if correct, one would have expected the more frequent occurrence of eclampsia. By the discovery of anaphylaxis, however, a possible clue was given. It would only require the solution of a certain amount of foetal syncytium, followed after an interval of some days by the solution of another small amount, to set up anaphylaxis if such were possible under these conditions.

If the doses of foetal proteids followed each other more closely, then immunity instead would ~~follow~~ result, which perhaps is the rule in pregnant women and hence the rarity of puerperal eclampsia. Anaphylaxis, then, is now recognised as a possible explanation of the nature of this disease and, such being the case, the above resumé may help us to understand the part it possibly plays also in Blackwater fever.

The ~~case~~ relations of the organisms of malaria ~~and~~ & their hosts is a very peculiar one, as in fact is the case with all haematozoa and their hosts. Here we have living masses of protoplasm, at times free in the plasma though, in the case of Plasmodia, for most of their time parasitic in the red corpuscles. It is evident that, under ordinary circumstances, these bodies being living maintain their existence and do not undergo solution or disintegration. It must happen, however, that at times naturally and after the administration of quinine frequently, a greater or small or number of the youngest merozoites, the free forms in the blood, die. This must lead to the presence in the plasma of a (dead) proteid foreign to it. In other words, we have the same state produced in the human body as would occur after the injection of an antigen such as a foreign blood into the peritoneal cavity of an animal for the purpose of preparing a precipitin. Now anaphylaxis is well-known as the result of the injection, after a certain interval, of another dose of the original antigen and this is manifested by severe and often fatal symptoms. In the case of blackwater fever it may be that an exactly analogous event takes place: that a number of the small free forms of the malarial parasite die naturally or are killed by the administered quinine: that their protoplasm, after solution in the plasma, sets going the process that may eventuate in the formation of a specific precipitin: that, after an interval sufficiently long to set up anaphylaxis, a second batch likewise die and enter into solution: and that blackwater fever is the resultant condition, the evidence of anaphylaxis to (dead) plasmodium proteid.

A view such as this would explain much. By it we can understand the rôle that quinine administration has, by some, been considered to play in the onset of blackwater fever: the occurrence of cases of the disease in malarial patients after their return to England, having never had it before, is understood: its occurrence usually after a year or two's residence in malarial districts and not soon after arrival, and recurrent attacks of the fever, are explained: and even its notoriety in some districts and absence in others may be accounted for by supposing that certain climatic conditions or other local causes exercise a lethal effect at irregular intervals on the successive broods of parasites, which have perhaps become more or less habituated to quinine. The suddenness of the onset of the symptoms is also highly suggestive.

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CYPRUS

No.84

Government House,
Nicosia,
22nd May, 1907.

My Lord,

Referring to Mr Secretary Lyttelton's circular despatch of the 21st July 1904 on the subject of the publication of interesting medical reports received from the Crown Colonies, I have the honour to enclose for transmission to Sir P.Manson, a copy of a report by Dr Cleveland, the District Medical Officer of Nicosia, on a case of Mycetoma of the foot which was treated by him in the Nicosia Hospital.

2. This report may be considered to be of sufficient interest to be published in "The Journal of Tropical Medicine", and should it be desired by Sir P.Manson, I can forward half of the foot which was amputated and also a microscopic stained film prepared by Dr.Cleveland.

I have, &c.,

(Sd.) C.A.KING-HARMAN.

THE RIGHT HONOURABLE
THE EARL OF ELGIN, K.G.,
&c., &c., &c.,
SECRETARY OF STATE FOR THE COLONIES.

O.C.

Staveley

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A CASE OF MYCETOMA in Cyprus ~~ON THE FOOT~~

~~AMPUTATION AND RECOVERY~~

by. Cleveland. District Medical Officer
Nicosia, Cyprus.

HISTORY.

The parents of a man named B. Andoni brought their son to the Out-patient Department of the Nicosia General Hospital, Cyprus, on the 2nd November 1906. Patient is aged about 25, ~~years~~. They came from the village of Kormakiti in the Kyrenia District, and call themselves Maronites. The patient, who is of weak intellect and partially blind, was employed by his father in looking after his pigs, and went about bare-footed in the fields. A history and dates of present illness were difficult to obtain, but he appears to have had good health when young. Some years ago (perhaps 10 years) he noticed a small painful ulcer on the right foot. The ulcer did not heal, and was followed by others, and the foot became swollen. He saw several Doctors, and got no benefit for treatment. He thinks the disease was caused by wearing tight boots, but generally he went bare-footed. His manner of life would probably be very dirty, as his village is a remote one at the eastern end of the Island, and not noted for cleanliness.

FAMILY HISTORY.

Father and mother both living and in good health. There were two other children, both dead. One died when two months old, the other at 18 years, of consumption. No history of syphilis.

Present

PRESENT CONDITION.

Patient was admitted to the wards for amputation of the right foot. He is a feeble young man, almost blind from extensive corneal disease (probably due to acute opthalmia) in childhood. He is of weak intellect, and poor development. With the exception of the right foot there is no abnormal condition. The whole of the right foot, and lower third of the leg is greatly swollen and covered with reddish yellow granulation like projections, each have an opening which admits a probe into the deeper parts of the foot in any direction. There is a foul serous and purulent discharge from these openings. The planter surface of the foot is convex, and the toes turned upwards. The general appearance of the foot closely resembles Madura Foot. On ~~the~~ 7th November, 1906, the foot was amputated through the middle third of the leg. The patient did well and the parts healed by first intention. He was discharged, cured, on ~~the~~ 25th November 1906, and there was no sign of a recurrence of the disease.

NOTE AFTER OPERATION.-

The foot was found to be extensively diseased, and presented all the appearances of Mycetoma of the white variety, many of the bones of the foot having disappeared. The foot was easily cut in a longitudinal direction with a knife, being of the consistence of cheese. On section it presented a yellowish oily mass with numerous cavities and sinuses

in

in all directions. These are filled with bright yellow granular particles. Films on glass slides were taken of the discharge and granular particles, and most of these yielded good specimens of ray fungus, closely resumbling Actinomyces and staining by Gram's Method. No attempt was made to cultivate the fungus.

WEEKLY RETURN

OF

Deaths in Calcutta for the week ending Saturday the 9th July 1898.

Total population of Calcutta by Census of 1891, 6,81,560—Males 4,46,746, Females 2,34,814.
Ditto of Urban Calcutta „ ... 4,66,460— „ 3,16,667, „ 1,49,793.
Ditto of Suburban Calcutta „ ... 2,15,100— „ 1,30,079, „ 85,021.

Class.	Causes of Death.	Non-Asiatics.				Mixed Races.				Asiatics.												Total deaths of all ages.								
		Males.		Females.		Males.		Females.		Hindoos.				Mahomedans.				Other classes.				Males.				Females.				Total.		
		Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.
I	ZYMOTIC DISEASES.																													
	ORDER I.																													
	1. Small-pox											1	–												1	–	1	1	–	1
	2. Measles											1	–												1	–	1	1	–	1
	3. Scarlet Fever																													
	4. Diphtheria																													
	5. Quinsy																													
	6. Croup									1	–											1	–	1				1	–	1
	7. Whooping Cough															1	–								1	–	1	1	–	1
	8. Typhus Fever / Enteric or Typhoid Fever / Simple continued Fever									17	2	8	5	3	–	3	6					20	2	22	11	11	22	31	13	44
	9. Erysipelas																													
	10. Puerperal Fever (Metria)																													
	15. Carbuncle																													
	18. Influenza																													
	13. Dysentery									4	3	3	4	2	2	1	2					6	5	11	4	6	10	10	11	21
	14. Diarrhœa							1	–	4	4	3	1	2	2	2	1					6	6	12	6	2	8	12	8	20
	15. Cholera									2	–											2	–	2				2	–	2
	16. Ague																													
	17. Remittent Fever									5	1	4	2	–	–	–	1	1	–			6	1	7	4	3	7	10	4	14
	18. Rheumatism									2	–											2	–	2				2	–	2
	19. Other Zymotic Diseases Plague							1	–	3	–	–	–	3	1	2	–					6	1	7	3	–	3	9	1	10
	ORDER II.																													
	1. Syphilis																													
	2. Stricture of Urethra																													
	3. Hydrophobia																													
	4. Glanders																													
	ORDER III.																													
	1. Privation																													
	2. Want of Breast milk																													
	3. Purpura and Scurvy																													
	4. ALCOHOLISM a. Del. Tremens. b. Intem.																													
	ORDER IV.																													
	1. Thrush																													
	2. Worms, &c.																													
II	CONSTITUTIONAL.																													
	ORDER I.																													
	1. Gout											1	–												1	–	1	1	–	1
	2. Dropsy									1	–	–	1	–								1	–	1	–	1	1	1	1	2
	3. Cancer																													
	4. Cancrum Oris (Noma)																													
	5. Mortification																													
	ORDER II.																													
	1. Scrofula																													
	2. Tabes Mesenterica					1	–			2	1	3	1	1	3	2	5					4	4	8	5	6	11	9	10	19
	3. Phthisis																													
	4. Hydrocephalus																													
III	LOCAL DISEASES.																													
	ORDER I.																													
	1. Cephalitis																													
	2. Apoplexy									1	–	1	1	1	–							2	–	2	1	1	2	3	1	4
	3. Paralysis																													
	4. Insanity																													
	5. Chorea																													
	6. Epilepsy																													
	7. Convulsions																													
	8. Brain Disease, &c.									2	–			–	1							2	1	3				2	1	3
	9. Tetanus																													
	10. Hysteria																													
	11. Otalgeny																													
	ORDER II.																													
	1. Pericarditis																													
	2. Aneurism									2	–											2	–	2				2	–	2
	3. Heart Disease, &c.																													
	ORDER III.																													
	1. Laryngitis					1	–					2	1	–	1	1	–	–	1			1	2	3	3	1	4	4	3	7
	2. Bronchitis																													
	3. Pleurisy									–	–	1	–	1	–					1	–	1	–	1	2	1	3	3	1	4
	4. Pneumonia							–	1	2	2	5	1	5	2	3	3					7	4	11	8	4	12	15	8	23
	5. Asthma																								1	–	1	1	–	1
	6. Lung Disease, &c.							1	–																					
	7. Goitre																													
	Carried over	1	–	–	–	1	–	3	1	48	13	33	17	18	12	16	18	1	1	1	–	69	26	95	52	36	88	121	62	183

Class.	Causes of Death.	Non-Asiatics.				Mixed Races.				Asiatics.										Total deaths of all ages.								
		Males.		Females.		Males.		Females.		Hindoos.				Mohammedans.				Other classes.										
																				Males.			Females.			Total.		
		Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Males. Town.	Males. Suburbs.	Females. Town.	Females. Suburbs.	Males. Town.	Males. Suburbs.	Females. Town.	Females. Suburbs.	Males. Town.	Males. Suburbs.	Females. Town.	Females. Suburbs.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.
	Brought forward	1	..	..	..	1	..	3	1	48	13	38	17	18	12	15	18	1	1	1	..	69	26	95	52	36	88	121	62	183
III	LOCAL DISEASES.—(Contd.)																													
	ORDER 4.																													
	1. Gastritis																													
	2. Enteritis																													
	3. Peritonitis																													
	4. Ascites									..	1											..	1	1				..	1	1
	5. Ulceration of Intestines ..																													
	6. Hernia																													
	7. Ileus																													
	8. Intussusception ..																													
	9. Stricture of Intestines ..																													
	10. Fistula																													
	11. Stomach Disease, &c. ..																													
	12. Pancreas Disease, &c. ..																													
	13. Hepatitis																													
	14. Jaundice									1	..																			
	15. Liver Disease, &c. ..									6	3	2	..	1	1	1	..					1	..	1				1	..	1
	16. Spleen Disease, &c. ..									..	2	2	1	1	..	1	1					7	4	11	3	..	3	10	4	14
	17. Colic													1	..							1	2	3	3	2	5	4	4	8
	18. Obstruction of Bowels ..																					1	..	1				1	..	1
	19. Hæmorrhoids																													
	20. Constipation																													
	ORDER 5.																													
	1. Nephritis																													
	2. Ischuria																													
	3. Bright's Disease (Nephritis) ..																													
	4. Diabetes																													
	5. Calculus (Stone) ..									..	1											..	1	1				..	1	1
	6. Cystitis									1	..											1	..	1				1	..	1
	7. Kidney Disease ..																													
	ORDER 6.																													
	1. Ovarian Dropsy																													
	2. Uterus Disease, &c. ..																													
	ORDER 7.																													
	1. Synovitis (Arthritis) ..																													
	2. Joint Disease, &c. ..																													
	3. Necrosis																													
	ORDER 8.																													
	1. Phlegmon																													
	2. Ulcer																													
	3. Skin Disease, &c. ..																													
	4. Abscess																													
IV	DEVELOPMENTAL DISEASES.																													
	ORDER 1.																													
	1. Premature Birth ..	1	..									1	..									1	..	1	1	..	1	2	..	2
	2. Cyanosis																													
	3. Spina Bifida																													
	4. Other Malformations ..																													
	5. Teething																													
	ORDER 2.																													
	1. Parturition																													
	2. Child-birth												1													1	1	..	1	1
	ORDER 3.																													
	1. Old age													2	..	1	2	..	..	1	..	2	..	2	2	2	4	4	2	6
	ORDER 4.																													
	1. Atrophy and Debility ..												1																	
V	VIOLENT DEATHS.																									1	1	..	1	1
	ORDER 1.																													
	1. Fractures and Contusions ..																													
	2. Gunshot Wounds ..																													
	3. Cut-shot									1	..											1	..	1				1	..	1
	4. Burns and scalds ..									..	1	1	..									..	1	1	1	..	1	1	1	2
	5. Poison																													
	6. Drowning																													
	7. Suffocation																													
	8. Otherwise									..	1	..	..	3	..							3	1	4				3	1	4
	ORDER 2. (Homicide.)																													
	1. Murder and man-slaughter ..																													
	ORDER 4. (Suicide.)																													
	1. Gunshot Wounds ..																													
	2. Cut-shot																													
	3. Poison																													
	4. Drowning																													
	5. Hanging												1	..	..	1	..								1	1	2	1	1	2
	6. Otherwise																													
	ORDER 5 (Execution.)																													
	1. Hanging																													
	Other violent deaths (not classed)																													
	Sudden deaths (cause unascertained)									1	1	1	2	2	2	3	1					11	3	7	4	3	7	8	6	14
	Causes not specified, or ill-defined	2	..																											
	TOTAL	3	..	..	..	1	..	3	1	58	23	46	23	28	15	22	22	1	1	2	..	91	39	130	67	46	113	168	85	243
										Deaths per 1,000 of population								per 1,000				18·9	15·6	15·1	28·3	28·2	25·1	17·6	20·8	18·6
	Still-births									1	2	1	..									1	2	3	1	..	1	2	2	4

Remarks—

URBAN CALCUTTA.

(1) The total number of deaths registered during the week ending 9 July was 158 against 159 and 216 in the two preceding weeks, and lower than the corresponding week of last year by 68. There were 2 deaths from cholera, against 6 and 10 in the two preceding weeks; the number is lower than the average of the past quinquennium by 8. There were one deaths from small-pox during the week, against 3 in the previous week. There were 2 deaths from tetanus against 2 in the previous week. The mortality from fevers and bowel-complaints amounted to 41 and 22 respectively, against 30 and 27 in the preceding week. The general death-rate of the week was 17.6 per mille per annum, against 25.9 the mean of the last five years.

∧ There were 9 deaths from Plague against 14 and 19 in the two preceding weeks

SUBURBAN CALCUTTA.

(2) The total number of deaths registered during the week ending 9 July was 95 against 107 and 96 in the two preceding weeks, and lower than the corresponding week of last year by 45. There were one deaths from cholera, against 3 and 2 in the two preceding weeks ~~the number is than the average of the past quinquennium by~~. There were one deaths from small-pox during the week, ~~against in the previous week.~~ There were one deaths from tetanus against nil in the previous week. The mortality from fevers and bowel-complaints amounted to 17 and 19 respectively, against 25 and 20 in the preceding week. The general death-rate of the week was 20.6 per mille per annum, against 30.5, the mean of the last five years.

∧ There was one death from plague near having occurred in the two preceding weeks.

(3) The general death-rate of the combined area is equal to 18.6

CALCUTTA:
The 15 July 1898.

J. McCosh, M. D., P. H.
Health Officer, Calcutta.

2054—11-1-97—2,000.

89186
6164

WEEKLY RETURN

OF

Deaths in Calcutta for the week ending Saturday the 2nd July 189

Total population of Calcutta by Census of 1891, 6,81,560—Males 4,46,746, Females 2,34,814.
Ditto of Urban Calcutta „ ... 4,66,460— „ 3,16,667, „ 1,49,793.
Ditto of Suburban Calcutta „ ... 2,15,100— „ 1,30,079, „ 85,021.

Class.	Causes of Death.	Non-Asiatics. Males. Town.	Non-Asiatics. Males. Suburbs.	Non-Asiatics. Females. Town.	Non-Asiatics. Females. Suburbs.	Mixed Races. Males. Town.	Mixed Races. Males. Suburbs.	Mixed Races. Females. Town.	Mixed Races. Females. Suburbs.	Asiatics. Hindoos. Males. Town.	Asiatics. Hindoos. Males. Suburbs.	Asiatics. Hindoos. Females. Town.	Asiatics. Hindoos. Females. Suburbs.	Asiatics. Mahomedans. Males. Town.	Asiatics. Mahomedans. Males. Suburbs.	Asiatics. Mahomedans. Females. Town.	Asiatics. Mahomedans. Females. Suburbs.	Asiatics. Other classes. Males. Town.	Asiatics. Other classes. Males. Suburbs.	Asiatics. Other classes. Females. Town.	Asiatics. Other classes. Females. Suburbs.	Total deaths of all ages. Males. Town.	Males. Suburbs.	Males. Combined.	Females. Town.	Females. Suburbs.	Females. Combined.	Total. Town.	Total. Suburbs.	Total. Combined.
I	ZYMOTIC DISEASES.																												
	ORDER 1.																												
	1. Small-pox									..	..	3	..									..	..	..	3		3	3		3
	2. Measles																												
	3. Scarlet Fever																												
	4. Diphtheria																												
	5. Quinsy									..	..	1	..	..	1	1	1					..	1	1	2	1	3	2	2	4
	6. Croup																												
	7. Whooping Cough																												
	8. Typhus Fever / Enteric or Typhoid Fever / Simple continued Fever									7	5	8	6	3	7	5	2					10	12	22	13	8	21	23	20	43
	9. Erysipelas																												
	10. Puerperal Fever (Metria)									..	..	1	..									..	..	..	1		1	1		1
	11. Carbuncle									1												1		1				1		1
	12. Influenza									5	2	5	3	3	4	3	2					8	6	14	8	5	13	16	11	27
	13. Dysentery									3	6	6	1			1	2	..	..	1	..	3	6	9	8	3	11	11	9	20
	14. Diarrhœa									1	.	1	1	2	1	2	.	.	1			3	2	5	3	1	4	6	3	9
	15. Cholera																												
	16. Ague									5	2	2	1	..	..	..	2					5	2	7	2	3	5	7	5	12
	17. Remittent Fever																												
	18. Rheumatism																												
	19. Other Zymotic Diseases																												
	20. Plague									16	1	1		5	.	2	1					15	1	16	3	1	4	18	2	20
	ORDER 2.																												
	1. Syphilis									..	1	1	..									..	1	1	1		1	1	1	2
	2. Stricture of Urethra																												
	3. Hydrophobia																												
	4. Glanders																												
	ORDER 3.																												
	1. Privation									..	..	..	1									..	..	..	..	1	1	..	1	1
	2. Want of Breast milk																												
	3. Purpura and Scurvy																												
	4. Alcoholism [a. Del. Trem. b. Intem.																												
	ORDER 4.																												
	1. Thrush																												
	2. Worms, &c.																												
II	CONSTITUTIONAL.																												
	ORDER 1.																												
	1. Gout									3	..	..	..	..	1	..	1					3	1	4	..	1	1	3	2	5
	2. Dropsy																												
	3. Cancer																												
	4. Cancrum Oris (Noma)																												
	5. Mortification																												
	ORDER 2.																												
	1. Scrofula																												
	2. Tabes Mesenterica					1	.			5	5	2	2			2	3					6	6	11	4	5	9	10	10	20
	3. Phthisis																												
	4. Hydrocephalus																												
III	LOCAL DISEASES.																												
	ORDER 1.																												
	1. Cephalitis																												
	2. Apoplexy									1	..	..	..	..	1							1	1	2	..	..	..	1	1	2
	3. Paralysis																												
	4. Insanity																												
	5. Chorea																												
	6. Epilepsy																												
	7. Convulsions																												
	8. Brain Disease, &c.									..	..	..	..	2								2	..	2	..	..	..	2		2
	9. Tetanus																												
	10. Hydæria																												
	11. Otalgia																												
	ORDER 2.																												
	1. Pericarditis																												
	2. Aneurism																								1		1	1		1
	3. Heart Disease, &c.							1	..																				
	ORDER 3.																												
	1. Laryngitis									.	1	2	.	.	2							.	3	3	2		2	2	3	5
	2. Bronchitis															1						.	1	1	1		1	1	1	2
	3. Pleurisy									4	1 2	2	1	1	.	4	4					5	2	7	6	5	11	11	7	18
	4. Pneumonia																												
	5. Asthma																												
	6. Lung Disease, &c.																												
	7. Goitre																												
	Carried over					1	.	1	.	15	26	38	8	16	17	21	18	.	1	1	.	62	44	106	53	34	92	120	78	198

Class.	CAUSES OF DEATH.	Non-Arbtics.				Mixed Races.				Asiatics.												Total deaths of all ages.								
										Hindoos.				Mohammedans.				Other classes.				Males.			Females.			Total.		
		Males.		Females.		Males.		Females.		Males.		Females.		Males.		Females.		Males.		Females.										
		Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.
III	Brought forward					1	.	1	.	45	26	35	16	16	17	21	18	.	1	1	.	62	44	106	58	34	92	120	78	198
	LOCAL DISEASES.—(Cont.)																													
	ORDER 4.																													
	1. Gastritis									1	"	1										1	"	1	"	"	"	1		1
	2. Enteritis																													
	3. Peritonitis																													
	4. Ascites									"	"	"	1									"	"	"	"	1	1		1	1
	5. Ulceration of Intestines																													
	6. Hernia																													
	7. Ileus																													
	8. Intussusception																													
	9. Stricture of Intestines																													
	10. Fistula																													
	11. Stomach Disease, &c.																													
	12. Pancreas Disease, &c.																													
	13. Hepatitis																													
	14. Jaundice																													
	15. Liver Disease, &c.									2	.	4	.	1	1	1						3	1	4	5	.	5	8	1	9
	16. Spleen Disease, &c.													1	6		2					1	6	7	.	2	2	1	8	9
	17. Colic									2	..	..	..									2	.	2	..	..	..	2	.	2
	18. Obstruction of Bowels																													
	19. Haemorrhoids																													
	20. Constipation																													
	ORDER 5.																													
	1. Nephritis																													
	2. Ischuria																													
	3. Bright's Disease (Nephritis)																													
	4. Diabetes								..	"	1	"	1									"	1	1	"	1	1	"	2	2
	5. Calculus (Stone)																													
	6. Cystitis																													
	7. Kidney Disease																													
	ORDER 6.																													
	1. Ovarian Dropsy																													
	2. Uterine Disease, &c.																													
	ORDER 7.																													
	1. Synovitis (Arthritis)																													
	2. Joint Disease, &c.																													
	3. Necrosis																													
	ORDER 8.																													
	1. Phlegmon																													
	2. Ulcer																													
	3. Skin Disease, &c.																													
	4. Tumour									1	.	1	.			1						1		1	2		2	3	.	3
IV	DEVELOPMENTAL DISEASES.																													
	ORDER 1.																													
	1. Premature Birth		/				/		..	..	..												1	1	1		1	1	1	2
	2. Cyanosis																													
	3. Spina Bifida																													
	4. Other Malformations																													
	5. Teething													1								1		1				1		1
	ORDER 2.																													
	1. Parturition																													
	2. Child-birth															1							..	..	..	1	1		1	1
	ORDER 3.																													
	1. Old age											1	1			2						..	..	..	1	3	4	1	3	4
	ORDER 4.																													
	1. Atrophy and Debility																													
V	VIOLENT DEATH.																													
	ORDER 1.																													
	(Accident or negligence.)																													
	1. Fractures and Contusions									3												3		3				3		3
	2. Gunshot Wounds																													
	3. Cut-stab																													
	4. Burns and scalds									/												1		1				1		1
	5. Poison																													
	6. Drowning					1																1		1				1		1
	7. Suffocation																													
	8. Otherwise									.	1				1							1	1	2				1	1	2
	ORDER 2.																													
	(Homicide.)																													
	1. Murder and man-slaughter																													
	ORDER 3.																													
	(Suicide.)																													
	1. Gunshot Wounds																													
	2. Cut-stab																													
	3. Poison																													
	4. Drowning																													
	5. Hanging																													
	6. Otherwise																													
	ORDER 4.																													
	(Execution.)																													
	1. Hanging																													
	Other violent deaths (not classed)																													
	Sudden deaths (cause unascertained)	1								3	.	2	4	1	8	2	2	1				11	5	16	4	6	10	15	11	26
	Causes not specified, or ill-defined																													
	TOTAL	1	1			2	.	2	.	63	28	43	23	21	29	25	29	1	1	1	.	88	67	147	71	48	119	159	107	266
										Ratio per 1,000 of population.												14.4	23.4	17.1	24.7	27.4	26.4	17.7	25.9	20.3
	Still-births									1	2	3	.	.	1	.	1					1	3	4	3	1	4	4	4	8

Remarks—

URBAN CALCUTTA.

(1) The total number of deaths registered during the week ending 2nd July was 159 against 216 and 216 in the two preceding weeks, and Lower than the corresponding week of last year by 117. There were 6 deaths from cholera, against 10 and 16 in the two preceding weeks; the number is Lower than the average of the past quinquennium by 8. There were 3 deaths from small pox during the week, against 1 in the previous week. There were 2 deaths from tetanus against 3 in the previous week. The mortality from fevers and bowel-complaints amounted to 30 and 27 respectively, against 47 and 29 in the preceding week. The general death-rate of the week was 17·7 per mille per annum, against 25·0 the mean of the last five years.

SUBURBAN CALCUTTA.

(2) The total number of deaths registered during the week ending 2nd July was 107 against 96 and 80 in the two preceding weeks, and Lower than the corresponding week of last year by 57. There were 3 deaths from cholera, against 2 and 4 in the two preceding weeks; the number is Lower than the average of the past quinquennium by 7. There were nil deaths from small-pox during the week, against ——— in the previous week. There were nil deaths from tetanus against 1 in the previous week. The mortality from fevers and bowel-complaints amounted to 25 and 20 respectively, against 16 and 27 in the preceding week. The general death-rate of the week was 25·9 per mille per annum, against 32·0, the mean of the last five years.

(3) The general death-rate of the combined area is equal to 20·3

J. M. Cook, M. D., I. M. S.
Health Officer, Calcutta.

CALCUTTA:
The 8th July 1898.

2054—11-1-97—3,000.

WEEKLY RETURN

OF

Deaths in Calcutta for the week ending Saturday the 16th July 1898.

Total population of Calcutta by Census of 1891, 6,81,560—Males 4,46,746, Females 2,34,814.
Ditto of Urban Calcutta „ ... 4,66,460— „ 3,16,667, „ 1,49,793.
Ditto of Suburban Calcutta „ ... 2,15,100— „ 1,30,079, „ 85,021.

Class.	Causes of Death.	Non-Asiatics.				Mixed Races.				Asiatics.								Total deaths of all ages.								
										Hindoos.				Mahomedans.				Other classes.								
		Males.		Females.		Males.		Females.		Males.		Females.		Males.		Females.		Males.		Females.		Males.		Females.		
		Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.
I	ZYMOTIC DISEASES.																									
	ORDER I.																									
	1. Small-pox									·	·	1	·									1	·	1	1	·	1
	2. Measles																									
	3. Scarlet Fever																									
	4. Diphtheria																									
	5. Quinsy																									
	6. Croup																									
	7. Whooping Cough									1	·	1	1	·	·	·	1					1		1	1	2	3	2	2	4
	8. (Typhus Fever																									
	Enteric or Typhoid Fever					1				18	5	10	6	5	3	·	1					24	8	32	10	7	17	34	15	49
	Malaria continued Fever										1												1	1					1	1
	9. Erysipelas																									
	10. Puerperal Fever (Metria)																									
	11. Carbuncle																									
	12. Influenza																									
	13. Dysentery									5	1	3	1	4	1	3	3					9	2	11	6	4	10	15	6	21
	14. Diarrhœa					1				4	1	6	1	1								5	1	6	6	2	8	11	3	14
	15. Cholera									5	·	1										5	·	5	1	·	1	6	·	6
	16. Ague																									
	17. Remittent Fever									9	·	5	2	1	·	1				1		10	·	10	7	2	9	17	2	19
	18. Rheumatism											1													1		1	1		1
	19. Other Zymotic Diseases										2												2	2				2	2
	20 Plague (Order 3)									6		2		2		1		1				9		9	3	·	3	12	·	12
	ORDER 2.																									
	1. Syphilis																									
	2. Stricture of Urethra																									
	3. Hydrophobia										1											·	1	1				·	1	1
	4. Glanders																									
	ORDER 3.																									
	1. Pediculus																									
	2. Want of Breast milk																									
	3. Podagra and Scurvy																									
	4. Atrophy (a. Ten. Town. b. Infants																									
	ORDER 4.																									
	1. Tetanus																									
	2. Wasting &c.																									
II	CONSTITUTIONAL.																									
	ORDER 1.																									
	1. Gout									1												1		1				1		1
	2. Do pay										1	1										·	1	1	1		1	1	1	2
	3. Cancer											1													1		1	1		1
	4. Cancrum Oris (Noma)											1	1	1	1							1	1	2	1	1	2	2	2	4
	5. Mortification																									
	ORDER 2.																									
	1. Scrofula									1												1		1				1		1
	2. Tabes Mesenterica																									
	3. Phthisis									2	4	4	2	3	3	3	·			1	·	5	7	12	8	2	10	13	9	22
	4. Hydrocephalus																									
III	LOCAL DISEASES.																									
	ORDER 1.																									
	1. Cephalitis																									
	2. Apoplexy											2													2		2	2		2
	3. Paralysis											1			1	1						1	1	2	1		1	2	1	3
	4. Insanity																									
	5. Chorea																									
	6. Epilepsy									1												1		1				1		1
	7. Convulsions												1												·	1	1	1	1	1
	8. Brain Disease, &c.									1												1		1				1		1
	9. Tetanus									1		2	1	1	1					1	·	2	1	3	3	1	4	5	2	7
	10. Hysteria																									
	11. Cataleptic																									
	ORDER 2.																									
	1. Pericarditis																									
	2. Aneurism																									
	3. Heart Disease, &c.															1									1		1	1		1
	ORDER 3.																									
	1. Laryngitis																									
	2. Bronchitis					1								1						1		2		2	1		1	3	·	3
	3. Pleurisy									·	1		1									1	·	1	1	·	1	2	2	4
	4. Pneumonia			·	1	1				·		7	·	5	1	7	3			1		2		2	·	1	1	·	1	3
	5. Asthma									7	3	7	·	5	1	7	3					12	4	16	14	3	17	26	7	33
	6. Lung Disease, &c.																									
	7. Goitre																									
	Carried over	·	·	·	1	3	·	·	1	62	20	49	17	26	11	6	8	1	·	4	·	92	31	123	69	27	96	161	58	219

Class.	Causes of Death.	Non-Asiatics.				Mixed Races.				Asiatics.										Total deaths of all ages.								
		Males.		Females.		Males.		Females.		Hindoos.				Mahomedans.				Other classes.		Males.			Females.			Total.		
		Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.	Town.	Suburbs.	Combined.
	Brought forward	-	-	-	1	3	-	-	1	62	20	49	17	26	11	16	8	1	-	4	-	92	31	123	69	27	96	161	58	219
III	LOCAL DISEASES.—(Cont.)																													
	ORDER 6.																													
	1. Cachexia																													
	2. Infantile					-	1			1	-									1	1	2				1	1	2
	3. Parturition																													
	4. Ascites																													
	5. Ulceration of Intestines																													
	6. Hernia																													
	7. Ileus																													
	8. Intussusception																													
	9. Stricture of Intestines																													
	10. Phthisis																													
	11. Stomach Disease, &c.																													
	12. Pancreas Disease, &c.																													
	13. Hepatitis																													
	14. Jaundice									·	1			·	1					·	2	2					2	2
	15. Liver Disease, &c.									4	1	5	2							4	1	5	2	2	4	6	3	9
	16. Spleen Disease, &c.									-	1	1	-	-	4	1	3			-	5	5	2	3	5	2	8	10
	17. Colic											1	-			-	1						1	1	2	1	1	2
	18. Obstruction of Bowels																													
	19. Hæmorrhoids																													
	20. Constipation																													
	ORDER 8.																													
	1. Nephritis																													
	2. Lithuria																													
	3. Bright's Disease (Nephritis)																													
	4. Diabetes																													
	5. Calculus (Stone)																													
	6. Cystitis																													
	7. Kidney Disease																													
	ORDER 9.																													
	1. Ovarian Tumour																													
	2. Uterus Disease, &c.											1	-										1	-	1	1	-	1
	ORDER 7.																													
	1. Synovitis (Arthritis)																													
	2. Joint Disease, &c.																													
	3. Rheumatism																													
	ORDER 8.																													
	1. Phlegmon																													
	2. Ulcer															-	1						-	1	1	-	1	1
	3. Skin Disease, &c.																													
	4. Tumour											2	-										2	-	2	2	-	2
IV	DEVELOPMENTAL DISEASES.																													
	ORDER 1.																													
	1. Premature Birth																													
	2. Cyanosis																													
	3. Spina Bifida																													
	4. Other Malformations																													
	5. Teething																													
	ORDER 2.																													
	1. Parturition																													
	2. Child-birth																													
	ORDER 3.																													
	1. Old age											1	-	1	1					1	1	2	1	-	1	2	1	3
	ORDER 4.																													
	1. Atrophy and Debility																													
V	VIOLENT DEATHS.																													
	ORDER 1.																													
	(Accident or negligence.)																													
	1. Fractures and Contusions											1	-	-	1					-	1	1	1	-	1	1	1	2
	2. Gunshot Wounds																													
	3. Cut-stab											1	-										1	-	1	1	-	1
	4. Burns and scalds																													
	5. Poison																													
	6. Drowning																													
	7. Suffocation																													
	8. Otherwise																													
	ORDER 2.																													
	(Homicide.)																													
	1. Murder and non-disclosure																													
	ORDER 4.																													
	(Suicide.)																													
	1. Gunshot Wounds																													
	2. Cut-stab																													
	3. Poison													1	-					1	-	1				1	-	1
	4. Drowning																													
	5. Hanging																													
	6. otherwise																													
	ORDER 8																													
	(Execution.)																													
	1. Hanging																													
	Other violent deaths (not classed)																													
	Sudden deaths (cause unascertained)	-	1							1	-	4	2	9	1	2	4	-	1				6	7	13	10	2	12	16	9	25
	Causes not specified, & ill-defined	-	1	-	1	3	1	1	1	75	25	72	20	80	23	17	15	1	-	4	-	101	50	151	94	37	131	203	87	290
	TOTAL													Ratio per 1,000 of population				per cent.				17.9	20.0	19.5	32.7	22.7	29.1	22.0	21.1	22.2
	Still-births									4	1	-	-	2	1	1	-			-	2	1	2	8	1	2	3	7	4	11

Remarks—

URBAN CALCUTTA.

(1) The total number of deaths registered during the week ending 16 July was 203 against 158 and 169 in the two preceding weeks, and [illegible] than the corresponding week of last year by 42. There were 6 deaths from cholera, against 2 and 6 in the two preceding weeks; the number is [illegible] than the average of the past quinquennium by 5. There were [illegible] deaths from small-pox during the week, against 1 in the previous week. There were 5 deaths from tetanus against 2 in the previous week. The mortality from fevers and bowel-complaints amounted to 51 and 26 respectively, against 41 and 22 in the preceding week. The general death-rate of the week was 22·6 per mille per annum, against 24·7 the mean of the last five years.

There were 12 deaths from plague against 9 and 14 in the 2 preceding weeks.

SUBURBAN CALCUTTA.

(2) The total number of deaths registered during the week ending 16 July was 87 against 85 and 107 in the two preceding weeks, and [illegible] than the corresponding week of last year by 55. There were nil deaths from cholera, against nil and 3 in the two preceding weeks; the number is [illegible] than the average of the past quinquennium by [illegible]. There were [illegible] deaths from small-pox during the week, against [illegible] in the previous week. There were 2 deaths from tetanus against 1 in the previous week. The mortality from fevers and bowel-complaints amounted to 17 and 9 respectively, against 17 and 11 in the preceding week. The general death-rate of the week was 21·1 per mille per annum, against 32·6, the mean of the last five years.

There were no deaths from plague against 1 and nil in the 2 preceding weeks.

(3) The general death-rate of the combined area is equal to 22·2

CALCUTTA:
The 22nd July 1898.

J. M. Cook, M.D., F.R.
Health Officer, Calcutta.

2054—11-7-97—2,000.

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INFANT FEEDING IN THE TROPICS.

W.E.Deeks,M.A.,M.D.

Chief of Medical Clinic, Ancon Hospital.

There are few problems more difficult for the practising physician to solve than the satisfactory artificial feeding of infants. Particularly is this true in the Tropics, where good cow's milk is not available. Unless the cattle are grain fed, the quality of the milk given is very inferior, and not suitable for infant feeding. Moreover, in warm climates bacterial growth is rapid, and before the milk can be properly taken care of it is so contaminated that its use for this purpose is a grave source of danger to the infant.

One has but to read of the difficulties encountered by the physicians in Africa, India and other tropical countries and the dreadful infant mortality, to appreciate the reasons why most white women prefer to go to a temperate climate to rear their children.

It is a well known fact also that very few women in the tropics are able to nurse their infants properly for any length of time, so that any method of artificial feeding which offers a satisfactory solution to this problem, can be thoroughly appreciated.

In Panama these conditions had to be met, as many of the employees of the Canal Commission brought their families with them, and, moreover, very many children were born here.

How this question has been solved, it will be my object now to show as evinced by the morbidity and mortality results of children admitted to Ancon Hospital between Jan. 1st., 1906, and March 1st., 1911. Not all the children requiring medical attention in the Canal Zone are admitted to Ancon Hospital, but most of them who are seriously ill are sent here, as are also many from the city of Panama.

Racial habits differ materially, and no analysis would be fair if this fact was not considered. I have, therefore, for comparative purposes, grouped the cases into four classes: Americans, Panamanians, White Foreigners and Colored. These have again been divided according to age into those under two years of age, and those over two but under four. In all there were 370 admissions under five years of age, of whom 238 were under two years. The following table shows the number of cases in the respective classes and ages above-mentioned, specifying the chief affections which we are considering, viz:- entero-colitis, and for comparative purposes, malaria. All affections except these are grouped under the head of "Other Diseases", brief reference being made below to the more important of them.

TABLE I.

		MALARIA					NUMBER OF DEATHS		
Nationality.	Age	Estivo Au.	Tert	Clinical	Ent. col	Other Dis	Malaria	Ent.-col	Other dis.
Americans	Under 2 yrs.	1	1	10	51	45			3
	Over 2 but under 5 yrs.	2		13	9	28			1
Panamanians	Under 2 yrs.	3	2	5	20	17	1	1	7
	Over 2 but under 5 yrs.	4		2	2	5	1		1
WhiteForeigners	Under 2 yrs.	3	3	9	9	10			2
	Over 2 but under 5 yrs.	17	10	15	3	8	1	1	
Colored	Under 2 yrs.	8	3	1	19	18	2	5	5
	Over 2 but under 5 yrs.	4	1	1	2	3	3	1	

Five of the deaths from malaria were due to estivo-autumnal infections. The ages and specific causes of death other than those due to malaria were as follows:

Americans. Meningitis, about 5 years; meningitis, 1 year; capillary bronchitis, 3 months; acute hydrocephalus, 7 months.

Panamanians. Meningitis, 14 months; purpura haemorrhagica, 3 months, and 8 months, 22 months, and 7 months; syphilitic meningitis, 4 months; typhoid, 2 years; broncho-pneumonia, 1 month, and unspecified, 7 months.

Spanish. Meningitis, 6 months; inanition, 9 days.

Colored. Tubercular meningitis, 5 months; sclerema and inanition, 4 months, and one day; disseminated tuberculosis, 4 months; infection undetermined, 1 year.

Among interesting cases which recovered were:- Endocarditis, in an American under 2 years; one typhoid and one lobar pneumonia in colored children under 2 years; 2 lobar pneumonias in American children between 2 and 5 years; and 1 lobar pneumonia in a Spanish child of the same age.

The total mortality from all causes was 35, or 9.3%. of these 13 occurred within 24 hours after admission and can scarsely be considered as treated cases. Eliminating these the mortality is 6.1%. An analysis of those who died from malaria shows that 4 died within 24 hours after admission; 3 were complicated with capillary bronchitis, pneumonia and nephritis respectively. One was admitted comatose, but lived for 32 hours.

Children admitted with constipation, colic and artificial feeding, are grouped with "Other Diseases." Of the cases grouped as "entero-colitis" they were diagnosed under the terms "entero-colitis," "ileo-colitis," "colitis," "dysentery," "diarrhoea," "marasmus," "malnutrition," and "bad feeding." They were all more or less grave digestive disturbances due to improper feeding. Of these 115 cases 99 were under 2 years of age. Two of these were admitted moribund and died within 24 hours

after admission. Eliminating these, we had 113 admissions, with 6 deaths, a mortality of 5.3%. Of these 6, one lived for two days, and one for four days after admission. Little can be done for infants when they are in such grave condition when admitted. Excluding these, the mortality is 3.6%.

That these results are due to proper feeding and not to climatic conditions, we have abundant evidence to show. Panama city affords us evidence on this point. I am indebted to Dr. J. C. Perry, Health Officer, of the city of Panama, for a list of the deaths among infants under one year and children from one to 4 years, inclusive, since the American Government was in charge of sanitation.

TABLE II.

MONTH.	1906.		1907.		1908.		1909.		1910.	
	Under 1 yr.	1-4 yr.	Under 1 yr.	1-4 yr.	Under 1 yr.	1-4 yr.	Under 1 yr.	1-4 yr.	Under 1 yr.	1-4 yr
January	28	1	22	3	27	7	28	8	25	7
February	10	3	15	1	14	4	24	7	24	5
March	13	1	13	1	21	4	21	7	26	5
April	19	1	17	3	23	1	23	1	57	6
May	35	11	21	4	26	3	23	1	35	12
June	16	7	35	4	43	7	34	3	45	9
July	26	10	45	10	41	7	29	9	29	7
August	18	5	20	5	28	9	21	10	24	11
September	22	2	29	2	25	5	28	1	34	11
October	15	2	25	2	31	6	27	7	46	12
November	22	2	34	10	37	7	29	6	52	15
December	23	5	40	7	32	4	25	4	57	24
Totals	247	50	316	52	348	64	312	64	454	124

Brev.

The total population for these years is estimated as follows:

1906.....25000
1907.....33000
1908.....37000
1909.....43000
1910.....45000

There are no reliable statistics of the birth rate in Panama, so that it is impossible to compare it with the death rate of children under one year of age.

If we take, however, the statistical report of the city of Providence, R. I., as compiled by Dr. Charles V. Chapin, we find that the population of 1909 was estimated at 217,000 or about five times that of the estimated population of Panama. The number of births in that year were 5,607, or one to 38.7 per capita of population. The death rate from diseases of all causes under one year of age was 756, or 13.4% of the birth-rate, and from diarrhoeal diseases alone 165, or a little less than 3%.

For the same year in Panama with one-fifth the population, the deaths under one year from all causes, numbered 312, most of which were due to diarrhoeal affections, as there was at that time no measles, scarlet fever or diptheria as contributary causes. Had the birth-rate in Panama been the same as in Providence, there would have been about 1100 births, of whom 312 died before they reached one year of age, or 28%.

The birth-rate, though, was probably higher, and it ia a safe estimate that 20% of the babies born in Panama died before they reached the age of one year, and the greater number of these from preventable causes.

The great increase in population has been through new arrivals, chiefly colored, and the deaths have been confined largely to the children of these, and of the Panamanians of the poorer class. They never use anything but the sweet condensed milks and paps made from all sorts of starchy foods when they are unable to nurse their babies. The result is entero-colitis, marasmus, inanition and death.

That these deaths are due to entero-colitis consequent on bad feeding, and not to malaria or any epidemic disease, is proven by the fact that there is no exacerbation in the curve of morbidity or mortality throughout any part of the year. It is more or less the same irrespective of season which influences other diseases.

It must be admitted that our mortality rate was extremely low, and particularly so in those cases due to entero-colitis and allied conditions. This being the case, it will now be my object to show the method of feeding which will give these results.

Although good cow's milk is available in the hospital from a herd under the direct supervision of the Hospital Superintendent, it is seldom used for infant feeding as it is not accessible for the use of children outside the hospital. Other means had therefore to be adopted which were of universal application, and the results have proven these to be perfectly satisfactory.

Dr. A. B. Herrick of this hospital initiated the method in 1906, and it has been modified as experience has taught us since, so that now we have a perfectly satisfactory and cheap method of infant feeding.

From a series of analyses that were made in the laboratory under Dr. S. T. Darling's supervision, by Mr. J. E. Jacobs, the following results on human milk were obtained:

TABLE III.

Nationality	Age of Infant	Total Solids	Fats	Lactose	Proteid	Ash.
American	?	12.15	2.20	7.09	2.68	.18
do	?	11.56	2.40	6.80	2.14	.22
"	?	10.69	1.37	7.24	1.88	.20
"	3 days	11.51	2.40	5.60	3.23	.28
"	12 days	12.20	3.20	6.20	2.71	.13
"	9 days	11.10	3.00	5.20	2.76	.14
"	9 days	11.80	3.15	5.60	2.90	.15
"	10 days	11.39	2.40	6.02	2.83	.14
"	4 mos	12.14	1.20	6.00	4.84	.10

CONT'D.

TABLE III - Cont'd.

Nationality	Age of Infant	Total Solids	Fats	Lactose	Proteid	Ash	
							W
American	6 mos.	10.91	3.20	6.30	1.34	.07	h
do	11 mos.	11.96	3.00	6.20	2.62	.14	i
"	11 mos.	8.25	1.00	6.00	4.84	.10	t
General	Average	11.22	2.33	6.25	2.48	.16	e
Panamanian	3 days	11.51	2.40	5.60	3.23	.28	
do	8 days	13.28	4.50	5.80	3.28	.20	
"	9 days	11.80	3.15	5.60	2.90	.15	C
Grenadian	9 days	11.30	3.40	5.30	2.36	.24	o
Jamaican	8 days	14.98	6.20	5.00	3.78	.18	l
do	8 days	14.38	4.40	5.80	3.97	.21	o
"	9 days	14.30	5.52	6.48	1.93	.37	r
Barbadian	8 days	12.03	3.00	5.50	3.53	.23	e
"	8 days	13.61	4.38	6.38	2.67	.18	d
"	?	11.98	3.20	7.00	1.58	.20	
?	?	13.18	4.00	5.20	3.81	.17	
General	Average	12.94	4.01	5.78	3.00	.21	

General average among American women within ten days of parturition.

11.60	2.83	6.52	2.88

From a general survey of the above table it can be seen that the average milk of the colored women is richer than that of the American women, and that the tendency of the milk of the American women is to deteriorate in quality as the infant's age increases. Moreover, it must be observed how extremely variable the quality of milk is in different mothers, and how important it is to have an analysis of the milk made if the child is not improving satisfactorily. Artificial feeding, therefore, in part or in whole becomes a necessity, and this had to be met with where fresh cow's or goat's milk was not available. Our object has been to ~~simulate~~ approximate as nearly as possible the composition of human milk.

Because of the large carbohydrate content (66 to 89 percent) in all prepared infant foods, and the practical impossibility of diluting them to approach the contents of mother's milk, we have excluded them from our diet list. We have found that if an infant can assimilate them, it became fat and inactive, with slow and difficult dentition, and suffered

from constipation or occasional attacks of diarrhoea. In warm climates this generalization can be made: That the whole group of malted milk and prepared infant foods with a high carbohydrate content is to be avoided in infant feeding.

We therefore have left to consider the condensed milks, sweetened and unsweetened. A series of brands purchased here in the open market were analysed by Mr. J. E. Jacobs, with the following results:

TABLE IV.

ANALYSES OF EIGHT SAMPLES OF CONDENSED MILK

	No. 1.	No. 2.	No. 3.	No. 4.	No. 5.	No. 6.	No. 7.	No. 8.
Total solids	74.99%	77.50%	75.42%	72.31%	31.55%	25.93%	27.03%	23.77
Fat	10.75	9.56	10.04	.39	9.60	8.40	8.40	7.20
Lactose	12.50	13.73	10.23	14.94	11.78	10.10	10.19	9.35
Proteids	7.66	11.71	9.63	8.97	8.25	5.94	7.01	6.25
Saccharose	42.50	40.80	43.63	46.00	0	0	0	0
Ash	1.58	1.70	1.89	2.01	1.92	1.49	1.43	.07

No. 1: Nestle's condensed Swiss Milk, prepared by Henri Nestle, Verey, Switzerland.

No. 2: Nestle's condensed Milk, prepared in England, Henri Nestle.

No. 3: Condensed Milk, Milkmaid brand, prepared in England, - Anglo-Swiss. Condensed Milk Company, Switzerland and London.

No. 4: Condensed Milk, Prepared in Lombardy.

No. 5: Nestle's Unsweetened Milk - Condensed.

No. 6: St. Charles Unsweetened Evaporated Milk, St. Charles' Condensing Company, St. Charles, Ill.

No. 7: Libbey's Unsweetened Sterilized Evaporated Milk. Libbey, McNeil & Libber, Chicago, Ill.

No. 8: Borden's Condensed Milk Co., New York, U. S. A.

Of 700 analyses the following average composition of cow's milk is given by Konig. Casein, 2.88%; Lactalbumin, 51% Fat, 3.68%, and Sugar, 4.90%. Considering the first two together as proteid, we have: Fat, 3.68%

Sugar,4.90%; and Proteid, 3.39%.

Cow's milk like human milk varies extremely. The race, food, period of lactation, hygienic surroundings and individual idiosyncrasies are all modifying factors. From the above analyses it can be seen that the sweet condensed milks are cow's milk reduced to from 35 to 40% of their volumes, and from 40 to 44% of cane sugar added thereto.

The Condensed milk made in Lombardy is evidently prepared from skimmed milk; as it is practically free from fats it should not be used for infant feeding. The unsweetened condensed, or concentrated milks are made from cow's milks reduced to about 40% of their volumes. If we average the composition of the three first sweetened milks, we have: Fats, 10.11%; Lactose,12.08%; Proteid,9.67%; and Saccharose, 42.31%. This will be the standard for our formulae. The average composition of the unsweetened, concentrated or evaporated milk is: Fat, 8.40%; Lactose, 10.35%; and Proteid, 6.86%. Using these analyses as our basis, the milk is modified according to age as follows, with the composition set after each formula:

TABLE V.

Sweetened Condensed	Unsweetened Condensed	Water	F a t s	Condensed Sugar	Proteid
1	3	60	.55	1.33	.47
1	3	50	.65	1.58	.56
1	3	40	.80	1.93	.68
1	3	30	1.03	2.51	.85
1	3	24	1.26	3.05	1.08
1	3	20	1.43	3.55	1.26
1	3	16	1.76	4.26	1.51
1	3	12	2.20	5.33	1.88
1	3	10	2.52	6.09	2.16
0	3	6	2.80	3.45	2.29

Human milk does not become richer as the infant grows older, but generally decreases in food value. The increasing demands of the infant are met by increased quantity, and not quality. It has been out experience here that the formula -- 1. 3.. 12. according to the above table, gives

as nearly a perfect result as possible after the first month. As cane sugar is much sweeter than milk sugar, there should be a lower percentage of it than of milk sugar.

It must not be overlooked that the unsweetened milks must be kept in a cool place after the can is opened, and carefully corked with sterile cotton. It is better, also, to purchase small cans, opening a fresh one every morning. As some of the cans are not properly sealed, always observe the milk closely on opening, for contamination or sourness.

The sweet condensed milks keep much better, but must not be exposed where flies, ants etc., have access to it.

TABLE VI.

TABLE FOR INFANT FEEDING.

Age of Infant	Formula	Amt. of ea. feeding.	Interval of feeding.	Number of feedings daily.	Diluent.
1 day	Plain boiled water two or three times.				
	X.Y.Z.				
2 days	1.3.60	1/2 to 1 oz.	2 hours	6 - 8	Boiling water
3 days	1.3.50	1/2 to 1 oz.	2 hours	8	" "
4 days	1.3.40	1 oz.	2 hours	10	" "
5 days	1.3.30	1 oz.	2 hours	10	" "
6 days	1.3.24	1-1/2 ozs.	2 hours	8 - 10	" "
7 days	1.3.20	1-1/2 ozs.	2 hours	8 - 10	" "
1-4 weeks	1.3.16	2 ozs.	2 hours	8	" "
4 wks - 3 mos	1.3.12	3 - 4 ozs.	2-1/2 hrs.	7 - 8	" "
3 mos - 6 mos	1.3.12	5 - 7 ozs.	3 hours	7	" "
6 mos - 9 mos	1.3.12	7 - 9 ozs.	3-1/2 hrs.	6	Barley water.
9 mos-12 mos.	1.3.12	8 - 10 ozs.	4 hours	5	" "

x. Sweet condensed milk.
y. Unsweetened condensed milk.
z. Boiling water or after six months of age, barley water.

To each bottle add one to two teaspoonfuls of lime water, or, if constipation is present, one teaspoonful of milk of magnesia. In preparing a bottle always add some boiling water to the measured quantity of sweet condensed milk; dissolve thoroughly; then stir in the unsweetened milk; then add sufficient boiling water to make the desired quantity; then the lime

water and a few grains of table-salt and allow to cool until the proper temperature is reached. One needs but a graduate of eight ounces or a bottle graduated to one quarter drams, and the process is simple.

It is always wise for the mother or the nurse to taste the milk before adjusting the nipple to the bottle in order to see that the preparation is not soured.

After six months barley water in whole or in part can be used as a diluent, and after nine months it or oatmeal jelly. They are made in this hospital by Miss Barr, in charge of the diet kitchen, as follows:

Barley water: One tablespoonful of pearl barley after being washed is added to one pint of cold water and soaked for 10 or 12 hours, and then strained. One quart of cold water is then added and boiled slowly for two hours. Water is added from time to time so that the end product consists of one pint. It is then seasoned with half a teasoppnful of salt, and strained through muslin or a fine sieve.

If Robinson's Patent Barley is used, Koplik recommends the following method of preparation: A heaping teaspoonful is suspended in a pint of cold water until the lumps have disappeared. The mixture is then placed in a small saucepan over a fire and stirred constantly for fifteen or twenty minutes after it begins to boil. Water is then added to make the mixture up to one pint.

Oatmeal Jelly: This is made as follows: One cup of rolled oats is soaked in two cups of cold water for ten or twelve hours, then strained through a fine sieve or muslin. One cup of cold water is now added, and the mixture is gently boiled for two hours, stirring frequently. One half teaspoonful of salt is now added, and when allowed to cool, the product jellies. Two to four teaspoonfuls of this jelly can be added to each bottle after the infant is nine months old. It is particularly valuable

if there is a tendency to constipation.

After the infant is one year old the formula, 1..3..10, can occasionally be utilized. From one year on also, an egg can be given daily, either in the milk; placed in boiling water for eight or ten minutes, when it jellies; or it can be lightly boiled and a plain cracker, rusk, or well-toasted stale bread with butter, can be given with it. Not more than five feedings should be given daily. After fifteen months, two eggs may be given daily, one in the morning and one in the afternoon and the number of feedings reduced to four. After eighteen months a light general diet can be initiated with four feedings daily, the last one of which should be plain milk, or a cereal with milk, without sugar. After eighteen months no sweet condensed milk is used, but one of the unsweetened condensed milks diluted with twice as much water. This makes a wholesome, nutritious milk food. After two years of age the child should be fed but three times daily, and absolutely nothing but water be given between meals.

Fruit juices fresh and unsweetened, preferably orange, should be administered every morning from the third month on directly after waking. At first ten or fifteen drops increasing gradually, so that at twelve months the child can be allowed the juice of one or two oranges. Green vegetables should be given after the eighteened month.

CONDITIONS ONE MEETS WITH THAT CONCERNS THE MOTHER. C9 S.

Teething. In well nourished children there are four periods when the teeth erupt, and give rise to more or less general disturbances during the first two years. They are about the sixth, eight, twelfth and eighteenth months. The time is uncertain, and may vary one or two months from the periods named. The disturbances may take the nature of constipation, vomiting, diarrhoea, convulsions, vicarious rashes, restlessness, fever, and irritability, particularly at night. The better nourished the child is, the

less likely are any of them to occur.

Vomiting. This is a common symptom. If artificially fed by the above described method, and no other cause can be attributed, use a weaker formula for a few days. Vomiting and absolute anorexia with high fever and sometimes diarrhoea, is a sympton of milk poisoning. It shows that either the milk was bad when the can was opened, or had not been carefully cared for after being opened.

Food with too much sugar and other carbohydrates in a child is directly responsible for fever, entero-colitis, eczema, eneuresis, rheumatism, recurring bronchitis, pustular dermatitis, stomatitis, and dry, lustreless hair. The so-called curds in the stool are common in artificially fed children. They are of little significance and are generally not curds, but fatty-acid soaps.

When a child is not thriving, never forget the possibility of constitutional lues. Mercury promptly gives relief.

The onset of the acute infectious diseases must always be considered, and in the tropics malaria particularly.

In this paper I have endeavored to show that tropical infant feeding is not a serious problem, and has given us here as nearly perfect results as can be obtained by any other method. The same measures can be applied in warm climates the world over. The feed is low in fat contents, but this is not as necessary a constituent of the food in warm weather as in cold. This method of feeding gives a sterile, wholesome food, which rarely ever disagrees and the large number of fine, health babies on the Isthmus bear eloquent testimony to its value.

Its cheapness should appeal to the poorest class of people. Even when purchased at retail prices in Panama, (30¢) thirty cents worth of milk

will make 160 ounces of food after the formula 1..3..12. This is at the rate of 3/16 cents an ounce, or 1-1/2¢ for a feeding of eight ounces.

In this method of infant feeding we have a sterile, cheap, perfectly satisfactory food, worthy of more extended use. That its use here has given us brilliant results has been fully demonstrated.

O.C.

Abeh

Some Notes on Filariasis in the Ibot-Ekpene District. Southern Nigeria. (50 miles from Calabar.)

Lake inmaire

During my recent tour in this district I examined the blood of 826 natives by day. & of 543 by night with the following results. —

Day blood. – Microfilaria Loa found in 12½%
Microfilaria Perstans – – 9%
Night blood. – Microfilaria Bancrofti – – 3%
Microfilaria Perstans – – 8½%

I only saw 4 cases of Elephantiasis, though it is very common further inland.

Nearly all those with Microfilaria Loa gave me a history of swellings & eye troubles. suggesting Calabar swellings & Filaria Loa in the eye. Some of these were not very reliable. but a fair number. given by rather intelligent natives. who on cross-examination appeared to be familiar with Calabar Swellings & Filaria Loa. were fairly trustworthy. I got histories of swellings & eye troubles in a number of children, though I never found the Microfilaria in any under about 13 or 14 years of age.

I examined the blood of 43 Europeans for Microfilaria I found Microfilaria Loa in one (9 years residence) & Microfilaria Perstans in another (7 years residence) Of these 43 Europeans. 19 gave me a history of Calabar Swellings & of these 8 also gave me a history of Filaria Loa in the eye.

Watching the Microfilaria Loa under the microscope I made the following observation. which I have not seen noted in any text-book on the subject. If the worm is carefully focused. with the ⅛ objective just before it ceases to move. a granular semi-fluid substance can be seen along the centre of the worm.

This substance is seen to flow slightly towards & approach the surface of the worm at one spot which is most probably the anterior V-shaped spot (see fig I) Then just as the worm ceases to move the V-shaped spot bursts & the granular contents flow out between the sheath & the worm, raising up the sheath for some little distance above & below the V-shaped spot (see fig II)

By P. J. Foran F.R.C.S.I.
M.O. Southern Nigeria

2 illustrations

Darjeeling

Some Notes on a Rare Case of Congenital Malformation.

— o —

by B. N. Ghosh L. M. S. (Cal. Univ.)
Member of the Asiatic Society of Bengal,
Lecturer on Materia Medica and Therapeutics, Calcutta Medical School and College of Physicians and Surgeons of Bengal.

— o —

Cases of congenital malformation are met with only occasionally, and they are of interest not only to the profession but to the general public as well. It is very difficult to account for this sort of defective growth — and how far our science fails to penetrate into the mysteries of Nature's laws of Growth & development?

The following notes are that of a newborn child I was called upon to see in a Mahomedan family of Calcutta, and as the case appears to be a very rare one, I take the privilege of your columns to bring it before the profession.

When I 1st saw the child I found to my surprise the case to be a curious amalgamation of defective developments in which.

The child was born in full term.

Points noted are chiefly :-

1. Talipes of the R foot. Besides there were six toes on that foot. (see Diagram)
2. Imperforate anus.
3. No external organs of generation, but only an atrophied Scrotum, without the testes in it is found in their place. (See Diagram)

4. The abdominal wall from the navel downwards in the middle line was partly deficient (See diagram) and there was a protrusion, basically the lower gut, with an opening at the end looking upwards and through which the child passed stools. This protrusion could not be separated from the wall, with which it was intimately fused. The navel coalesced with it protruding mass, the umbilical cord coming out from the top. (See diagram)

5. Urine was passed in drops from a very small opening just under the protruding mass (See diagram). But there was nothing like a urethra.

The child is otherwise healthy. Takes milk well. No other organ seems to share in the defective growth. Heart sounds normal. The child is still alive (10 Dec)

The whole protruded portion was not covered with skin but had a mucous lining. Quite soft and red.

It appears to me that the urachus had no meso-rectum developed to keep it in situ, it turned upwards and found its way through the abdominal wall which was deficient and got fixed there. The opening at the end of it which still serves the outlet for stools is looking upwards and is big enough to admit a fine pencil.

There being no external organs of generation & no urethra the neck of the bladder opened directly at the lower part of the deficient wall.

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6163

"A Comparative study of Methods of Examining Faeces for evidences of parasitism" (1)

by

Maurice C. Hall. (Med Zoology)

(abstract).

(1) U. S. Department of Agriculture, Bureau of Animal Industry (Bulletin No. 135.

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This valuable paper deals with the results of a series of tests of the various methods that have been advocated for the examination of faeces for evidence of ~~animal~~ metazoal infection. These tests were undertaken with the purpose of discovering a satisfactory routine method ~~for routine examination~~ which would be capable of general application and which in the shortest time would most surely give indication of an even slight infection in ~~any kind of~~ faeces of any consistence and character

Examinations of faeces are of two kinds (a) naked eye inspection for the adult parasites – whole or in fragments. and (b) microscopical investigation for eggs and embryos. The latter is the more important in ordinary work and most influenced by the technique employed.

Hall enumerates the following known methods of microscopical examination :– Amongst others

a. Smear method. This is the method most commonly in use at the present time. A small particle of the suspected stool is picked up with a stick or stirring rod, smeared on a glass slide in a drop

(1)

of water or salt solution, covered with a cover-glass and examined under the microscope. Hall considers that it is satisfactory provided about ten slide preparations are examined.

(b) The Sedimentation method consists in allowing the faeces if sufficiently fluid, to settle, if they are too solid they are shaken up with a quantity of water and then allowed to stand for a little. The supernatant fluid is decanted as long as any matter will float and the sediment is finally examined.

(c) Burette Method. — This is merely the above varied by taking off the sediment from the bottom through a stop-cock.

(d) Centrifuge Methods. — The centrifuge gives a more rapid and certain concentration of material but opinion does not seem to favour its use in faecal examinations.

(e) Sieve methods — These consist in the use of one or more sieves to screen out course particles of food. Stiles says "it is sometimes convenient to pour the entire mass through a sieve rejecting the portion left in the sieve; or to wash the faeces in a sieve holding the latter under water. As a rule, however, the sieve is not very useful in faecal examinations.

(f) Filter method. — Gastaiger reverses the above method for certain purpose. In his search in the stalls of cattle for eggs

of ascaris he soaks manure, straw etc in water; filters this through a filter and examines the residue for eggs.

(g) Bass's Saline solution Method.

~~According to Bass~~ If a quantity of infected faeces ~~he~~ be put into a nine-tenths saturated solution of common salt and shaken well all hook-worm eggs will rise to the surface when the fluid has been allowed ~~on allowing the fluid~~ to stand ~~and~~ A single drop to be from the surface ~~of the fluid~~ may contain large numbers of ova. The method depends upon the fact that the specific gravity of the egg is lower than that of the solution.

(h) Bass's Calcium Chloride ~~Method~~ Centrifuge method.

a quantity of faeces is mixed with 1 to 10 of water and centrifugalised. The fluid is poured off and more of the diluted faeces added. The process is repeated so that the sediment is rewashed several times until all matter that can be is removed in this manner. a solution of Calcium chloride of specific gravity 1·05 is now used in place of water. This carries off everything having a lower specific gravity. a stronger solution, one of 1·25 sp. gr. is now used and the eggs rise to the top. A few drops from the surface may be examined or, ~~better~~, diluted first with water to lower the sp. Gravity to below 1·05. ~~When~~ In the centrifugalised sediment should contain most of the eggs in the original quantity of faeces.

(3)

(i) Garrison's Calcium Chloride Sedimentation Method.

The stool is repeatedly washed and sedimented for such number of times as is necessary to give a clear supernatant fluid and the supernatant fluid decanted. There then remains a heavy sediment ~~containing the eggs.~~ By the specific gravity of which is much higher than that of the eggs it contains. By mixing this sediment with a solution of Calcium Chloride containing 350 grams to the litre, which ~~gives~~ has a specific gravity of about 1·2., the eggs float and the rest of the sediment sinks. The top layer can then be decanted off and examined microscopically.

(j) Pepper's adhesion method for ankylostomes.

Pepper takes advantage of a stickiness that is the property of the eggs of ankylostomes. Washed and sedimented faeces are put on a slide for a few minutes and then gently immersed in water; after everything else has been washed away the eggs are still found adhering to the slide. By repeating the process numerous eggs may be collected on the same slide. This method does not apply for the eggs of Ascaris, Trichocephalus or Taenia.

(K) Telemann's Chemical Method.

By shaking up small portions of faeces in a mixture of equal parts of pure HCl and of Ether, filtering of the larger particles and centrifugalising the filtrate three layers will be found in the

fluid. viz an upper layer of dissolved fats in Ether. a middle layer of bacteria small particles in acid and a sediment of cellulose, muscular fibres etc in which the eggs are easily to be found owing to the greater degree of concentration. Pfeiffer commends this method especially for Bilharzia eggs.

As a result of a prolonged practical study of the above and other methods Hall concludes that the attainment of successful results depends mainly upon proper concentration & that this is accomplished by careful communition of the faeces, the use of sieves, sedimentation, centrifuging, and washing in water. He has elaborated a new method the description of which is quoted here in detail as the subject is one which deserves attention on account of its practical value to all workers in tropical medicine.

Hall's Method.

To printer — copy from printed matter (pages 18 to 22) appended

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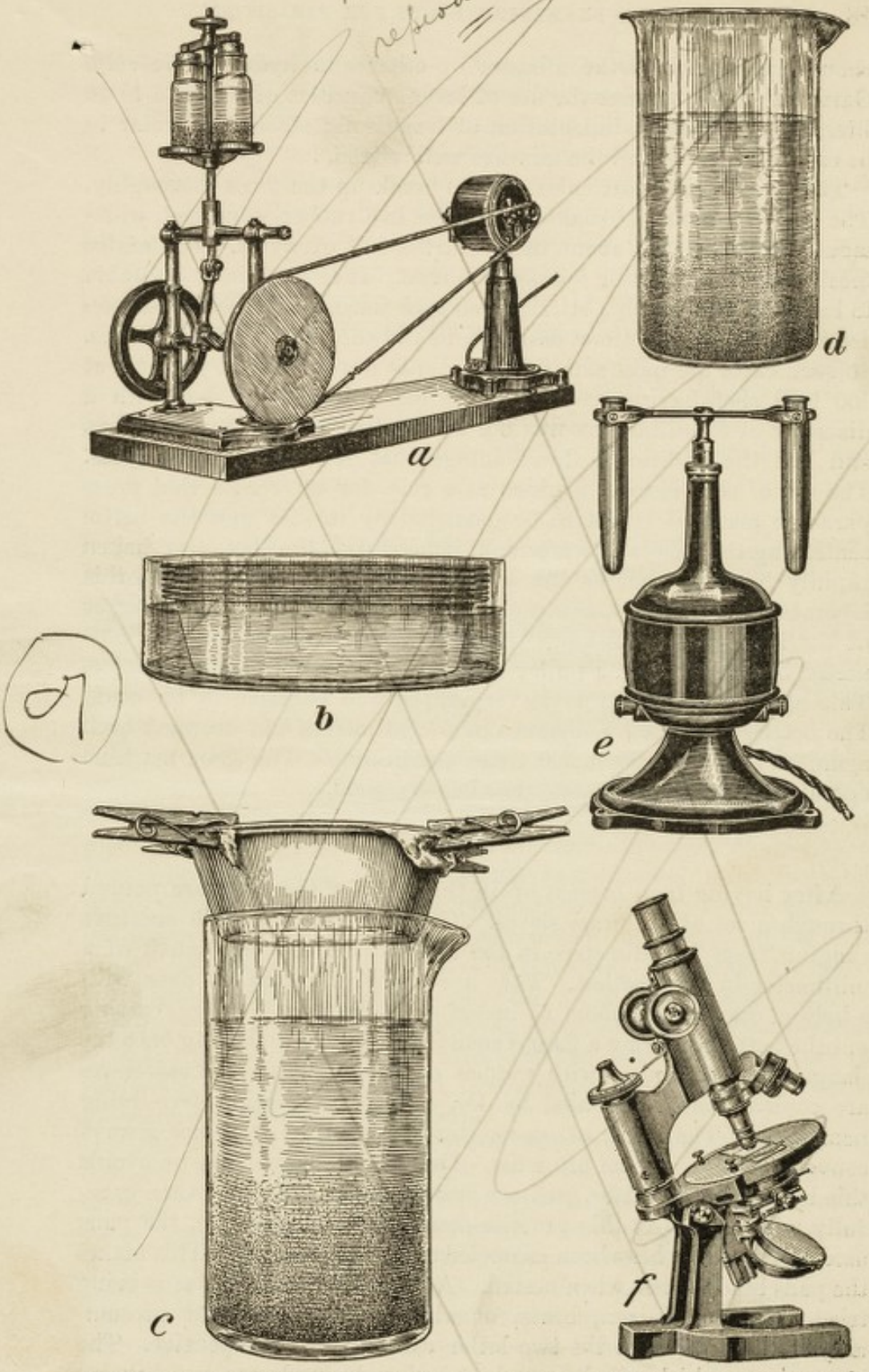

FIG. 1.—Apparatus used in the writer's method of examining feces: *a*, Shaker; *b*, brass sieves and dish; *c*, silk sieve and jar; *d*, beaker; *e*, centrifuge; *f*, microscope.

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should be shaken in the mixture of ether and hydrochloric acid. Garrison (1910) advises the use of large quantities of water, 5 to 10 liters, for the first sedimentation of large solid stools, the water to be run in violently and the mixture well stirred.

The writer takes particular care to break up the feces thoroughly. The method consists in shaking the feces in a rubber-stoppered, wide-mouthed glass bottle about three-fourths full of water. The entire fecal sample, up to 4 or 5 ounces, is used. It is sometimes desirable to break or crush with a stirring rod such hard fecal masses as sheep feces. It is also sometimes desirable to add shot to hard fecal masses. In such cases the most satisfactory results were obtained with about 100 lead shot having a diameter of 3.8 millimeters; shot with a diameter of 8 millimeters was not as effective in breaking up feces, and had the additional disadvantage that it blackened the glass. The use of shot is to be avoided as a rule, for the reason that gross parasitic material is apt to be damaged by it. At first the bottle containing the feces and water and, if necessary, the shot, was shaken rapidly by hand, but as the amount of fecal examination in the laboratory warranted the use of a machine for this work, a shaker of the kind used in mixing "milk shakes" was installed and connected by belting with a pulley wheel fitted on an electric-fan motor. This apparatus (fig. 1, *a*) is very rapid and effective in its work. The bottles are lifted a distance of 5 centimeters and dropped back again at a rate of about 500 times per minute. The same machine operated by hand would doubtless be very good.

SIEVING.

After having been broken up in this manner the feces are poured through a set of six brass sieves. The sieves have a mesh aperture ranging from 3 millimeters in the largest to about one-fourth of a millimeter in the smallest. They are made by taking tin pans with a bottom diameter of about 6½ inches and sides 2 inches high, cutting out the bottom, leaving a flange near the sides, and soldering onto the flange brass screening with meshes of various sizes. These sieves are copied from a set used by Dr. Cobb in collecting free-living nematodes. The pans, of course, tend to rust, as it is not always convenient to dry them after use. Dr. Cobb tells me that he avoids this by the use of oil or grease warmed on the pans and then carefully wiped off. As this practice seems inconvenient also, the pans used by the writer have been enameled to prevent rusting. This makes the pans bind a little when nested. A set coated with shellac is being tried. Galvanized-iron, brass, or aluminum pans would presumably be better, though the two latter would be more expensive. The brass sieves which can be purchased already made are not beveled

and hence can not be nested. They can be superimposed, one on another, but the result is a stack which is so high that it does not permit of six of them being set in a shallow dish of water with the water standing above the screen in the upper sieve.

The sieves are nested in the order of mesh aperture, with the coarsest on top, and placed in a large porcelain evaporating dish, or a large glass crystallization dish (fig. 1, *b*). The fæces are poured into the top screen and pass through the screens to the evaporating dish, particles of different sizes being held by the different screens. The use of fewer screens would not be a gain, as too coarse material poured on a screen clogs it. Tap water or normal salt solution is poured in the upper sieve until the water stands in the evaporating or crystallization dish at a level above that of the bottom of the upper sieve. This sieve is lifted and shaken a little until the fine matter has passed through. It is then lifted out and put in a large crystallization dish half full of water or salt solution and the matter it contained examined on the screen or washed into the dish. Gross parasitic material is picked out, the screen rinsed, the dish emptied and refilled if the amount of discoloration or trash present warrants it, and the process is repeated with the remaining sieves. The gross material left on the screens is thoroughly cleaned, and the likelihood of wasting time examining citrus pulp vesicles, vegetable fibers, etc., as possible parasites is reduced to a minimum.

The sediment left in the evaporating dish after removing the finest sieve is poured onto a screen of miller's silk bolting cloth with a mesh aperture of 0.117 to 0.134 millimeter and the finer particles washed through into a tall jar (fig. 1, *c*). The mesh aperture of this sieve diminishes as the cloth shrinks with use, and in a cloth which has been in use for several months it has diminished to a size of 0.070 to 0.080 millimeter. Such a mesh is too fine to permit the passage of the eggs of such important species as *Fasciola hepatica*. Some No. 120 mesh brass screen has recently been obtained which has a mesh aperture rated at 0.117 millimeter, but varying from 0.103 to 0.120, according to the writer's measurements. This promises to be a very satisfactory substitute for the bolting cloth. Presumably it will retain its mesh aperture, and will be more durable.

When the shot are used, they are poured with the fæces into the coarsest of the brass sieves. They are subsequently poured from the sieve into a petri dish with whatever coarse fecal matter may accompany them and the fecal matter easily removed by a stream of tap water. It might be supposed that fecal matter of some sort would stick to the shot, but it does not do so. Parasitic material that might adhere can be destroyed by dry heat. Lead shot have the advantage over steel shot that they may be kept in a formol solution without rusting, while steel shot would need careful drying, or else keeping in oil.

7 PTO

Where small amounts of feces are used, two pieces of brass tubing, 2 inches in diameter and 1¼ inches high, beveled to fit one in the other, and the lower one furnished with three projections to hold it on glassware of not over 3 inches in diameter, are used to hold the bolting cloth of the silk sieve, the cloth being caught and held taut by the beveled surfaces. Where large amounts of feces are used, and large amounts—not to exceed 4 or 5 ounces—should be used whenever obtainable, two enameled tin pans, with a bottom diameter of 4½ inches and with the bottom cut out so as to leave a narrow flange, are used in place of the brass tubing. The cloth is held between the upper flanges of the two tins, and the cloth and flanges held together by four small clothespins of the sort provided with a wire spring to hold the jaws together. This device I also owe to Dr. Cobb. The sieve formed of the tins and the bolting cloth is the right size for use with a jar 10 inches high and 5 inches in diameter (~~see fig. 1, c~~). When necessary a soft brush is used to brush the feces through the fine brass sieves or the silk cloth. Cobb (~~1904), as previously noted~~, uses a brush for the same purpose. These sieves will work better if water is poured through them or if they are dipped in water before the fecal matter is poured on.

The sieves, as well as all other apparatus coming in contact with the fecal material, are washed promptly with boiling water, the sieves being also scrubbed with a stiff brush. This prevents any parasitic material from remaining to contaminate a subsequent fecal specimen, and thereby giving inaccurate findings. A microscopic examination of the silk sieve shows that it washes clean very readily, and when rinsed retains very little of the material poured on it. ~~Experiment shows that eggs pass through this cloth very readily, less than half of 1 per cent of even such large eggs as *Toxocara* remaining when the fecal matter is first brushed through~~. A smear made from the residue on the bolting-cloth sieve showed 4 eggs in one case where the slide preparation from the centrifuged sediment showed 860 eggs; another smear from the residue on the bolting cloth showed 1 egg where the slide from the sediment showed 475 eggs. In the latter case, a fair estimate of the amount of material on the cloth and the amount from which the smear was made indicates that there were probably not more than 10 eggs left on the cloth, while thousands had passed through it. Those that are left are held by the jelly-like residue obtained at this point, and not because the screen mesh is defective or too small. The writer is not aware of any parasite eggs which are too large in their smaller diameter to pass through a mesh with a diameter of 0.117 to 0.134 millimeter. The number remaining after clean water has been poured through the cloth into the jar is entirely negligible.

In working with human feces, or where dangerous infection may be present, the silk cloth may be kept in a jar of formol solution

when not in use. In the course of a large number of experiments nothing has yet indicated that parasitic material from one examination has remained to subsequently contaminate other fecal specimens. Parasites that might be suspected of remaining after the cloth had been washed in boiling water might be destroyed by prolonged boiling or subjection to dry heat—experiment shows that eggs so treated are distorted or characterized by the formation of air spaces or oily areas—or fresh pieces of cloth could be used each time. ~~This last, however, would be somewhat expensive, as this cloth retails in Washington in half-yard widths at about $5 a yard. It would be cheaper to use the No. 120 mesh brass screen. This costs $1.85 a square foot, but would be permanent~~.

SEDIMENTING AND CENTRIFUGING.

The feces which pass through the silk sieve are sedimented with plenty of water in the jar. After decanting, the sediment is transferred to a beaker (~~fig. 1, *d*~~) and may now be washed if desired. The entire sediment, or as much as seems desirable, is then centrifuged (~~see fig. 1, *e*~~), repeated centrifuging with the addition of fresh material adding to the total centrifuge sediment, and may be washed at this point also, as advised by Pepper (1908) and Bass (1909). The writer sometimes washes the material at both points, the second supplementing and completing the first. It is usually sufficient to wash the sediment in the centrifuge. Bass has called attention to the important fact that a centrifuge should only be run the minimum time necessary to bring down the eggs. This time will vary with different centrifuges. With a centrifuge running 3,500 revolutions per minute Bass allows 4 to 10 seconds. I find this enough time with a centrifuge running 1,230 revolutions per minutes. After the material in the two centrifuge tubes is washed in water, one tube is left alone; the water is poured off the other and calcium chloride solution, with a specific gravity of 1.250, is added to the sediment. After centrifuging, a slide preparation is sometimes made from this tube direct. In most cases the top cubic centimeter is pipetted off, shaken up with 14 cubic centimeters of water, and centrifuged. This is the more satisfactory and certain method.

PREPARATION OF SLIDES.

By means of a long pipette, a drop of sediment is drawn up from the bottom of the tube in which water alone is used, placed on a slide under a cover glass, and examined with a microscope (~~fig. 1, *f*~~). A second slide is made from the other tube. This second slide is either made directly from a drop taken from the surface of the calcium chloride solution, or from the bottom in case the top cubic centimeter

has been added to water and centrifuged. The second slide is used as a check on the first. It sometimes has fewer eggs, especially when pipetted direct from the top, but it is a cleaner preparation, is easily examined, will sometimes have more eggs, especially if made from the sediment where the top cubic centimeter of the 1.250 solution has been centrifuged with the addition of water, and occasionally throws additional light on the material under examination. The pipettes are rinsed thoroughly, and when dried are heated in a Bunsen flame for a short time to destroy any eggs that might adhere, thus preventing contamination in subsequent examinations."

CONCENTRATION OBTAINED BY THE USE OF SIEVES.

In examining the feces of 35 sheep, the entire amount of feces passing through the sieve was centrifuged in order to give a uniform comparative study and to determine the amount of concentration attained by the use of sieves, and due to them alone. To eliminate other factors, the sediment was not washed and the centrifuge was run for long periods till everything had come down. Centrifuging the entire amount of feces necessitated the repeated filling of the tubes of a two or four arm centrifuge. A comparative examination of slides made from the sediment obtained by centrifuging a single tube full of the material, with slides made from the total sediment, showed that the concentration was the same in both cases, a result which would be expected from a theoretical standpoint. While the concentration is the same, the total amount of parasitic material present is, of course, much less in the single tube.

Using moist fecal pellets, the concentration obtained was 4:1. The concentration varies with animals of other species, with food habits, and with the condition of the particular fecal specimen examined. At the same time, the concentration is always sufficient to warrant the use of the sieves. The microscopic field obtained after treatment of feces in this way is very much more satisfactory than the field obtained in the smear method, and where the same number of slides are examined the likelihood of finding evidences of existing parasitism is certainly more than four times greater in cases where the feces have been subjected to thorough sieving.

SUMMARY OF METHOD.

The writer's method is, then, merely a modification of existing methods, and might be termed a comminution-sieving-sedimentation-centrifuge method in which water alone is depended on as a medium for these operations, a slide made after centrifuging in a calcium chlorid solution with a specific gravity of 1.250 being regarded principally as a check on the method as given.

adaptability of Hall's method.

The writer does not claim that the method advocated here is the best possible method. It is however the method which his experience shows to be the best for routine examinations of various kinds of faeces after comparative tests with other methods. It serves very well for the faeces of man and of the carnivora, herbivora and birds. ~~so far~~ It is not only of service in examining for worm parasites but also for coccidea (It has not been tested for other protozoa). ~~Presumably the comminution method would damage flagellates, ciliates or amoebae.~~"

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WEIGHTS of ORGANS ascertained in the Course of POST MORTEM EXAMINATIONS on AFRICANS, BRITISH EAST AFRICA.

By J. A Harcus [illegible] C.M.G. M.D. Medical Officer of Health at Mombasa

Tribe	Disease	Brain	Heart	R. Lung	L. Lung	Liver	Spleen	R.Kidney	L.Kidney
Mkikuyu, Ad. M.	Typhoid	2.15oz	9¼oz.	14½oz.	14oz.	2.15½oz	7¾oz.	4½oz.	4¼oz.
Mkikuyu, Ad. M.	Dysentery	2.14½oz.	9½oz.	9½oz.	7¾oz.	2.7oz.	3oz.	3¼oz.	3¼oz.
Mkavirondo, Ad. M.	Trypanosomiasis	2.9½oz	9½oz.	1.10½oz.	1.14¼oz.	1.14½oz	11½oz.	3½oz	3¼oz.
Mkikuyu, Ad. M.	Malaria	3.2oz.	11½oz.	15½oz	1.8oz	3.4½oz	7½oz	4¼oz	4½oz
Mkavirondo, Ad. M.	Cerebro-spinal meningitis	3.10½oz	12oz.	1.9oz.	12½oz	4.12oz.	14½oz	4¼oz	4½oz
MSwahili, Ad. M.	Septicaemia	2.8½oz	8½oz	1.1oz	10oz.	2.12½oz	10oz.	4½oz	4¼oz.
Mkamba, Ad. M.	Cerebro-spinal meningitis	3.1oz.	10½oz.	11½oz	9oz.	3.5oz.	13¼oz	4oz.	5oz.
MSwahili, Ad. M.	Bronchitis	3.4½oz	13oz	2.4oz	1.9½oz	4.4½oz	13½oz	5oz.	5½oz
Mkamba, Ad. M.	Pneumonia	2.12 oz	9¾oz.	1.12 oz.	1. 9 oz	2.15 oz	1. 2¾oz.	5 oz.	5 oz.
Mkikuyu, Ad. M.	Dysentery	2.15½oz	7½oz.	12 oz.	10 oz.	2. 5½oz	2½oz.	3½oz	4¼oz.
Somali, Ad. M.	Com.Fract.Skull	3. 4¼oz	6¾oz.	1. 7½oz.	1. 1¾oz.	2.13½oz	7¾oz.	6 oz.	4¾oz.
Mchaga, Ad. M.	Pneumonia	2.12½oz	10 oz.	1. 3½oz.	2. 2¾oz.	3. 9¾oz.	1. 1¾oz.	4 oz.	4¾oz.
Mkamba, Young Ad.M.	Plague	3. 1¼oz.	7½oz.	11 oz.	10½oz.	3.10½oz.	14½oz	4 oz.	4¾oz
Mkikuyu, Ad. M.	Malaria	2.12½oz	9½oz	8 oz	7 oz.	3. ¾oz	1. 8 oz.	3½oz.	4 oz.
Mkamba, Ad. M.	Pneumonia	2.10½oz.	9½oz	9 oz	15 oz	3.12½oz	1. 7½oz.	4½oz	5 oz
Mkamba, Ad. M.	Pneumonia	2.10½oz	8½oz	13½oz	9¾oz	3. 8½oz	10½oz.	4½oz.	5 oz
Nandi, Ad. F.	Dysentery	-	6¾oz	9¾oz	7 oz	4.10 oz	6 oz	5 oz	5 oz.
Mchaga, Boy	Phthisis	2.15 oz.	6¼oz	12½oz	1. -	2.15¾oz	5½oz	4¼oz	3¾oz.
MSwahili, Ad.M.	Burns	2. 6½oz	8½oz	11¾oz	13½oz	2. 7½oz	4 oz	3½oz	3¾oz
Hybrid Child	Plague	2. 8½oz	4¼oz	6¾oz	5½oz	1. 8½oz	6¼oz	3 oz	3 oz
Mkikuyu Boy	Plague	2.14¾oz	8¾oz	10½oz	8¾oz	2.11¾oz	4 oz	3¾oz	3½oz
Mnyamwezi, Ad. M.	Lympho-Sarcoma	3.- ¼oz	8¾oz	2. 4¼oz	1. 6¼oz	3. 5¼oz	2.13 oz	6¼oz	5 oz
Mkavirondo Boy	Septicaemia	2. 9¾oz	6¼oz	8¼oz	7¼oz	2.12¾oz	12¼oz.	4¾oz	4½oz
Mkamba, Adolescent male.	Pleuro Pneumonia	2.11¼oz	8½oz	1. 8¼oz	1.15¼oz	3. 9½oz	13½oz	4¾oz	5½oz
Mkikuyu Boy	Meningitis	2.15½oz	6¾oz	7¼oz	3¾oz	1. 8¼oz	6½oz	2¼oz	2½oz
MSwahili, Ad. M.	Septicaemia	2.15¾oz	9½oz	1. -¾oz	14½oz	2. 5.oz	1. 2¾oz	6¼oz	8 oz
Mkikuyu, Ad.M.	Pneumonia	3. 1¾oz	10¼oz	1. 6¼oz	15½oz	4. 3½oz	1.10½oz	4¾oz	3¾oz
Mkikuyu, Ad.M.	Abscess of Liver	2. 9¾oz	7¾oz	12¾oz	10¾oz	5. 3¾oz	5½oz	3¾oz	4¼oz
Lumbwa, Ad. F.	Dysentery	2.6¾oz	7½oz	10¼oz	9½oz	2.14½oz	9 oz.	3½oz	4 oz
Mkamba, Ad. M.	Dysentery	2. 9¾oz.	7¼oz	10 oz	7¾oz	3. 1¼oz	1. 2½oz	4 oz.	5 oz.
MSwahili, Ad. M.	Pneumonia secondary to Liver abscess	2. 8¼oz	8 oz	1.12½oz	13¾oz.	2.13½oz	5 oz.	3¾oz.	3¾oz.
Mkikuyu, Ad. M.	Malaria	2.13¾oz	8½oz	8¾oz	7¾oz	3. 1¼oz	10¾oz.	3¼oz.	3½oz
Mkikuyu, Ad. M.	Malaria	2.12¾oz.	10 oz.	12½oz	1. 3½oz	3.15½oz	14¾oz	3¾oz.	3¾oz
Mkikuyu, Ad. M.	Gunshot wound	2.15½oz	11¾oz	1. 1¾oz	14½oz	2.10½oz	3½oz	3¾oz	3¾oz
Mkamba, Ad. M.	Pneumonia	2. 8½oz	12 oz	2.11 oz	1. 1¾oz	3. 5¾oz	1. 8¾oz	4¾oz	4½oz

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Tribe	Disease	Brain	Heart	R.Lung	L.Lung	Liver	Spleen	Right Kidney	Left Kidney
Masai, Ad. F.	Dysentery	2. 4½oz	3¾oz	7 oz	7 oz	1.13 oz	3½oz	3 oz	3½oz
Mkamba, Ad. M.	Pneumonia	2. 5¾oz	9¾oz	4. 8¾oz	12 oz	3.15 oz	13¾oz	6½oz	7¾oz
Mkamba, Ad. M.	Pneumonia	2.13 oz	7¾oz	2. 2½oz	1. 2¾oz	4 lbs	1. 1½oz	5½oz	5½oz
Mkavirondo Boy.	(Provisional) Gastro enteritis	2. 4½oz	4½oz	6½oz	5½oz	2. 6½oz	5 oz	3¾oz	3 oz
Mkikuyu, Ad. M.	Pneumonia	2. 7¾oz	11 oz	2. 1 oz	1. 1½oz	3. 5½oz	8½oz	4½oz	4 oz
Mkamba, Ad. M.	Pleurisy	3. 1 oz	10½oz	1.- ¾oz	1.13½oz	3. 9½oz	9½oz	5¼oz	6 oz
Mkamba, Ad. M.	Intestinal Obstruction.	2. 6¾oz	7½oz	11 oz	8½oz	2. 9½oz	7 oz	4 oz	4 oz
Mnyamwezi, Ad. M.	Pneumonia	2.10¾oz	1. 3 oz	2 lbs	9¾oz	3.14¾oz	10 oz	7 oz	6½oz
Mkavirondo, Ad. M.	Effects of cold	2.15 oz	6½oz	11½oz	9¾oz	2.10¾oz	6 oz	3¾oz	3¾oz
Mkikuyu, Ad. M.	Pleurisy	2.13½oz	13½oz	2. 3 oz	1.14 oz	4. 6½oz	1. 1½oz	5½oz	5½oz
Mkamba, Ad. M.	Pneumonia	2.14 oz	12½oz	1. 3½oz	2.12½ oz	4. 3 oz	1. 9½oz	5½oz	6¾oz
Mkikuyu Boy.	Spirillum fever	2.10¾oz	6¾oz	7 oz	6 oz	3.12½oz	6½oz	3¾oz	3½oz
Mkikuyu, Ad. M.	Pneumonia	3. 3½oz	11 oz	2.11 oz	1.10 oz	3.13 oz	13½oz	4½oz	4¾oz
Mkikuyu, Ad. M.	Quartan fever	2.11½oz	7½oz	1. 1 oz	13½oz	2.13 oz	12½oz	3¾oz	3¾oz
Embu, Adolescent Male	Malaria	2.11½oz	6¾oz	11 oz	1. 1½oz	2.12¾oz	11 oz	3 oz	3½oz
Mkikuyu, Ad. M.	Malaria	2.12 oz	7½oz	14½oz	7¾oz	2. 9 oz	11½oz	3½oz	3¾oz
Mkikuyu, Ad. F.	Malaria	2.12½oz	10½oz	14oz	1 lb.	3. 1 oz	8½oz	7½oz	5 oz
Mkikuyu, Ad. M.	Rupture of Spleen	3. 2½oz	9½oz	9¾oz	7¾oz	2.10¾oz	6¾oz	3 oz	3½oz
Mkavirondo, Ad. M.	Pneumonia	2. 8 oz	12 oz	2.12½oz	2 lbs	5. 6½oz	11½oz	5 oz	5½oz
Mkikuyu, Ad. M.	Abscess	2. 9¾oz	7½oz	9½oz	9 oz	2. 7 oz	5½oz	3½oz	3 oz
Mkikuyu, Ad. M.	Pneumonia	3. 6 oz	6¾oz	1.15½oz	14½oz	2. 7¾oz	4 oz	4 oz	3¾oz
Embu, Ad. M.	Diarrhoea	2.11½oz	6½oz	14¾oz	6¾oz	1.13¾oz	¾oz	3 oz	2¾oz
Mkikuyu, Ad. M.	Malaria	3. 2½oz	8 oz	1.- ½oz	11¾oz	3. 2½oz	12 oz	4 oz	4 oz
Mkavirondo, Ad. M.	Pleurisy	3 lbs	10¾oz	1.14½oz	15½oz	3.11½oz	5¾oz	5¾oz	5 oz
Mkikuyu, Ad. M.	Pneumonia	3. 1½oz	9 oz	2. 9 oz	2. 3 oz	3. 9¾oz	4 oz	5 oz	5 oz
Mchaga, Adolescent Male	Tuberculosis	2.12 oz	9½oz	2.- ½oz	1. 4½oz	2.15½oz	7 oz	4 oz	4½oz
Mkikuyu, male child	Broncho Pneumonia	1.13½oz	1¾oz	3½oz	2¾oz	6 oz	¾oz	¾oz	¾oz
Mkikuyu, Ad. M.	Pneumonia	2.9 oz	6¾oz	1. 9 oz	12½oz	3. 5 oz	1. 7 oz	4 oz	4 oz
Embu, Ad. M.	Pneumonia	2.11½oz	10½oz	1. 6¾oz	15 oz	3. 1 oz	10¾oz	4½oz	4¾oz
Mkamba, Ad. M.	Pneumonia	3. 1½oz	10 oz	2. 2 oz	1. 3 oz	4.- ½oz	1. 3½oz	5½oz	5½oz
Mkikuyu, Ad. M.	Pneumonia	2.12¾oz	5¾oz	14½oz	3.- ¾oz	3. 8½oz	10½oz	5½oz	4¾oz
Mkikuyu, Ad. M.	Pneumonia	2.11½oz	12½oz	11½oz	3. 4½oz	4 lbs	1. 8 oz	5 oz	5½oz
Mkikuyu, Ad. M.	Colitis	2. 7½oz	6¾oz	1. 2 oz	8 oz	2. 7½oz	3 oz	4½oz	4 oz
Mkikuyu, Ad. M.	Pneumonia	2. 7¾oz	8¾oz	2. 2½oz	12½oz	2.15½oz	1. 4 oz	3¾oz	3½oz
Mkikuyu, Ad. M.	Pneumonia	2. 3¾oz	6 oz	13 oz	6¾oz	2. 4½oz	3 oz	2¾oz	3¾oz
Mbere, Ad. M.	Pneumonia	2.12½oz	11½oz	1.14½oz	4..1 oz	3.11 oz	1. 3 oz	5½oz	5¾oz
Mganda, Ad. M.	Pneumonia (secondary to malaria)	2.15 oz	10½oz	1.12 oz	1. 7¾oz	3.15 oz	5½oz	4¾oz	4½oz

Tribe	Disease	Brain	Heart	R.Lung	L.Lung	Liver	Spleen	Right Kidney	Left Kidney	
Mkamba, Ad. M.	Meningitis	2.13 oz	13½oz	1. 2½oz	14½oz	4. 4 oz	1. 9½oz	5½oz	6 oz	
Masai, Ad.M.	Syncope following abdominal injuries	2.11½oz	9½oz	11½oz	9½oz	3. 5½oz	13 oz	4½oz	5 oz	
Mkikuyu, Ad.M.	Shock following injuries.	2.11½oz	8 oz	7½oz	6½oz	2. 3 oz	4 oz	3 oz	3½oz	
Mkerewe, Ad.M.	Dysentery	2. 9½oz	6½oz	12½oz	9½oz	2. 6½oz	3½oz	4½oz	4½oz	
MSwahili, Ad.M.	Typhoid	2.12 oz	8½oz	10½oz	8½oz	3. 2 oz	8½oz	4½oz	4½oz	
Mnyamwezi, Ad.M.	Cirrhosis of liver	3. 3oz	8½oz	14 oz	15½oz	2.12½oz	9½oz	4½oz	5½oz	
Mkamba, Ad. M.	Peritonitis	3. 1 oz	9½oz	10½oz	12 oz	2.12 oz	5½oz	4½oz	4½oz	
Mkikuyu, Ad. M.	Typhoid	2.13½oz	5½oz	12 oz	10 oz	2. 8 oz	14 oz	4 oz	4 oz	
Mkamba Boy	Pneumonia	2.10½oz	6 oz	12½oz	12½oz	2.11 oz	13½oz	4½oz	4½oz	
Mkikuyu, Ad. M.	Pneumonia	2.13½oz	10½oz	2.15½oz	15½oz	2.12½oz	13½oz	5½oz	5½oz	
Mnyamwezi, Ad.M.	Dysentery	2.15½oz	10½oz	10½oz	8½oz	3. 6½oz	9½oz	6 oz	6 oz	
Mkamba, Ad. M.	Pneumonia	2. 9½oz	7½oz	14½oz	1. 1 oz	2. 6½oz	13½oz	4½oz	4½oz	
Mkamba, Ad. M.	Meningitis	3. 8½oz	10½oz	1. ½oz	15½oz	2.11 oz	7½oz	4½oz	4½oz	
Mkikuyu, Ad. M.	Pneumonia	3. 3½oz	10½oz	3. 2½oz	3. 5½oz	3. 5 oz	13 oz	4½oz	5½oz	
Mkikuyu, Ad. M.	Dysentery	2. 8½oz	7 oz	8½oz	7½oz	2. 8 oz	11 oz	4½oz	4½oz	
Mkikuyu, Ad. M.	Pleuro Pneumonia	3. 2 oz	12½oz	1. 8½oz	3 lbs	5.11½oz	1.11 oz	6½oz	6½oz	
Mkamba, Ad.M.	Pneumonia	3 lbs	9½oz	1. 8½oz	1. 9 oz	4.14½oz	15½oz	6½oz	5½oz	
Mkamba, Ad. M.	Pleurisy	3. 2 oz	11 oz	1. 3½oz	1. 6½oz	5. 1 oz	1. 4 oz	6½oz	5½oz	
Mkikuyu, Ad. F.	Burns	2.14½oz	12½oz	12½oz	12½oz	4. 6½oz	5½oz	6½oz	4 oz	
Mkamba, Ad. M.	Pleurisy	2.12½oz	9½oz	1. 8 oz	1.- ½oz	3.13½oz	13½oz	10½oz	----	Haemorrhagic Kidney with clot within
Mkamba, Ad. M.	Phthisis	2. 5½oz	5½oz	1.12½oz	1. 8½oz	4½oz	2. 1½oz	4½oz	4 oz	
Mkamba, Ad. M.	Pleurisy	2.10½oz	8½oz	1. 1 oz	7½oz	3. 6½oz	1. 8½oz	4½oz	4½oz	
Mkamba, Ad. M.	Pneumonia	3.- ½oz	12½oz	2. 4½oz	1.11½oz	3. 7 oz	1. 7½oz	7½oz	5½oz	
Mkikuyu, Ad. M.	Malaria	3. 3 oz	9 oz	1. 6½oz	1. 3½oz	3. 6½oz	12 oz	4 oz	4 oz	
Mkikuyu, Ad. M.	Malaria	2.13½oz	10½oz	14½oz	14½oz	3.10½oz	13 oz	4½oz	4½oz	
Mkikuyu Boy	Typhoid	2. 4½oz	5½oz	6½oz	5½oz	1.15½oz	4½oz	4 oz	4 oz	
Mkikuyu Boy	Pleurisy	2.10½oz	5½oz	10½oz	6½oz	2. 8 oz	14 oz	4½oz	3½oz	
Mchaga, Ad. M.	Pneumonia	2.15½oz	10 oz	1.14½oz	1.12½oz	3. 9½oz	14 oz	4 oz	4 oz	
Mkamba, Ad. M.	Pneumonia	2.11½oz	10½oz	2.12½oz	1.- ½oz	2.11½oz	12½oz	4½oz	4½oz	
Mganda, Ad. M.	Dysentery	2. 6½oz	6½oz	1. 9½oz	2. 5½oz	3. 1 oz	5 oz	4 oz	3½oz	
Mkamba, Ad. M.	Pneumonia	3. 6½oz	9½oz	3.11½oz	1. 4½oz	4. 3 oz	1. 7 oz	7½oz	6½oz	
Mkavirondo, Adolescent male	Trypanosomiasis	2. 5½oz	6½oz	11½oz	6 oz	3. 4½oz	11½oz	3½oz	4½oz	Small ulcers in the intestine cerebro + spleen
Mkikuyu, Ad. M.	Pleurisy	2.12½oz	8½oz	1. 8 oz	12½oz	2.13½oz	12½oz	4½oz	5½oz	
Nandi, Adolescent F.	Tuberculosis	2. 9½oz	6 oz	6½oz	6 oz	2. 5 oz	7½oz	5½oz	7½oz	
Mkamba, Ad. M.	Pleuro Pneumonia	2.15½oz	11 oz	1. 6½oz	1.14½oz	3. 9½oz	12 oz	6 oz	6½oz	
Mkamba, Ad.M.	Pneumonia	2.10 oz	5 oz	9 oz	5½oz	1. 9½oz	4½oz	2½oz	3 oz	

Tribe	Disease	Brain	Heart	R.Lung	L.Lung	Liver	Spleen	Right Kidney	Left Kidney
Nandi, Ad. F.	Dysentery	3. 1½oz	8½oz	15½oz	15½oz	3.15½oz	11 oz	5oz	5 oz
Mkikuyu, Ad. M.	Dysentery	3. 4½oz	10 oz	1. 2 oz	14½oz	3.10½oz	7½oz	4½oz	5½oz
Mkamba, Ad.M.	Pneumonia	2.14½oz	7 oz	2.12 oz	14½oz	3.12½oz	10½oz	5 oz	4½oz
Mkikuyu, Ad.M.	Typhoid	2. 8½oz	8½oz	1.10½oz	1. 1 oz	3. 5½oz	8½oz	3½oz	3½oz
Mkikuyu, Ad.M.	Pneumonia	2.15½oz	12½oz	3. 1 oz	10½oz	3.15 oz	15½oz	5½oz	6½oz
MSwahili, Ad.M.	Pleurisy	2. 9 oz	10½oz	13½oz	1. 3 oz	3. 2½oz	1.- ½oz	5 oz	3½oz
Mkikuyu, Ad.F.	Pneumonia	2. 9½oz	11½oz	2. 1½oz	1. 4½oz	3.14½oz	10 oz	5½oz	6 oz
MSwahili, Ad.M.	Pneumonia	2.10½oz	15½oz	2. 7 oz	11b.	4. 4½oz	15½oz	5½oz	5½oz
Mkikuyu, Ad.M.	Drowning	2.11½oz	8½oz	9½oz	8½oz	3. 6½oz	7½oz	3½oz	3½oz
Mganda, Ad.M.	Epilepsy	3.- ½oz	15½oz	1. 8½oz	1.10½oz	5. 6½oz	1.- ½oz	3½oz	4½oz
Mkikuyu, Ad.M.	Comp.Fract.ofSkull	2. 3½oz	10 oz	13½oz	12½oz	3. 2 oz	4½oz	4½oz	4½oz
Mkikuyu, Ad.M.	Burns	2. 9½oz	6½oz	13½oz	6½oz	2. 7½oz	4½oz	2½oz	2½oz
MSwahili, Infant F.	Con.Syphilis	1. 2½oz	1½oz	1½oz	1½oz	8½oz	1½oz	½oz	½oz
Mgogo, Ad.M.	Pneumonia	2.11½oz	8½oz	13 oz	2. 6 oz	4. 9½oz	10 oz	6½oz	6 oz
Mkamba, Ad.F.	Pneumonia	2. 5½oz	6½oz	1. 5 oz	14½ oz	2.15 oz	9½oz	4½oz	4½oz
Mkaromojo, Ad.M.	Dysentery	3. 1½oz	6½oz	9½oz	7½oz	2.13½oz	3½oz	4½oz	4½oz
Masai Ad.F.	Encephalitis	2.15½oz	5½oz	6 oz	11½oz	2.11½oz	6 oz	3 oz	3½oz
Mkamba, Ad.M.	Meningitis	3. 6½oz	10½oz	15½oz	10½ oz	4. 4 oz	9½oz	5 oz	5 oz
Mkikuyu, Ad.M.	Malaria	3. 1½oz	9 oz	1. 1 oz	12½ oz	3. 8½oz	1. 8½oz	3½oz	3½oz
Mkamba Boy	Pleurisy	3 lbs	5½oz	15½oz	9½oz	3.10½oz	2. 2 oz	5½oz	5½oz
Hybrid F.Child	Colitis	2. 5½oz	2½oz	2½oz	2 oz	1. 1½oz	1½oz	1½oz	1½oz
Mkamba, Ad.M.	Pneumonia	2.15½oz	14½oz	3. 3½oz	15½oz	5 lbs	8½oz	6½oz	6½oz
Abyssinian Ad.M.	Aortic Stenosis	3. 2½oz	1. 4 oz	1. 2½oz	1. 3½oz	3. 2½oz	7½oz	4½oz	5½oz
MSwahili, Ad.M.	Arthritis	2. 8½oz	15½oz	1. 7 oz	15 oz	3. 3½oz	6½oz	5½oz	5½oz
Mkikuyu, Ad,M.	Malaria	2. 7 oz	8½oz	8 oz	7½oz	3. 1½oz	9½oz	3½oz	4 oz
Mkikuyu, Ad.M.	Drowning	2. 8½oz	7 oz	9½oz	8oz	2. 5½oz	4½oz	3 oz	3 oz
Mkikuyu, Ad.M.	Typhoid	2. 5½oz	5½oz	7½oz	12½oz	3 lbs	1. 2½oz	4 oz	4½oz
Mkikuyu, Ad.M.	Typhoid	2. 8½oz	7½oz	1. 2½oz	11 oz	3. 2 oz	13½oz	4½oz	4 oz
Mkamba, Ad.M.	Tuberculosis	2.11½oz	6½oz	1.13½oz	1. 9 oz	3. 5 oz	1. 5 oz	5½oz	4½oz
Mkamba, Ad.M.	Pneumonia	3. 2 oz	10 oz	1. 5½oz	3. 8½oz	4. 9½oz	2.1½oz	5½oz	6½oz
Mganda, Ad.M.	Meningitis	2.14 oz	8½oz	10½oz	7½oz	4. 6½oz	11½oz	5½oz	4½oz
Mkikuyu, Ad.M.	Meningitis	2.13½oz	9½oz	1. 1½oz	1. 1½oz	2.10½oz	5½oz	6½oz	none
Masai, Ad.M.	Tuberculosis	2.15½oz	7½oz	2. 1 oz	1. 7½oz	3. 2½oz	10 oz	4½oz	4½oz
Mnyamwezi Ad.M.	Pneumonia	2.10 oz	1. 1½oz	14½oz	2. 9oz	4. 1 oz	1 lb.	5½oz	8½oz
Mkamba, Ad.M.	Pneumonia	3. 3 oz	8½oz	1. 4½oz	10½oz	3.13½oz	2. 1 oz	6½oz	6½oz
Mganda, Ad.M.	Pneumonia	3. 2½oz	13 oz	1.12½oz	2. 8½oz	4. 9½oz	1. -½oz	4½oz	5 oz
Masai, Ad.F.	Tuberculosis	2. 4½oz	3½oz	8½oz	8 oz	2. 1½oz	3½oz	2½oz	2½oz
Nandi, Ad.F.	Ruptured Uterus	2.10½oz	8oz	10½oz	9½oz	4. 1 oz	13½oz	4 oz	3½oz
Mkikuyu, Ad.M.	Spirillosis	2.11 oz	9½oz	10½oz	13½oz	3. 7½oz	1.13 oz	4 oz	6 oz
Mnyamwezi Ad.M.	Phthisis	2. 8½oz	13½ oz	1. 7 oz	1. 2½oz	2.15 oz	6 oz	5 oz	5 oz

Tribe	Disease	Brain	Heart	R.Lung.	L.Lung.	Liver.	Spleen	Right Kidney	Left Kidney	
Somali, Ad.M.	Pneumonia	-	10¼oz	1. 6 oz	14½oz	3. 1½oz	11½oz	4¼oz	4½oz	
MSwahili, Ad. M.	Dysentery	2. 8¾oz	7½oz	9½oz	8½oz	2.10½oz	4 oz	3½oz	4 oz	
Mkikuyu, Adolescent Male.	Pneumonia	2. 6½oz	9¾oz	1.- ½oz	13¾oz	3. 5½oz	1. 9¾oz	3¾oz	4 oz	
Mkikuyu, Adolescent Male.	Pleurisy	3. 3½oz	10½oz	13½oz	1. 2½oz	3.11¾oz	1.- ½oz	4 oz	4½oz	
~~Masai, Ad. F.~~	~~Burns~~	~~2. 9 oz~~	9 oz	9½oz	7½oz	3. 4½oz	7 oz	3½oz	3¾oz	
Mkikuyu, Ad. M.	Gunshot wound	2.12¾oz	7¾oz	9¾oz	9½oz	2.13½oz	4 oz	3¾oz	4 oz	
Mkikuyu, Ad. M.	Syncope	2. 7¾oz	9¾oz	11½oz	6¾oz	2. 2¾oz	2¾oz	2¾oz	3½oz	→ Following wound of left foot. Shock
Mkikuyu, Ad. M.	~~Caseating Lungs~~ Pulmonary Tuberculosis.	2.13½oz	6¾oz	2. 3¾oz	1.11½oz	3.10½oz	12½oz	4½oz	2¾oz	
MSwahili, Ad. M.	Meningitis	3. 2¾oz	10½oz	14 oz	14 oz	4. 4¾oz	2.10½oz	5½oz	5½oz	
Mkamba, Ad. M.	Pneumonia	2.14½oz	11¾oz	3. 6½oz	10½oz	3.12½oz	2. 1½oz	5½oz	5½oz	
Mkikuyu, Ad. M.	~~Caseation of Lungs~~ Pulmonary Tuberculosis	3. 4 oz	1. 1 oz	2. -	2. 2. oz	4. 4½oz	14½oz	7 oz	6½oz	
Mkikuyu, Ad. M.	Fracture of Spine	2.12½oz	7¾oz	7¾oz	7½oz	2.-	13½oz	2¾oz	3½oz	
Mkamba, Ad. M.	Old Pleurisy	2.15½oz	6¾oz	8½oz	7½oz	3.12½oz	1. 8½oz	11¾oz	10oz	
Mkikuyu, Ad. M.	Adherent Pericardium	2. 9½oz	9½oz	8 oz	11½oz	2.13 oz	11½oz	3¾oz	3¾oz	
Msoga, Ad. M.	Peritonitis	2.11¾oz	12½oz	1. 3½oz	1. 2½oz	4. 3½oz	5½oz	5½oz	5½oz	
Mkikuyu, Adolescent Male	Meningitis	2.13¾oz	8½oz	12½oz	9¾oz	3. 2½oz	9½oz	4½oz	4¾oz	
Nandi, Ad. M.	Beriberi	3. 3 oz	13¾oz	12½oz	11¾oz	3.13½oz	7¾oz	5½oz	5½oz	
Mkikuyu, Ad. M.	Pneumonia	2.15½oz	9½oz	2. ½oz	1.14½oz	3. 1½oz	11½oz	4¾oz	4 oz	
Mnyamwezi, Ad. M.	~~Caseation of lungs~~ Pulmonary Tuberculosis	2.13½oz	10½oz	2. ¾oz	2.15 oz	2.15½oz	5½oz	6½oz	6¾oz	
Nandi, Ad. M.	Beriberi	2.7¾oz	11 oz	7¾oz	7 oz	2.10½oz	2¾oz	4¾oz	4½oz	
Mkikuyu, Ad. M.	Local Injuries	2. 5¾oz	5 oz	7¾oz	9¾oz	2. 1½oz	5½oz	3 oz	3 oz	
Mkamba, Ad. M.	Pneumonia	3lbs.	13½oz	1. 2½oz	1. 9½oz	5.15 oz	2lbs	6¾oz	7 oz	
Nandi, Ad. M.	Pneumonia	3. 3¾oz	9½oz	2. 7 oz	2. 2 oz	4. 2 oz	5½ oz	5½ oz	5½oz	
~~Mkikuyu Boy~~	~~Pneumonia~~	~~2.15 oz~~	5½oz	13 oz	4½oz	1.15¾oz	8 oz	2¾oz	2¾oz	
Mkikuyu, Ad. M.	Dysentery	3. 2 oz	7½oz	10½oz	7¾oz	2.13 oz	9¾oz	4½oz	4½oz	
Mkikuyu, Ad, M.	Pneumonia	3.- ¾oz	1. 1½oz	2. 2 oz	2. 2 oz	4. 1½oz	7½oz	3¾oz	5¾oz	
Mkamba, Ad. M.	Pneumonia	2.14 oz	11¾oz	11¾oz	1..9½oz	3. 3¾oz	7½oz	4¾oz	4½oz	
Mkikuyu, Ad. M.	Acute Tuberculosis	2.13½oz	9¾oz	1. 6½oz	1.11½oz	2.13½oz	10¾oz	3½oz	4 oz	
Masai, Ad. M.	Malaria	3. 1½oz	7½oz	10¾oz	7¾oz	2.15 oz	13 oz	3¾oz	3¾oz	
Mkamba, Ad. M.	Pneumonia	2.13 oz	9½ oz	1. 1 oz	1.10½oz	3. 2½oz	2. 9½oz	5¾oz	6 oz	
Mkikuyu, Ad. M.	Pneumonia	2. 9¾oz	14½oz	1.15½oz	2. 6¾oz	4. 6 oz	12¾oz	5¾oz	5½oz	
Mkikuyu, Ad. M.	Burns	2.10¾oz	8½oz	1.- ½oz	1.- ¾oz	2. 3½oz	2½oz	3¾oz	3¾oz	
Mkikuyu, Ad. N.	Burns	2.14½oz	11½oz	9 oz	7 oz	2.11¾oz	4½oz	3 oz	2 oz	
~~Masai, Ad. F.~~	~~Tuberculosis of~~ Lungs	~~2. 5¾oz~~	6½oz	1.15¾oz	1.10½oz	3.13¾oz	7 oz	4¾oz	4½oz	
Mkikuyu, Ad. M.	Dysentery	2.15¾oz	5¾oz	8½oz	7½oz	2.13¾oz	6¾oz	4¾oz	4½oz	
Mkamba, Ad. M.	Pneumonia	2. 9½oz	. 8 oz	1½oz	1.12¾oz	3. 6 oz	13¾oz	5¾oz	6½oz	
MSwahili, Ad. M,	Gonorrhoea	2.12¾oz	9¾oz	8½oz	7½oz	2. 4¾oz	3½oz	6¾oz	6 oz	

Tribe	Disease	Brain	Heart	R.Lung	L.Lung	Liver	Spleen	Right Kidney	Left Kidney
Mkamba, Ad. M.	Sarcoma	2. 8 oz	8¼oz	14¼oz	11½oz	2. 8 oz	10¼oz	2. 1¼oz	2. 7¼oz
Mbere, Ad. M.	Pneumonia	3. 5½oz	6½oz	1. 11oz	14 oz	2.13½oz	15 oz	4½oz	3½oz
Meru, Ad. M.	Pleurisy	2.15 oz	14½oz	1. 2½oz	1. 6½oz	3. 5½oz	15 oz	4 oz	5 oz
Mbere, Ad. M.	Pneumonia	2.15 oz	10½oz	1. 4¼oz	3. 1¼oz	4 lbs	1. 7¼oz	6¼oz	6¼oz
Mkikuyu, Ad. M.	Pneumonia	2.11½oz	9½oz	13 oz	1.11½oz	3.13½oz	13 oz	4½oz	4½oz
Mkikuyu, Ad. M.	Pleurisy	2.14½oz	11 oz	14 oz	11¾oz	4. 3 oz	1.14½oz	5¾oz	6 oz
Mchaga, Ad. M.	Fract. of Skull	2.11½oz	5¾oz	7¾oz	6 oz	1.12½oz	7 oz	3 oz	4 oz
Mkamba, Ad. M.	Tuberculosis	2.12¾oz	11½oz	10 oz	1. 8 oz	3. 5¾oz	1. 6½oz	7¼oz	8½oz
Embu, Ad. M.	Dysentery	2. 9½oz	6½oz	14¾oz	15½oz	2. 6½oz	3½oz	3¾oz	3½oz

J. A. Haram.

167 cases

actual weight

ISTHMIAN CANAL COMMISSION
DEPARTMENT OF SANITATION
ANCON, CANAL ZONE

COL. W. C. GORGAS, U.S.A.,
CHIEF SANITARY OFFICER

In reply refer to File No.....

May 29th, 1911.

Estivo-autumnal Parasite
By Dr. H. M. James
Ancon Hospital
Ancon
Canal Zone
Central America

The Editor:
The Journal of Tropical Medicine & Hygiene,
83-91 Great Tichfield St.,
London, England.

Sir:-

In the ~~December~~ February number of the Journal of Experimental Medicine, New York, (Vol.XIII,page 263) is an article by Dr. Mary Rowley-Lawson, on the Estivo-Autumnal Parasite. In this article the author describes what she believes to be flagellating crescents in the peripheral blood, with an account of the manner of the flagellation, the subsequent impregnation of the female crescent by a flagellum, and consequent sporulation of the fertilized form. This process is described as occurring in its entirety in specimens taken from the peripheral blood, and one is led to believe that this third cycle of the parasite, with all the details as described, happens normally in man, and the resulting parasites are those which are responsible for relapses.

To ascertain the true cause of relapse in malaria, and to learn how to prevent it, has been for many years the object of not a few students of this disease in Tropical countries. Only those who daily treat malaria, and who witness relapses in cases treated according to the best knowledge of the malady which is at present available, fully realize the importance of the solution of this problem. A very great percentage of the malaria on the Canal Zone is due to relapse and

not to re-infection, and for this reason any information as to the genesis of relapses is of value to us, and to others who daily treat malaria in countries where this disease is both endemic and and epidemic.

I failed to find in the author's paper any mention of control experiments with fresh blood. I cannot conceive that the changes in the gametes as described by her would be noted in stained specimens only, in so far as they refer to the formation and extrusion of flagella while the parasite is in the crescent stage. The same is true of the appearances described for the parasite while it is in the asexual form. Had control experiments with fresh blood been made, and if the author had been familiar with the work of Marchiafava and Bignami on the position of the parasites with reference to the erythrocytes, the error of assuming the organisms to be extra-corpuscular could not have been made. To make deductions from stained specimens only when it is possible to control the appearances under consideration with fresh blood observations, is to argue from very insufficient premises, to say the least. I wish, however, to bring before you, what seem to me to be ~~the~~ fundamental errors which make her deductions of no value whatsoever. This latter is to be regretted, for the colored plate at the end of her article contains beautifully delineated and remarkably accurate representations of certain phases in the life history of the crescents, and it is unfortunate indeed that to these representations cannot be attributed their proper interpretation, as the illustrations are the best I have hitherto seen.

The author begins her paper with a description of the male and female forms of the crescents. She interprets the staining

reactions of these in a manner directly the reverse of what is true. It is the male crescent or microgametocyte and not the female or macrogamete, whose cytoplasm takes on a light hue after the use of one of the modifications of the Romanowski stain. The author describes the macrogamete as staining a light blue and the microgametocyte as taking on a darker blue color. The error of this assumption is obvious to anyone who is at all familiar with the staining reactions of crescents. Very frequently the cytoplasm of the microgametocyte does not take the stain at all, and this is true not only of the crescents, but also of the male gametes of tertian and quartan malaria. In fact, among the protozoa in general, the microgamete, since it is destined for future reproduction, contains a relatively abundant supply of cytoplasm, which reacts to the basic part of the polychrome stain in proportion to its amount, while the microgametocyte, which supplies chromatic material only, is relatively poor in cytoplasm, and for the most part takes a hyaline appearance after the use of the stain.

Following this fundamental error, the author goes on to describe a process of flagellation in the supposed microgametocytes, which in reality are macrogametes. This process is described as occurring in the peripheral blood, prior to its withdrawal from the body.

In this respect it is to be regretted that the author did not give details as to the manner of obtaining the specimens. I shall endeavor to make my statement clear. The so-called flagellation is not a process of flagellation at all, but a description of the reduction of the chromatin of the macrogamete. In Plate XXIX, Figures 1-10, inclusive,

are interpreted by the author as various degrees of flagellation in the microgametocyte, and as occurring in the peripheral blood before withdrawal, the supposition being that the specimen dried too rapidly for the representation to be otherwise. In reality the figures represent moist chamber preparations of female crescents. The appearances as described for the parasites immediately after withdrawal are wholly inaccurate, and never take place if the blood is dried at once. However, if preparations are exposed to dampness either accidently or in a moist chamber, the process, as described, begins immediately, but it occurs, as I have said, in female crescents and not in males. The so-called flagella are not true flagella, but are forms taken by the chromatin during reduction. The true flagella are very different in appearance, and had the author been familiar with their appearance, she would not have confused them. I make these assertions confidently, as I have done a great deal of work recently and in the past, on moist chamber preparations of crescents and I am entirely famaliar with the appearances of the male and the female forms from the time they leave the peripheral blood, both in fresh and in stained preparations until flagellation and reduction have been completed.

There is no doubt whatever that the forms described by the author are not to be found as she infers, in the usual preparations, but are to be obtained only in moist chamber preparations or after the slides have been exposed to dampness, accidently or intentionally. Reduction in both male and female forms takes place very rapidly after exposure to air or dampness, and the so-called flagellate appearances occur after exposure of one minute in a moist chamber, or if, in the presence of atmos-

10

pheric dampness, the slide does not dry immediately.

Similarly, in Plate XXIX, Figures 12-23 are all reduction stages of the macrogamete. I think you will agree with me that the author has not made the proper differentation in the delineation of the male and female crescents. Figures 24 and 25, which are supposed to represent microgametocytes after flagellation are nothing more than macrogametes, whether before or after reduction I cannot say, as the details are not sufficiently clear, but probably the latter.

The whole description of this so-called flagellation displays remarkable unfamiliarity with a knowledge of the morphology not only of estivo-autumnal parasites but of other species as well. One can attribute to inexperience the careful and laborious account of reduction in the macrogamete as a process of flagellation in the microgametocyte, but it is not so easy to understand how the author could have described as occurring in the peripheral blood the forms that appear only after exposure to dampness, or in a moist chamber.

Much of the description of the asexual cycle is on par with that of the process of flagellation. The "loop" attachment, by which the parasite is supposed to maintain its perilous position on the outside of the erythrocyte, is nothing more than a fixation of one of the amoeboid shapes of the organism. If the parasite is observed in the fresh blood, especially if the patient has taken quinine, the "loop" will be seen to withdraw into the body of the plasmodium, and to have an entirely different significance from that attributed to it by the author.

The "bar" forms are very common in fresh blood specimens from cinchonized patients, and their interpretation as a proof that the //

parasite is extra-corpuscular is only one of the many mistakes made in the article.

I am, sir,

Very truly yours,

H. M. James.

Physician,

Ancon Hospital,

bfo. Ancon, Canal Zone.

P.S.

This letter is sent through the Acting Chief Sanitary Officer, and if you care to publish it, I shall be very glad to have you do so, omitting, of course, the postscript.

H. M. James.

12

T.M.

6159

OFFICE RECEIVED 30 APR. 1907 JEYES' SANITARY COMPOUNDS CO. LTD.

Test No. .....

Date April 30 1907

Meningococcus, 24 hours Nagar

~~B. Pestis, 18 hours' Agar~~ Culture at 37° C.

Room Temperature 15°-18° C.

Sample.	Dilution.	Time culture exposed to action of disinfectant—minutes.			Sub-Cultures.	
		1	2	3	Period of Incubation.	Temperature.
Cyllin	1 : 9000	++	reduced	greatly reduced	4 Days	37°C
"	1 : 8000	—	—	—	"	"
Phenol	1 : 160	++	reduced	greatly reduced	"	"
"	1 : 150	—	—	—	"	"

3

∴ Carbolic Acid Co-efficient $\frac{9000+8000}{160+150} = \frac{17000}{310} = 54.8$.

$\left[\frac{9000}{160} = 56.2 \; ; \; \frac{8000}{150} = 53.3\right]$

E. Klein.

$$
\begin{array}{r|l}
155 & 8500 \\
 & 775 \\
\hline
 & 750 \\
 & 620 \\
\hline
 & 1300 \\
 & 1240 \\
\hline
\end{array}
\quad (54.8
$$

T. M. O. C.

89186

6159

Note on

THE DISINFECTION OF THE MENINGOCOCCUS
(Diplococcus intracellularis)
with Cyllin & Carbolic Acid.

by

E. Klein, M.D., F.R.S.,

* * * * * * * * *

A nasgar slope culture (nutrose ascites fluid agar) was obtained from the spinal fluid (lumbar puncture) of a case of cerebro-spinal meningitis. This culture was perfectly typical for meningococcus, viz., a trans-lucent filmy growth of a sticky viscid consistency and composed of gram-negative cocci. From this primary culture a surface plate of nasgar was prepared, and from a single isolated colony a further sub-culture on nasgar slope was made. The bacterial emulsion was prepared in the following manner: a vigorous growth of a 24 hours nasgar slope was distributed in 5cc of distilled water, or broth containing 10 per cent of normal horse serum; no difference was noticed in respect of resistance of the microbe to the disinfectant when the emulsifying fluid was prepared with the former or the latter.

To 5cc of the disinfectant 10 drops of the turbid emulsion of the meningococcus were added and after the desired time of

exposure (2½, 5, and 7½ minutes) ~~three~~ 3 loops of the mixture were transferred to, and rubbed over, the entire surface of a Nasgar slope. The inoculated tubes were then incubated at 37°c. It may be of interest to note in passing that no growth appeared in any of the tubes after 48 hours incubation.

A trial Nasgar slope inoculated with a single loop from a mixture of 10 drops of the bacterial emulsion and 5cc of distilled water brought forth an unaccountable - in many places almost confluent - number of colonies.

In the following table* the results of the experiments are summarised :-

(Take in table).

Take in after letter. Folio 3

3

From this it follows that absolute phenol when diluted in the proportion of 1 part to 150 parts of water kills the meningococcus in one minute, the same effect being produced with Cyllin when diluted in the proportion of 1 part in 8,000 parts of water, whilst 1 part of pure phenol in 160 parts of water shews a good growth in one minute, a reduced growth in two minutes, and a greatly reduced growth in three minutes, Cyllin performing the same work when diluted in the proportion of 1 part to 9000 parts of water. The Carbolic Acid coefficient for Cyllin is therefore $\frac{9000 + 8000}{160 + 150} = \frac{17000}{310} = 54.8$

* This table gives the results of a series of experiments made with a particular strain of the meningococcus in a definite brew of Nasgar; it does not claim to apply to all strains of the microbe, or to all brews of Nasgar: but although the actual dilutions of the control and postulant would vary for different brews of Nasgar, the ratio between phenol and Cyllin would be approximately the same - a fact which has been borne out by other investigators when determining the carbolic acid coefficient for other organisms.

3

OC. Hodgson 205/2 89186 1 6158

A NOTE ON THE DISCOVERY BY MR. A. GREY OF GUINEAWORM IN A LEOPARD.

by

Robert T. Leiper M.B., F.Z.S.

Helminthologist to the London School of Tropical Medicine.

Proof to 60 Elms Road Clapham Park S.W.

There are ~~several~~ anomalies in the geographical distribution of Dracontiasis ~~that have no adequate explanation.~~ One of the most curious has been the absence of the disease in Africa south of the Equator although endemic ~~whilst its prevalence extends~~ northwards from the line to ~~as far as~~ the Tropic of Cancer. It has been suggested that possibly the particular species of Cyclops essential to the development of the parasite may not occur in the southern half of the continent. ~~This theory seems scarcely tenable for~~ however Quite recently a mature guineaworm has been found in a Leopard at Broken Hill, North West Rhodesia, by Mrs C. Grey. I am indebted to Sir Patrick Manson for the specimen and for kind permission to record it. In a letter dated Nov. 27th. 1909. Mr. Grey says "The leopard appeared to have guineaworm. I killed it the night before last near my house but could not get at it till yesterday morning. On skinning it I found pieces of worm visible in eight or nine places in the connective tissues and appearing to come from beneath it. The first worm I noticed was sticking out of the bullet-hole in the shoulder. The leopard was in good condition and had a very good coat. I have never heard of the guineaworm being found in man here. The doctor here has never had a case. I send a piece of the worm" and some films.

The piece of worm sent resembles exactly the guineaworms found in human beings on the West Coast of Africa. In appearance it is white and glistening. In length 45 cms in breadth ☐ mm. Both extremities are unfortunately missing. The body is filled by a single uterine tube crowded with embryoes which correspond ~~both~~ exactly in size and structure with those of Dracunculus medinensis from man.

There are a number of records in Literature of the occurrence of Guineaworm in the lower animals. They may be divided into:-

(1)(~~*~~).Those based on common report: Under this heading are to be grouped the references to the presence of guineaworm in Cattle and dogs in the writings of Avenzoar 1490. De Marchais 1727 and Pruner-Bey 1847. Hussem 1771 states explicitly that Doerssel found a Guineaworm in a dog at Buenos-Ayres and at another time in a dog at Curaçal. Kuchenmeister 1855 ~~states~~ is of opinion that the parasites may be met with in aquatic birds! ~~also~~! Heckenroth (1906) is quoted by Bartet as authority for the statement that the disease occurs in sheep and cattle in West Africa.

~~(2)~~ Papers describing actual cases. These record infections in the Horse, Dog, Wolf, Jackal, Hunting Leopard and Monkey.

Smyttan (1925) says "I have never heard of quadrupeds being subject to "Dracunculus but an instance of its occurred here (in India) last rains in one of the "officers' dogs. After being lamed for a few days in one of his fore-legs "he was observed by several officers(some of whom had had too painful experience "to mistake the thing) to pull a Guineaworm out with his teeth"

Piot (1889) describes six cases ~~in dogs~~ in Egypt, in viz:-

(1) A European dog in which the worm made its appearance on the inner surface and in the upper third of the right fore-leg. The owner had already extracted a fragment 18 cms. long and after some days Piot removed 30cms. more.

(2) A native dog, in which a worm presented on each of the four limbs viz.

(a) on the middle of the internal surface of the right fore-leg.

(b) on the lower third and posterior surface of the left fore-leg.

(c) on the middle of the outer surface of the metatarsus of the right hind-leg.

(d) on the lower third of the inner surface of the left hind-leg.

Shall have to go in after Griffith (1858) on page 4

D.C.

(3) A Jackal, killed at Aboutig in Upper Egypt, in which there were three worms. One worm lay coiled up on the inner surface of the left knee-joint, another on the inner surface of the ~~left~~ right leg stretching as high as the upper third of the thigh. A third, situated in the upper third of the left fore-leg and of which a fragment 12 cms. in length was extracted.

(4) a native dog which had a number of subcutaneous tumours in the right hypochondriac region, on the sternum and on the inner aspect of the left fore-leg.

(5) a dog from which Dr. Sonsino and Mr. Walter Innes extracted a piece of guineaworm about 40 cms. long from the hind foot.

(6) A wolf in which a guineaworm was found lodged in the subcutaneous tissues of the left thigh.

Forbes (1838) relates that "A tattoo was exhibited at Dharwar (Bombay) having a worm protruding from the right hind fetlock: it was of the usual size and made its appearance as a boil: I examined it before and after extraction, and could perceive no difference in any respect from the human Dracunculus. My friend Dr. Walker gave me a specimen of a guineaworm extracted from the neck of a dog and I have undoubted reports of a similar occurrence at this station".

Clarkson (1844) records "A Case of F. medinensis in a horse of Australian Breed at Katagherry India in 1837" He says "A small enlargement on the lower part of the large pastern, in the centre of which, from a peculiarly irritable sore a guineaworm was seen hanging out about the length of two inches. It had somewhat the appearance of a small tendon, being about 2 m broad, and half that in thickness: the portion that had been most exposed to the air was withered and shrivelled. On pulling gently at the worm two inches more were

2 m

1 3

drawn out; and it then became fixed. Fomentations were directed to be tried. On t the next morning two inches more of the worm were readily withdrawn from the opening. In this state it continued for three days when the worm was no longer visible".

Cobbold (1881) showed, at a meeting of the Linnean Society a specimen of guineaworm that he had received from Veterinary Surgeon F.Smith. The worm was taken from a pony at Secunderabad, India. Dr.Cobbold remarked that " the occurrence of "Dracunculus medinensis in the horse is extremely rare-so much so that some "authorities including Fedschenko have altogether denied its presence in Solipeds" "A comparison of the embryos from Mr.Smith's specimen with some of the young from" "an ordinary human guineaworm recently sent by Dr.Macallum of Bombay showed" "however that the embryos are in all respects identical."

Griffith(1888) saw a guineaworm in a fox-terrier bitch belonging to an officer in Cairo,Egypt. "The worm was situated along the front of the fore-arm extending from the elbow to the foot: it could be distinctly felt underneath the skin, coiled up like a piece of twine. The dog appeared in good health but an abcess had formed in the region of the foot at the extremity of the worm". The worm when extracted measured ~~two and a half~~ 2½ feet in length: it was submitted to Dr. Osman Bey Chalib,a well known naturalist,who confirmed the diagnosis.

The Editor of the Veterinary Journal(1888) commenting upon this case said "Whilst at Tientsin,North China,we found the mature Dracunculus in an abcess on the outside of the tarsus of a horse which had been brought from India. The specimen was sent home to Professor Dick and is now in the Museum of his School in Edinburgh

Note to Printer Proof 1889 Jan. to come in here → (from Page 2-3.)

Batliwala (1893), a Veterinary Instructor in Burma, described a case in the Horse at the Bombay veterinary Association. "The animal was,previous to date,in perfect

health. On examining him, I found him lame on the near hind-leg, but there was nothing to account for the lameness except that a small ulcer of about the size of a four-anna piece was found on the outer side of his fetlock joint with a little swelling around it. The swelling was painful to the touch. On pressure a few drops of pus together with a threadlike white structure, came out of the ulcer. On pulling it out, gently, I was able to draw forth a few inches of this threadLike structure. As it looked very much like guineaworm, which I may say is very common in man at Katyawar, I tied up the end of the worm with a piece of thread and wrapped it round the fetlock. The next morning I was again able to draw out about an inch more of the worm and kept it tied round the fetlock. On the third day however while pulling it out as usual the worm gave way and the whole thread now measured in its dry state about 5 in ~~five inches~~ in length".

Cazalbou (1909) found a dog at Timbuctu, where the worm was escaping between the interphalangeal articulations of the fore-leg.

Cinotti (1908) quotes a case of guineaworm in a bitch that had arrived in Italy from Egypt five or six months previously.

Cazalbou (1909) found three guineaworms in a monkey (Cercopithecus callitrichus) One situated in each of the lower limbs and the third presenting in the neighbourhood of the umbilicus.

Of special interest in connection with Mr. Grey's find is a paper published by Valenciennes in 1858 describing a number of filariae that were coiled up in the connective tissues under the skin of the legs and abdomen of a Hunting Leopard (Felis jubata) from Kordofan. One of these parasites had made a perforation on the inner aspect of the limb a little above the lower extremity of the tibia. Valenciennes named these worms ~~Filaria~~ aethiopica as ~~because~~ the head appeared to be more slender than that of the ~~Filaria~~ medinensis preserved in the

Paris Museum, but Blanchard considers them identical.

To sum up, most of the ~~cases~~ above records give sufficient detail to enable one to acquiesce in the diagnosis. ~~In the actual occurrence of~~ Upon the parasite in the dog. We have the authoritative opinions of Dr. Osman and Professor Railliet. ~~The~~ and of ~~parasite~~ Dr Cobbold for that from the horse, ~~was examined and reported upon by Dr. Cobbold~~, so that there seems little room for doubt that the Guineaworm is able to infect and attain maturity in ~~the de certain~~ these, if not in all, of the domesticated animals.

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R. T. Leiper 2/2/10

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Leiper

9

SUNDAY SCHOOL HOSPITAL
AMERICAN PRESBYTERIAN MISSION
CHANGTEH, HUNAN, CHINA

O. T. LOGAN, M.D.

Tpr.

SCHISTOSOMA JAPONICUM DYSENTERY IN AN AMERICAN CHILD.

By. O.T.Logan, M.D., Medical Missionary,
Changteh Hunan China.

While at Kuling, a mountain resort near Kiukiang, last summer, I was called upon to treat the 13 year old son of a missionary stationed near Yochow, Hunan. He was apparently suffering of ordinary dysentery in the subacute stage. As is my custom in all disorders of the intestinal tract, I made a microscopic examination primarily to endeavor to ascertain whether the boy had amebic or bacillary dysentery. To my surprise, every field of the microscope was well sprinkled with eggs of Schistosoma japonicum. ~~and consequently the whole aspect of the case changed~~.

Upon questioning the parents, it was found that the boy had been in the habit of wading barefoot in the ponds and Tong Ting Lake since the summer of 1908 and that in the latter part of that summer he had an attack of fever with slight bloody movements that were not regarded seriously. During the early part of this fever he was afflicted with giant urticaria which was regarded as angio-neurotic edema. This is noteworthy, as Lambert of Kiukiang reported in The China Medical Journal last year (Unfortunately most of my copies of this publication for 1910 have been destroyed so I cannot give the exact reference) a peculiar fever that was accompanied by cutaneous symptoms similar to those of the case in hand. This fever always followed after patients had spent some time shooting in the swamps around Kiukiang which are now known to be infested with Schistosoma japonicum. Only those who waded barefooted were affected. Houghton of Wuhu first suggested the probability that the wading fever reported by Lambert might be the first stage of Schistosomum infection. It will be interesting to hear further reports from these wading fever cases, as doubtless we shall within the present year.

On Sept. 3rd. 1910. A blood count of our patient showed:

Eosinophiles,	20%
Polyneuclears,	48%
Large mononeuclears,	16% (16%)
Small "	16%

Number of cells counted 350. Mononeuclears larger than the average polyneuclear cells were classed as "large".

There can be little doubt that this child was infected either through his skin or mucous membrane, as there is the slightest probability that he ever drank any water that had not been boiled. This coincides with the findings in Japan. Katsurada's He writes (Centralblatt f. Bakterioligie, Feb. 1 1910) that he failed to infect cats with S.japonicum by feeding, but could infect cats and dogs heavily with one and a half hours immersion in infected fields. In this connection, it is worth mentioning that it is impossible to keep foreign bred dogs in Kiukiang as they all sooner or later contract this disease. I examined the stools of a fox terrier that was reared in Hankow last year and found that the eggs of the fluke under discussion were abundant.

~~Our American boy is now in his native land and is making good progress physically. He is under the care of well known physicians who will doubtless report the case at a later date.~~

~~February 15th.1911.~~

A full report of our American boy's case will appear in an early number of The China Medical Journal. At present he is in his native land and is reported to be in fair health. He is under the care of well known physicians who will doubtless report the case later.

February 15th.1911.

To The Journal of Tropical Medicine and Hygiene,
London.

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A NOTE ON THE TRANSMISSION OF LEPROSY.

by

E.C.Long, M.R.C.S., L.R.C.P.,
Principal Medical Officer, Basutoland.

The means by which the Leprosy Bacillus is introduced into the human body have not yet been definitely established.

Commensal feeding, direct inoculation or contagion have been generally accepted as being the most likely methods of introduction, but no proof of any of these methods has yet been adduced, and I believe the Leprosy Bacillus has hitherto not been found outside the human body.

The fact that Leprosy is most common among people of unclean personal habits and living in unhygienic surroundings points to some co-existing source of contagion.

The possibility of the bacillus being carried by vermin occurred to the writer. With a view to testing this hypothesis, bed bugs obtained from huts which had never been inhabited by lepers were caused to bite lepers in the neighbourhood of leprous nodules on the face. The bugs were then killed and the alimentary tract and its contents examined. In every bug that bit freely a bacillus was found which in shape, size, and staining reactions is similar to the bacillus leprae.

Control bugs from the same hut gave in every instance a negative result.

The experiments are still in progress and it is intended as soon as circumstances permit to solve the following questions:-

1. How long the bacilli remain in the bug's body.
2. If bugs which over a period of weeks have fed on the blood from leprous nodules present any evidence of growth of bacilli in their tissues.
3. What organs of the bug contain bacilli.

4.

4. Whether any other vermin, e.g. fleas and lice, contain bacilli.

These experiments are only regarded as preliminary to a thorough investigation of the subject, but the question is so important that I have ventured to record my results in the hope that other observers may be induced to experiment on the same lines. I think it will be found that lepers may be inoculated by infected bugs and this point can be elicited by causing infected bugs to bite lepers on parts of the skin on which there are no leprous lesions.

If my hypothesis is correct a great many facts regarding the spread of leprosy which have hitherto been inexplicable would be made clear. Inquiries into the past history of certain lepers go to show that they could only have become infected with leprosy through some intermediate host.

The fact that plague is carried by fleas warrants, at any rate, the inference that leprosy may possibly be carried by bugs. Further the fact that only a percentage of human beings are attacked by the bed bug, or for the matter of that by the flea, would serve to explain why it is that only some of those people who live in close association with lepers become lepers also.

All lepers that I have questioned admit that bugs bite them freely, and it is not unreasonable to assume that such a voracious feeder as the common bug must, in the course of his nightly meal, ingest a considerable number of leprosy bacilli if his bites are in the neighbourhood of leprous nodules which are often swarming with the bacilli leprae. If such infected bugs were thereafter to bite a non-leper there would be a good chance of the bacillus being introduced into his system.

The following history of a recent case of leprosy

leprosy is only explicable by assuming some such method of infection as suggested above.

A native, X, residing in a village about three miles from Maseru presented himself as an out-patient about three months ago with some well marked "tubercular" leprous patches on the face. They had appeared about six weeks previously. There are no lepers in his village and none of his relatives are lepers. Inquiries into how he had spent his time and where he had been during the preceding year elicited the fact that he had during that period visited on three or four occasions a village about fifty miles away, where there was one leper who was, however, driven from the village during the period in question. X had been in the leper's hut but had never partaken of food there. After the leper had been driven away X spent one night in the hut and was severely bitten by bugs there.

The closest questioning failed to elicit any further evidence of contact with lepers or their dwellings, and one is almost forced to the conclusion that X was inoculated by leprosy-infected bugs or other parasites on the one night he spent in the infected hut.

THE PARTHENOGENESIS OF THE FEMALE CRESCENT BODY BY H. M. H E E B

ARMY SURGEON IN THE NETHERLANDS EAST - INDIES .-

Blanchard 1914

As is well-known the parthenogenesis of the tertian gamete was first observed by Schaudinn in a patient, Mrs. Kossel, and exactly described by this observer. This very important discovery, which must be considered of great value, as giving a clear and natural explanation of the cause of relapse, particularly in the case of persons, who have long since left the tropics and are no longer exposed to malarial influence, was afterwards confirmed by Dr. van HILST KAMERWEY and then by Drs. MERZ and BLÛML.

It was self-evident that an analogous metamorphosis of the tropical and quartan parasites must be sought on similar lines. When I was in Charge of the civil medical service at Koeta-Radja, I often had an opportunity of preparing blood-slides, obtained from natives suffering from fever, who had never been treated with quinine and many of whom were subject to a severe tropical malarial fever.

For a longtime my researches were fruitless, until two and a half years ago, I examined some blood-slides of a Bengalese suffering from fever, in which I discovered the particular forms of parasites shown in plates 1, 2 and 3.

Although, after an exhaustive examination of every conceivable hypothesis, I finally came to the conclusion that I probably had to do with the parthenogenesis of the female tropical gamete, still, as I hesitated to publish such a discovery without accurate verification and confirmation by competent authorities on Malaria, I thought it more prudent to wait for my furlough in order to seek the requisite information in Europe at Bordeaux and Hamburg.

In the former city Profr. le Dantec declared plates 1 and 2 to be sporulation parasites, but quite different from those, which are found in the common schizogenesis of the tropical parasites; but although he did not commit himself to any further explanation as to what they were, yet in every case they were derived from a gamete. At the Tropical Institute of Hamburg the experienced protozoölogist Dr. von Prowazek declared plates 1 and 2 to be parthenogenetic

-tic forms of the femle crescent bodies. Prof. Nocht, to whom I then submitted the prepared slides with the request that he would let me know his opinion, found plates I and 2 very interesting. He had never seen such forms and he supposed, that, everything considered, my view as to parthenogenesis was quite right. The temporary assistant and army doctor named Rodenwaldt and moreover Dr. Gonder, Dr. von Prowazek's assistant, suggested the possibility, although the probability was not very great, that we might have to do with two microgametocytes, which had just divided their chromatic mass, to form the microgametes, which would have been expelled forthwith.

Against this view I thought I could bring some well-founded objections, which I have still to formulate later on. Dr. Werner was of opinion that an exact criticism was scarcely possible because the prepared slides were stained with a Giemsa-solution, with which a small quantity of a solution of carbonate of potassium was mixed; for in such circumstances it was not inconceivable, that other substances of the cell and the parasite might also have been stained like chromatic substances. Dr. Mayer also found the demonstrated parasitic forms very peculiar but hesitated to express a definite opinion.

~~Now, since what precedes~~ I have shown how very difficult the interpretation of the demonstrated parasitic forms is, even for such authorities on malaria as. Prof. le Dantec, Prof. Nocht, Dr. von Prowazek and others, I therefore determined to further elucidate this point by reproducing to the best of my ability in coloured plates and with a descriptive text the above-described segmenting parasites.

Now coming to the subject itself the facts are as follows: If I am not mistaken the Bengalese already mentioned came to me during my consulting hour with a temperature between 38° and 39° ~~Celsius~~. The patient looked very feeble, ~~was~~ anaemic, cachectic, with an enlarged spleen and liver, but had never taken quinine. I prepared two slides in the usual way with a drop of blood.

These slides were made to congeal in a few seconds by rapid swinging of the arm, then they were immediately fixed with methylated spirits and afterwards stained for two hours with Giemsa-solution (Dr. Grübler Leipzig) 1 to 20 mixed with two or three drops of carbonate of potassium 1 to 1000.

with Chart-

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The Etiology of Blackwater Fever.

by G.J.Pirie, M.B; Ch.B., D.P.H. Abd.
W.A.M.S.

Summary 80

In these days, when there is still great doubt as to the true etiology of Blackwater Fever. I may be pardoned for sending these few notes on the peculiar similarity of the conditions that led up to attacks of Haemoglobinurea in four cases~~e~~ of Blackwater, which I have had under treatment during six years on the West Coast of Africa.

Case 1 (S) employed on The Gold Coast. This European had been several years in India, and had suffered from malaria there. Subsequently he came to West Africa, where he had repeated attacks of Malarial Fever. He said he had not been able to take Quinine as even a few grains (one or two) were sufficient to cause his urine to turn red. One afternoon he visited some friends some ~~mile~~ miles away, and while there he felt ill, and was persuaded to take three grains of Quinine by the mouth. He returned about 7pm. and came up to the hospital. while still feeling ill he sat down to have some dinner, and had just finished his soup when he felt very chilly, and soon after had a rigor. Blood films were taken, and in them were found a few subtertian Malarial parasites. Owing to his definite statement about Quinine no more Quinine was given. He was put to bed about 8'30pm., and about 12 midnight his urine first showed signs of haemoglobinurea. This increased till about 4am. when the urine was dark port wine colour. After that ~~th~~ the urine began to clear, and by 4pm. in the afternoon of the next

next day it was again clear. He informed me that he had had several attacks just such as this, and all after taking small doses of Quinine for what he called "Fever".

Case 2 (B) an engindriver. This European was doing his second tour on the Coast. He had had an attack of Blackwater, from which he had recovered. He was still in hospital when I saw him, and his urine had then been clear for three days. While still in hospital and about a week after I arrived, he complained of headache and sickness. His temp had risen and blood films were taken and ~~stained~~ stained by Leishman, and examined. Large numbers of subtertian rings were found. Owing to his just having recovered from Blackwater, he was not at first given Quinine. His temperature however continued to rise, and when it got to 104'6 F. and was still rising, I decided, that, in the presence of the parasites Quinine must be given. Accordingly 9grains Quinine Bihydrochloride were given intramuscularly about 4pm. About 11pm. he passed some haemoglobinuric urine. This increased till the urine was dark red, and then the urine began to clear. Twenty four hours after the ~~one~~ onset of the haemoglobinurea his urine was clear. His convalesence after this was uninterrupted.

This European had come from a highly malarial station, and he told me he had repeated attacks of " Fever" there.

Case 3 A railway Foreman. This man had been some years in South Africa. He had been one year in West Africa, and had been very careless with himself, and had especially neglected his Quinine. He was admitted to hospital about 11am. with a temp of 103 F. and

and on bloodfilms being taken and examined enormous numbers of s subtertian parasites were found. Practically every infected red cell had two rings. The patient was in a very collapsed condition, and 9 grs of Quinine were given inàramuscularly. His temp during the afternoon continued to rise, and at 5pm. was in spite of all treatment 105'8 F. At that time another intramuscular of 9 grains of quinine was given. Profuse perspiration started at 7'30pm., and by 1 am. his temp was just over normal. During the second 24 hours of his illness he was given about 40 grains of quinine by the mouth. During the forenoon of the third day his urine became haemoglobinuric. This never got greater than cause the urine to be dark red, and in twelve hours passed off and his urine became normal.There was slight rise of temperature with the haemoglobinurea.

Case 4 a Sergeant. He complained of headache and had a slight rise of temp to 101 F. Blood films showed a few subtertian rings. He was given during the first day of illness 20 grains of quinine bisulphate by the mouth, and his temperature came down to normal in the evening. During the second day he again took altogether 20 grains of quinine, and at 8 pm. passed some haemoglobinuric urine. His urine rapidly darkened till almost black, and his temperature during the haemoglobinurea rose to about 102 F. The quinine was stopped when the haemoglobinurea began. The urine continued dark port wine colour and at times almost black till the afternoon of the third day, when it began to clear, and was normal two days later. Two days later it was attempted to give the patient a tonic in the form of iron and arsenic Compound B.W.&Co. These contain each 2½ grains quinine. One tabloid was given in the forenoon, and at 1 pm.

there was distinct haemoglobinurea. No more tabloids were given and in four hours the urine began to clear. his urine ~~---~~ remained clear since. The four hourly chart of this case is interesting, as it shows during the continuance of the haemoglobinurea a daily rise of temperature, beginning in the forenoon and continuing all day and falling during the night, the typical appearance of a malarial temperature.

In all these cases no malarial parasites, though carefully looked for, were found after the onset of the haemoglobinurea.

The interesting points in these cases are :-

1--- The finding in each case of subtertian rings in the peripheral blood of the patients, before the onset of the haemoglobinurea.

2 --- In each case quinine was taken or given in order to treat the malaria, also before the haemoglobinurea.

3 --- In case "4" the recurrence of the haemoglobinurea when a small dose of quinine (2½ grs.) was given in the iron and arsenic compound tabloid. (see chart of case "4".)

4 --- The typical malarial temperature chart of case "4" during the continuance of the haemoglobinurea. There were no malarial parasites at this time in this patients peripheral blood, but it looks almost certain that sporulation was taking place in some of the internal organs.

Each of these patients had the appearance of having been well saturated with malarial poisoning. The first and third case had not taken or had taken quinine very irregularly. Number two had not used his mosquita net and had most probably,(though he said the contrary) taken his quinine irregularly. Case four had taken 5grains quinine daily. All these cases

cases developed Blackwater more or less severely. Fortunately however Blackwater does not occur in every case, where there has been a history of repeated attacks of Malaria, which have been treated with quinine. It would be absurd to dogmatise on the results of merely four cases, but it appears to me to be more than a mere coincidence, that in each of these cases the onset of the haemoglobinurea should have followed the attempt to treat an attack of Malarial Subtertian Fever with ~~quinine~~ quinine.

I ~~would~~ think that only those persons whose resistance to the haemolytic action of the Malaria parasite has been reduced by the continuous sporulation of the parasite in their system are susceptible, and that in those persons a dose of quinine may be the actual exciting cause. This condition of susceptibility is induced by ~~the~~ neglect of, or irregularity in the taking of the daily dose of five grains of quinine. In cases where one has been assured that five grains of quinine have been taken regularly every day, it may be that certain types of ~~co~~-constitution are so peculiarly susceptible to Malarial infection, that the five grains of quinine daily are not sufficient to prevent the continuous sporulation of the parasite in the system, though that dose may be sufficient to prevent any illness or great discomfort sufficient to make the patient take to bed. These persons get anaemic and debilitated and gradually develop that unstable condition of their red blood corpuscles that may ultimately end in an attack of Black Water Fever.

In the majority of Europeans on the West Coast of Africa 5grains of Quinine per day are sufficient to prevent any great danger from Malaria.

Malaria, and I believe that the prevention of Malaria is the prevention of Blackwater Fever.

For prevention of Malaria Five grains of Quinine a day is the maximum dose one can expect any European to take without great risk of Dyspepsia and its subsequent debility.

Any European in whom 5 grains of quinine daily is insufficient to prevent the continuous sporulation of Malarial parasites , is a person unsuitable for life on the West Coast, and it cannot be too strongly urged that once a European has had Blackwater however slightly he should not again return to West Africa.

I must apologise for having taken up so much space over these few cases. I trust however that others with similar or dissimilar experiences of Blackwater will come forward and describe them

The chart shows the daily rise of temperature during the haemoglobinurea and the relapse of the haemoglobinurea after the iron arsenic &quinine tabloid.

THE MEDICAL SUPERINTENDENT LEPER ASYLUM TO THE COLONIAL SECRY.

COPY.

MUCURAPO

December 22.1887.

Sir,

I have the honour to acknowledge the receipt of your letter No. 3306 of the 19th. instant, forwarding, from His Excellency the Governor, a copy of correspondence between the Colonial Office and the Royal College of Physicians on the subject of Leprosy, also a copy of the Barbados Agricultural Reports of Dec. 6 containing a letter on the same subject.

My views on the question of contagion and heredity, and on the subsidiary question of communication of Leprosy by vaccination have already been stated in detail in two official letters of July 14 and a few days later. They have also been published in a more condensed form in the British Medical Journal of August 20. and September 17.

In the last mentioned Journal I stated that I was prepared at any moment to change my opinion in the light of a dawning science. Hitherto however I have not been able to find scientific ground for believing in the contagion of Leprosy.

Since

Since the date of these letters some facts have come to my knowledge which show still further the very unsettled state of the subject.

1. Bordoni Affeduzzi working at Turin says he has succeeded in cultivating the bacillus of leprosy. (Published in Kock and Flugge Zeit. Schift fur Hygiene Sept. 1887)

My own culture experiments so far have been negative.

2. Hanson in Norway has probably succeeded in inoculating rabbits with perfectly fresh leprous material (Lancet Aug. 27 1887 p. 429)

3. In a letter received by me from a specialist in London, by last mail, occurs this passage:-

"I hear privately from Mr. Watson Cheyne that a "gentleman in Berlin who had inoculated rabbits "with leprosy, and had considered that the "inoculation had had no effect, killed some of "them after a year and found to his surprise "that leprous nodules were beginning to develope "in the abdominal viscera. These facts have not "yet been published, but Kock assured Mr. Cheyne "of their accuracy and that he himself though "incredulous at first, believed that the "inoculation had taken effect."

4. On Dec. 16 (by a most strange coincidence the very day I received the above letter) I made a post mortem examination of a dog which I inoculated with leprous material on April 5 1884 - three and three quarter years ago. I found no local result, but in the liver and spleen I found nodules decidedly suspicious in character. My own impression, after miscroscopic examination, is that they are not

leprous

leprous, for I have failed to find the bacillus: I have however forwarded pieces of the nodules to London by this mail, in order to have the advantage of another opinion.

I quote these four statements to show how actively work is going on in different parts of the world, and how much may come to light in the short space of three or four months.

Of course all this work required confirmation and as I said before I see no sufficient reason, as yet, for accepting the theory of contagion.

The letter of Arch-deacon Wright, copied into the Barbados Reporter I had already read in the "Times" of Nov. 8. which was sent out to me two mails ago. It is strange that though this letter is being widely quoted and copied, the very able and moderate leader in the "Times" of the same day does not receive the same share of attention. The well known fact is there pointed out that leprosy had died out over a great part of the Continent of Europe; and it is further stated that another reason for the apparent increase of leprosy is the increased attention which has been paid not only to leprosy but to sufferers from disease of any kind,

"insomuch that cases which a few years ago might
"easily have escaped observation are now certain
"to become matters of public interest and comment"

The writer of the leader goes on to say that the number of cases which have been seen in England in the last fifteen years is some fifty or sixty, and that in every instance the disease has either been

inherited

inherited or was originally contracted in some other Country.

I notice that Dr. Bernier is quoted as saying that lepers are constantly received in the St. Louis Hospital in Paris. During my course of study at this, the leading skin hospital in Paris and indeed in Europe, in which I attended Dr. Bernier's clinique among others, I only saw one Leper. He had come from abroad.

The rapid increase of leprosy in the Sandwich Islands is certainly difficult of explanation, but, judging from a bundle of reports which I received from Honolulu last May, medical opinion, even in Hawaii itself is very devided. The question of contagion is only a relative one after all. If eventually it is proved that leprosy is caused by a bacillus, the point to be determined will be whether the bacillus is more easily derived from another individual than from air water or other sources. Cohnheiun has proved that tubercle bacillus are daily entering the lungs of every town dweller, yet only a certain number become phthisical, and these in consequence of special depressing or damaging influences. So with leprosy: it is possible that the leprosy bacilli is constantly entering the body, but there must be ther other factors to determine who shall become lepers.

To return to the subject of the correspondence I think the weight of evidence at present fails to show any scientific ground for insisting on compulsory segregation.

I have &c.

(Sd) Beaven N.Rake.

The Surgeon General.

THE SPREAD OF CANCER AMONG THE DESCENDANTS OF THE LIBERATED AFRICANS OR CREOLES OF SIERRA LEONE.

By

DR. W. RENNER, M.D.,
SIERRA LEONE.

1913?

[1910]

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THE SPREAD OF CANCER AMONG THE DESCENDANTS OF THE LIBERATED AFRICANS OR CREOLES OF SIERRA LEONE.

By W. Renner M.D.
Sierra Leone

I have been rather struck within recent years with the increasing number of cases of Cancer of various organs especially of the breast that have, in the course of my practice, come under my observation, and this particularly so, among the descendants of the liberated Africans commonly called Creoles, who form the bulk and principal portion of the population of the Colony proper.

2. In consequence of this, I have been induced to again look into the returns of cases admitted into the Colonial Hospital as well as into my private case book, to see how far I would be justified in speaking of an increase of this disease among the Creoles, its apparent rarity among the resident aborigines in the Colony and in the Hinterland of Sierra Leone, and how far this apparent increase is due to causes which may be traced to the influences of Europ ean civilization and the adoption of European mode of living.

3. In pursuing the investigation of this subject, I would first consider the case of the resident Aborigines in the Colony and in the Hinterland. From the fact that this disease is rarely seen or met with among the hundreds of female aborigines who are treated regularly every year in the Colonial Hospital, and that the Medical Officers of the Protectorate Districts, especially those who are stationed in large towns where there are established Dispensaries at which the natives have been encouraged to attend for treatment, have in their official returns not shown the presence of new growths among their patients, we can safely assume that Cancer as a disease is very rare among the Aborigines. This is however only fairly but not altogether satisfactory as there may be cases which are kept in the back ground by the people and which even when on his patrols in the District

the

the Medical Officer cannot get hold of. Therefore in spite of the non-entry of cases of cancer in the official returns, I would rather not say that the aborigines are immune from the disease, but that the disease is apparently rare among them.

4. With reference to the Creoles in the Colony proper an examination of the records of Hospital cases and those of Medical Practitioners would show that within the last forty years Cancer, as a disease, has been spreading among them.

5 In 1900, I endeavoured in a paper entitled "Prevalence of malignant disease among the natives" which formed the appendix to the Colonial Hospital Medical Report, to tabulate cases of malignant new growths that were admitted into the Hospital within the years 1870-1900, a period of thirty years. Out of 22,453 cases of all kinds treated in the Hospital, only twenty cases of malignant disease were recorded. These cases were simply described as "Malignant Tumour", "Carcinoma", "Sarcoma", "Malignant Growth", but without any details being given as to the particular nature of the disease or of the organ or tissue invaded.

6. To fully illustrate my meaning I append an extract from the paper showing in decades the number of cases treated and the number of malignant growths noticed at each period.

3

Period.	Total number of cases treated.	Nature and number Malignant Growths noticed & treated.	
1870-1879	6,509	Cancer of th Breast	2,
		Sarcoma	1.
		Malignant disease of Jaws.	1. = 4.
1880-1889	5,334	Cancer of the Breast	2.
		Cancer of the Liver	2.
		Carcinoma	1.
		Malignant Disease	1. = 6.
1890-1899	9,392	MAlignant Growths	2.
		Malignant Tumour	1.
		Cancer of the Liver	1.
		Epithelioma	1. = 5.
~~Grand~~ Total			= 15.

7. If to this third period (1890-1899) be added the 1,218 cases treated in 1900, and among which were:-

Epithelioma of the lower lip	1
Scirrhus of the Breast	2
Cancer of the Pancreas	1

there would be for the period 1890-1900, 10,610 cases treated including:-

Epithelioma of the lower lip	1
Malignant Growths	2
Malignant Tumour	2
Epithelioma	1
Cancer of the Liver	1
Scirrhus of the Breast	2
Cancer of the Pancreas	1

8. Tabulating the period under review the proportion of disease of malignant nature to all the other diseases treated shows

shows clearly as follows:-

Year.	Total patients treated.	Diseases malignant.
1870-1879	6,509	4
1880-1889	5,334	6
1890-1900	10,610	10
	22,453	20

9. Between the dates 1900-1909, there were 10,163 cases admitted into Hospital. Of these there were:-

Carcinoma of the Breast	10
Adeno Sarcoma of the Groin (Recurrent)	1
Do. Do. " Breast Do.	1
Carcinoma of the Uterus	3
Papilloma of the Bladder	1
Carcinoma of the Rectum	3
Sarcoma of the Shoulder joint	1
Chondrosarcoma of the upper Jaw	1
Carcinoma of the Oesophagus	1
Mellanotic Sarcoma of the foot (Recurrent)	1
Epithelioma of the Tongue	1
Sarcoma of the Arm	1
Do. Eye	1
	= 26

These growths, that is those collected between 1900 and 1909 have been verified by microscopic examination at the Cancer Research Institute of the University of Liverpool.

10. I have in compiling this return not taken in any figures from the Princess Christian Mission Hospital of which I am one of the Consulting Surgeons, as these records are not available having been destroyed last year (1909) by a destructive fire which consumed the entire building. But I am in a position to say that a large number of cases was subjected to operation by me and the other Surgeons of that Institution.

11. The above figures are interesting as indicating the

presence

presence of cancerous and other malignant growths in the descendants of the Liberated Africans or Creoles in this Colony. I admit that it is not safe to base definite conclusions as to the extent or the increase or spread of this disease upon statistics alone and, therefore, I do not consider the figures in the last preceding Table as accurately representing the extent of the increase or spread of the disease. For a number of cases must exist which, unfortunately, the general Practitioners do not come across and there must be many sufferers who do not seek the aid of the Hospitals. These latter are more or less in the hands of the native fetish doctors who lull them with the false hope of cure by stating that their disease is the result of witchcraft and that they (the fetish doctors) are the only persons capable of treating their disease and giving them the desired relief. Some of these patients have remained in their hands trusting still in their occult powers until either becoming hopelessly incurable, the fact at last dawns in their minds that they must seek other aid, or they arrive at that stage where nothing remains for them but to long for that relief from their terrible sufferings which death alone can give.

12. The existence of Cancer and other malignant growths among the Creoles, and its absence or rarity among the Aborigines are due in my opinion to the civilized habits of, and the civilising influences operating upon the former, and to the primitive mode of living of the latter. The Creoles have adopted the mode of living, the food and dress of the European - have to a great extent discarded the simple food of their forefathers, have been craving for and indulging in preserved and imported foreign food, - have substituted the European for the natural African environment and entailed on themselves in their pursuit for wealth and luxury the anxieties and worries incidental to civilisation and consequent liability to premature decline.

13. Reverting to the question of food, while the Creoles in a Tropical Country like Sierra Leone consume a large

quantity

quantity of meat which is absolutely unnecessary, the Aborigines, - the Timnes, the Mendis, Kurankos, and others, confine themselves mostly to grain and vegetables, which really should form the bulk of the dietary of the native in the Tropics, and eat very little of flesh and meat with the result, that the latter are on the whole healthier and are free from the tendency to engender and propagate foreign diseases.

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~~14.~~ On the question of the relation of diet to disease, some Medical Authorities have asserted that Butcher's meat is undoubtedly one of the means by which Cancer is propagated. Although I cannot yet for want of sufficient data absolutely accept this theory, yet the fact that cancerous growths are common among the well-to-do Creoles who can with ease afford meat and other articles of European diet as portion of their daily dietary, strongly appeals to me as lending a support to that theory.

~~15.~~ Epitheliomatous growths are very rare among both Creoles and Aborigines. By this I mean, Epithelioma of the lip, tongue, cheek, &c., such as have been met with among the negroes of the West Indian Islands, the Southern States of North America and in India, and are considered to be the result of local irritation. This rarity is possibly due to the fact that the Creoles are not great smokers of clay pipes and are to a large extent the possessors of good teeth. I have however noticed a marked tendency to degeneration of the teeth among all classes from children upwards, - a condition which was certainly absent twenty five years ago and which I put down to the use of European articles of diet as Saccharine foods, preserves, and sweets. If this tendency is not checked I would not be surprised if in another fifty years Epithelioma of the tongue should become as common a disease as Cancer of the Breast in this Colony.

~~16.~~ But in the Aborigines also there is a marked absence of these Epitheliomatous growths and yet both men and women and even children are great clay pipe smokers. Their teeth are however 9

however kept beautifully and pearly white and that tendency to decay which has been noticed in the creoles is altogether absent in them.

17. The presence of Cancer of the Breast among the creoles of this Colony nullifies, in my opinion, the theory advanced by Dr Hersey, Principal Medical Officer of British Central Africa in his Paper on "The rarity of Cancer among the Aborigines of British Central Africa" published in the British Medical Journal of December 1, 1906 in which he states:-

"Of the various theories advanced to explain the origin of "cancer the theory that it is purely local in origin appears "to have obtained the widest acceptance. Premising that cancer "of the breast is local in its origin that for physiological "reasons the breast is subject to great and sudden alternations "in its functional activity both at puberty and during pregnan- "cy and lactation, it appears to me that any disturbance of the "physiological functions of the breast during lactation would "act as an exciting causative agent of no small value in the "production of cancer. All native children have of necessity "to be brought on the breast . In civilized communities even "among the poor, there is a marked and growing tendency to "wean infants at the outset. IN consequence of the physiolo- "gical increase of ~~blood~~ supply of blood to the breast during "lactation, the artificial suppression of the latter may act "as a powerful exciting cause in the production of cancer. "If this is not so how is it to be explained that aboriginal "women, who are always compelled to suckle their young are so " immune from cancer of this organ that it is practically "unknown amongst them"?

~~18.~~ To this I have to observe that the cases of cancer of the breast which I have enumerated above as occurring between the years 1900 - 1909, have all come within my personal observation and knowledge. They were all multiparous women who had nursed their infants from twelve to eighteen months each time

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time before weaning and they had become again pregnant after an interval of from three to twelve months; so that the mammary glands in them were in a state of constant excitement and activity. The physiological activity of the mammary glands is not in my opinion a reasonable theory to account for the apparent immunity or rarity of Cancer of the Breast amongst the native women of British Central Africa. The presence and increasing number of this disease amongst the civilized native women descendants of the Liberated Africans in Sierra Leone must point out that we must look for some other means brought by their contact with and embracing of civilization.

~~19.~~ The important fact of hereditary predisposition has not been lost sight of, but so far I have been unable to trace the existence of any growth whatever in their family history of the majority of the cases examined. In one case however there was distinct hereditary origin for which I can vouch as I treated the grandmother for Cystic Adeno Sarcoma of the right breast, the grand-daughter for Carcinoma of the Uterus and the great-grand-daughter for Recurrent Adeno Sarcoma of the Groin.

~~20.~~ It may be interesting as bearing upon the question of civilization as a contributory cause to the origin and spread of Cancer and other diseases among aboriginal population to compare the history of two African peoples on the West Coast of Africa. The Sierra Leone Creoles and the Fantis or natives of the Gold Coast.

~~21.~~ In the case of the Sierra Leone Creole, his ancestors of various tribes and languages, different habits and social customs and of various temperaments, dispositions and idiosyncrasies had, as an outcome of the suppression of the Slave Trade and Slavery being and located in Sierra Leone. Possessing nothing of the elements of a nation in common, they could not combine and unite and so become a distinct entity and evolve if not a language, a common social custom and mode of living best suited to their state and condition as children of the Tropics. Their eagerness in their ignoranceto forsake their

own

own native social customs and to embrace and adoptt the language, dress, mode of living of their benefactors - an eagerness largely born of gratitude - was only paralleled by the earnest solicitude on the part of their benefactors to help them to throw off what was "native" and which was unwisely regarded by the latter as relics of barbarism and heathenism - and assist them in the adoption of their language, customs and mode of living. As ignorance and tribal differences slowly gave way to education and intermarriages, the tedency not to assimilate what was best in their native brethren or to unite, but to imitate, adopt and practise what was foreign, grew and grew until by the process of evolution the Sierra Leonean has become more English than any other people on the West Coast of Africa. The initial cause of this was the absence of a common nationality, tribal origin or clanship. For how different would the Creoles have been if their fore-fathers were all from the same country and of the same tribe. They would have preserved their language, traditions, customs and habits and have been better ableto resist, for a considerable period at least, the inroads of certain civilized habits and influences - the adoption of which has proved detrimental to their very existence.

22. Now let me take the case of the Gold Coast and see how far European civilization has affected a large majority of the people there who are one in language, habits and customs. These people have been in contact with European nations for five centuries. Their country was visited by the Phoenicians; they have had at various periods and for more or less long duration constant intercourse with the Danes, Dutch, Portuguese, Germans and English who have at intervals and in succession held and fortified their Coast towns. Yet after so many centuries of close contact with Europeans they have by virtue, of their race instinct, and race-unity resisted as one people the inroads of European civilization into their social life, and as a result, we find the absence or rather rarity of Cancer and

other (10)

other new growths amongst them. How long this condition will last remains to be seen. Native customs die hard, but with the establishment of permanent peace in their country, the opening of their country by Railways, the constant influx in increasing numbers of Europeans exploiting the country and working the Mine the equally rapid increasing number of the offspring of such Europeans as a result of intercourse with the native coloured women, descendants of older decades from Danish, Dutch, German and English forefathers, and with the other non-hybrid native women, the liberal and progressive system of education now being established all over the country, I am very much afraid that the social habits and native customs of the Gold Coast people will befor long disappear before the mighty onslaughts of European civilization, and with such disappearance arise those conditions, which I believe, as the outcome of this civilization are the origin of Cancer. I entertain this apprehension also in regard to the Nigerians and other Native Territories which are are being exploited by, or made as a home for, the white man.

~~23.~~ I have in the above sketch attempt simply to give facts and state my impressions based on those facts. I haver refrained from making definite conclusions or positive statements and necessarily so, where the field of investigation is so wide and my opportunities for making researches are so limited. If however this sketch should serve to induce others to collect data and prosecute researches as opportunity offers so that definite conclusions may be arrived at and should the Creoles to reflect and alter their present mode of living so as to avert becoming the victims of this dire disease which up to the present baffles medical science, I shall not have made the attempt in vain.

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The International Plague Conference. (Reuter's Telegram) April

Mukden, ~~April~~ 15th 1911

The second week of the International Plague Conference has now drawn to a close, and it is probable that another week will see its deliberations almost concluded. Many extremely interesting topics in regard to the recent epidemic have been discussed. Perhaps one of the most important was the question of the infectivity of the breath in cases of pneumonic plague. ~~Dr~~ Dr. Strong, the American Delegate read a very interesting paper on this question. Infectivity of the Breath He recounted the results of a series of tests made by him in Mukden. Agar plates, previously sterilised, were exposed before plague patients at various distances for various ~~purposes~~ periods, and under various conditions. Dr Strong found plague bacilli on the plates in almost every instance in which the patient had coughed during the period of exposure, but his experiments demonstrated that breathing alone did not result in plague bacilli being scattered into the air. His conclusions may be summed up as follows: during normal and dyspnoeic respiration of primary pneumonic cases, plague bacilli are not usually expelled by means of the expired air; during coughing of such cases, even when sputum visible to the naked eye is not expelled, plague bacilli in large numbers may become widely disseminated into the air surrounding the patient. He expressed the opinion that infection might be carried for a distance of several yards by coughing, that it might also be disseminated by forcible talking, and that

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doctors and nurses in attendance on cases of plague pneumonia should protect themselves with overalls, masks, and glasses, and sterilise their clothing immediately after leaving the wards.

Infectivity of Corpses

Professor Zabolotny, of St. Petersburg, spoke upon the question of the infectivity of corpses. In corpses exhumed three months after death, during the recent epidemic, he had found living plague bacilli, a fact of tremendous importance in connection with the possibility of a recrudescence of plague in Manchuria, through the medium of marmots and other rodents.

Prophylatic Inoculation

~~Probably~~ The most controversial subjects so far discussed are Prophylactic inoculation, and Serum Therapy, and up to the present no definite conclusions have been arrived at upon either. In regard to the first Dr. Strong stated that his conclusions were that vaccination in plague with a properly attenuated culture, is as harmless in human beings as vaccination against small-pox; during the present epidemic prophylactic inoculations by dead cultures have been very frequently shown to be ineffective, some individuals inoculated three times in this manner having succumbed to plague; the Conference should thoroughly investigate the question of true vaccination against plague; a suitable culture for such vaccination should be recognised internationally as a standard culture, and placed with some well-known bacteriological institute from which it can be obtained at all times; vaccination with such standard culture should alone be countenanced.

Professor Galeotti, the well-known Italian scientist considered vaccination with living cultures too dangerous, and advocated the use of the bacteria nucleo-proteid, prepared by Professor Lustig and himself, as a safer, and equally efficacious prophylactic.

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Serum Therapy

Serum therapy provoked even more discussion. In the course of it, cases were mentioned in which plague pneumonia patients had been given as much as 800 and even 1,000 c.c. of anti-pest serum, without any effect other than prolonging life for a day or two. The results of investigations of this subject are most disappointing. By experiments with animals, Dr. Martini, one of Koch's assistants, has shown that unless serum treatment, in large doses, is given immediately after infection, it is unlikely to prove effective in cases of plague-pneumonia. He had found in the case of rats that serum to the amount of about 1/60th of the animal's weight was necessary to ensure protection and that even then the protective effects only lasted about five days.

sub committee appointed

A sub-committee has been elected to sift the evidence in regard to the various prophylactic and serum treatments advocated by different schools, and it is hoped that before the Conference disperses some definite recommendations will be made. It is worth recording that the Japanese claim to have cured five cases of pneumonic plague at Dairen (Dalny) with a serum prepared in Tokio, but the tests by which they confirmed the diagnosis of plague are not generally regarded as sufficiemtly exhaustive.

Pathological Anatomy

The following are the conclusions of a most exhaustive and comprehensive paper on the above subject, prepared by Drs. Strong and Teague, based upon the result of twenty-five autopsies conducted by them.

Epidemic plague-pneumonia results from inhalation, the

the primary point of infection being the bronchi. Through the bronchi the plague bacilli reach the lung tissue, and rapidly multiplying there, produce at first pneumonic changes of the lobular type, and shortly afterwards more general lober involvement of the lung tissue. The blood becomes quickly infected, and a true bacteriaemia results in every case. Secondary pathological changes occur, particularly in the spleen brochial glands, heart, blood-vessels , kidneys and liver. The fact that the bronchial glands at the bifurcation of the trachea are always much more severely affected than any of the other lymphatics, argues against the theory that epidemic pneumonic plague is primarily a mmm septicaemic disease, and that the lungs are affected secondarily from the blood. Moreover, in the earliest stage of the disease, the blood may be free from plague bacilli. The condition observed in the trachea, and bronchi, in epidemic pneumonic plague, is pathognomic of this condition alone. From the appearance of the mucous membranes of the throat and larynx, a diagnosis of pneumonic plague may sometimes be made. The mmmmmm tonsils become secondarily infected in pneumonic plague, just as other lymph glands, for example the bronchial ones, become so infected. However, in pneumonic plague, death occurs before any very marked microscopic changes occur in the tonsils. There is no doubt also that the tonsils may become primarily infected in epidemics of pneumonic plague, just as has occurred in sporadic cases during epidemics of bubonic plague. This, however, is not the common mode of primary infection, and in such cases involvement of the lymphatic glands of the neck occurs early in the course of the disease. The fact that the oesophagus was found to be normal in every case examined, constitutes another

argument against the idea of primary intestinal plague infection since in many of these pneumonic cases, plague bacilli must have been repeatedly swallowed in the bronchial secretions and saliva.

In the discussion upon this paper Dr Tang, of Mukden, referred to the cases of one donkey and nine mules which had died with all the symptoms of pneumonic plague, and had infected men who had been in contact with them. Dr Martini explained how, by the passage of a strain of bubonic bacilli through the lungs of several rats in succession, the virulence of the strain was so much increased that after four or five passages rats died in 30 instead of 72 hours. This, he thought, proved, that passage from lung to lung during the recent epidemic accounted for the virulence of the strains of bacilli isolated in Manchuria.

Clinical observations.

Clinical data was the next subject upon the programme. Various speakers referred to the character of the plague encountered in the recent epidemic, but no definite evidence was given in regard to the so-called abdominal type, of which a few cases are said to have occurred. A curious feature of plague pneumonia, confirmed by several observers, is the frequent absence of marked physical signs in the chest, even in advanced stages of the disease. Though the Tarabagan, a species of marmot has been generally regarded as responsible for starting the recent epidemic, Dr Gray, of Peking, pointed out that there was no evidence of any marked outbreak of Tarabagan disease during the past winter, and suggested that the epidemic might have been started by a small local outbreak - of which there have been many examples- near the Manchurian frontier, and that the Tarabagan hunters gathered in the ~~neighbour-~~

neighbourhood, were particularly susceptible to the disease owing to the hardships, and scanty nourishment they put up with during the hunting season.

The Decline Spontaneous.

Evidence given by several delegates pointed to a spontaneous decline of pneumonic plague, quite apart from the precautions adopted to combat the epidemic, the disease having disappeared about the same time in cities where every possible measure was taken to stamp out the infection, and in smaller towns and villages where no modern sanitary measures were employed. A very interesting description was given by Dr Christie, of Mukden, of a woman, who though she never contracted the disease herself, was the means of infecting eleven other persons in the course of fourteen days. So far as can be ascertained, this is the only case in which infection was carried by a person who was not suffering from plague.

In contrast to the scientific side of the discussions was an informal account, given by Dr Aspland, of Peking, of the methods adopted by a Chinese docter, of the old school, in treating plague. This doctor claimed that he had cured himself of the disease, and though he attended upwards of a hundred cases, without effecting a single cure, and without adopting any of the precautions observed by doctors of the modern school, he escaped unscathed. The main portion of his treatment was "slapping", either with the sole of a shoe, or a hempen cord. This drastic remedy, he claimed, drove the disease out of the system.

The TARABAGAN.

Twelve Tarabagans (marmots) were brought down from Harbin

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during the past week, and they were at once taken in hand by Dr Petrie, of the British delegation, who chloroformed them all in turn to search for fleas. An average of four of these insects was found upon each animal. The tarabagan flea is believed to be a new species, as ~~at~~ no-one has reported their discovery hitherto. If it can be proved that they will bite human beings an important link ~~between~~ in the chain connecting the marmot with the Manchurian epidemic will have been forged. Consequently, the single flea so far captured alive is being jealously preserved until it is hungry enough to test whether it will make a meal off man.

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THE MODE OF TRANSMISSION OF LEPROSY.

By

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The mode of transmission of leprosy from the infected to the healthy is a question which has hitherto baffled research. Of the infectivity of the disease no one who has studied its history and has carefully investigated any considerable number of cases, their previous life and social relations, and who considers the evidence without prejudice, can, I think, have any doubt.

In the first place, a word as to heredity. It is exceedingly improbable that an ovum, still less a sperm cell, could contain so relatively large an intruder as a lepra bacillus and yet suffer so little derangement of its great but excessively delicate potentialities as to render successful fertilisation and development possible. This view was first enunciated by Virchow in discussing the germinative transmission of tuberculosis and it has never been adequately refuted. We are aware that the infections of pebrine disease of silk worms, and of Syphilis, are capable of being deposited previous to fertilisation in the parental elements, but in both these cases the progeny, mature or otherwise, show widespread infection and the diffuse ravages of the particular disease immediately, or more usually shortly after, birth. I have had the opportunity on two occasions of examining the tissues of the offspring of leper parents which were either stillborn or had died soon after birth; in neither of them was the lepra bacillus or anything suggestive of its presence discoverable. In well-marked cases of tubercular leprosy

leprosy bacilli may be demonstrated in the ovaries, prostate, seminal fluid and elsewhere in the genital tracts, but careful and repeated search has failed to discover the organism in either the sperm or germ elements. This, of course, is negative evidence, nevertheless it is not entirely valueless. Placental transmission, unless as an accident, also seems highly improbable. The bacilli are at any time very difficult of demonstration in the blood. During acute exacerbations of the disease with the formation of fresh tubercles, fever, sickness and other signs of general infection, they are not so difficult to find. But pregnant females, in my experience at any rate, are remarkably free from these superimposed attacks of acute leprosy, however much they may relapse afterwards. Microscopic examinations of placentae have shewn no bacilli nor lesion attributable to their previous presence.

Children are undoubtedly more susceptible to infection than adults, and as a rule the disease makes rapid and destructive progress in their young tissues. Infection, however, takes place not only by reason of an increased susceptibility in their junior years, but also on account of the unlimited opportunities afforded for its transmission during the early period of dependence and intimate contact of parent and offspring.

Commensal feeding and the fact that the bacillus flourishes in the products of tryptic digestion suggest the possibility of a primary alimentary infection; the bacilli, perhaps, after ingestion, reach the small intestine and find a suitable medium whence they subsequently make their way into the general circulation and so establish the disease. But considering the delicacy of the lepra organism in its initial stages of artificial growth

growth, the adverse conditions it is likely to meet in the gastric and intestinal tracts, the absence, so far as my enquiries have gone, of an early clinical history in consonance with such an infection and the negative results of post-mortem examinations of early cases of the disease as regards involvement of the intestinal tract and mesenteric lymphatics, it seems to me that this mode of transmission of the infection, if it does occur, must be rare.

Inhalation of dried bacilli floating as dust may also be suggested as a probable path of infection. Ulceration, with dense bacillary infiltration of the nasal mucous membrane, is the rule in tubercular leprosy. But again, although an early lesion, it is not initial: in fact, it is not observed until the general infection is distinctly pronounced. Infiltration, nodule formation and ulceration of the larynx and adjacent mucous membranes are very frequent but only as late, or sub-terminal, complications in tubercular leprosy.

Leprotic involvement of the lungs is very uncommon. Clinically I have never known it, and in my last 50 post-mortem examinations microscopic inspection has always failed to discover undoubted lepra bacilli or granulomatous reaction due to their presence, although the tubercle bacillus and the results of its presence were very frequently observed.

In brief, then, we have no reason to believe that the infection of leprosy is transmitted ante-partum and both the mode and the site of primary infection have hitherto remained undiscovered.

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As a result of observations on a large number of cases of leprosy, I was led to direct increasing attention to the skin as the probable site of primary inoculation. Over a year ago, therefore, I decided to conduct my researches in this matter with a view to establishing or eliminating any rôle the commoner and more widely distributed domestic insects might play. I considered the common house-fly (Musca domestica). The stomoxys genus and the haematobia irritans or horse fly are vicious biters and on a priori grounds, more probable carriers of infection, though the latter rarely bites man, but they are uncommon on this island and I determined to work, in the first place, with the more abundant material at hand. Further, I examined the common flea (pulex irritans) also the local varieties of mosquito (culex and anopheles), and finally the bed bug (acanthia lectularia). My methods were simple. The insects were confined for a period of from 24 hours to 20 days in a test tube with a little cotton wool. The test tube was kept in the laboratory incubator. The hungry insects were then placed in a large watch glass with some cotton wool and fixed by adhesive plaster and bandages to the selected site. In the case of flies and mosquitoes a light wire frame with netting was found preferable. An infiltrated area on the posterior surface of the forearm was found to be suitable and convenient. The following table summarises the results of the examination of smears of insects so fed, made within half an hour of feeding:-

70 flies.—	Enclosed over ulcerated leprotic surfaces. Two acid-fast bacilli found in stomach of one fly and one in stomach of another.
80 mosquitoes:—	One contained three acid-fast bacilli.
60 fleas:—	Two contained acid-fast bacilli 2 bacilli in one flea, one in the other.
75 bugs:—	20 contained acid-fast bacilli.

The bugs frequently contained acid-fast bacilli in the alimentary canal. The mosquitoes, fleas and flies very rarely contained bacilli or they rapidly disappeared or were no longer recognisable; indeed, in the cases of the mosquitoes and fleas I ~~hesitated~~ hesitated some time in consideration of the isolated red rods as to whether they were actually bacilli or stained fragments of tissue; I therefore venture no further statements about them. After the preliminary experiments, attention was concentrated on bugs. Lepra bacilli are undoubtedly imbibed by the bug and are demonstrable without much difficulty in smears made from the debris of the intestinal canal. I say lepra bacilli advisedly; they retain their acid-fast staining and shew the morphologic characteristics associated with this organism. No acid-fast bacilli were found in unfed bugs. They are usually isolated, but I have counted as many as 12 in one field. They do not readily disappear, but can be found up to 16 days after the insects are fed. Acid-fast bacilli answering all characteristics of the lepra bacillus were also found in smears made from the tissue juices and circulating medium of bugs fed on lepers, in smears of the macerated head and of the proboscis (5 days after feeding) and, finally, I found a bacillus once in the excreta deposited on the inner surface of the test tube.

On the 2nd of May last, some bugs were fed on a patient with well-marked tubercular leprosy who at the time shewed a fresh crop of nodules, was feverish, and, in fine, exhibited all the signs of an acute exacerbation of the disease. When the insects had been affixed to the patient for 8 hours they were removed and found distended with blood. Again on the 15th day of the same month the

same

same insects were re-applied to the same patient for a few hours. Next morning, the 16th, one insect was found dead. I made a smear of the stomach contents and also of the body tissues well apart from the intestine. The former contained a few bacilli; in the latter, stained at the same time and by exactly the same method, field after field displayed squadrons and platoons of acid-fast bacilli. Their individual appearance differed considerably, however, from the classic lepra organism. They were shorter, some of them almost coccoid; they were more homogeneous in staining, displayed no granularity, had occasionally a median constriction, occasionally a terminal clubbing. The great majority were arranged in groups, the individuals of the groups showing a tendency to adhere in parallel formations, like the cohesion of floating logs. They appeared to me to be young lepra bacilli in full prolific activity - not having yet assumed the senile or degenerative forms usually observed in the impacted bacillary masses of human tissues.

Owing to the recent work of Drs:Clegg and Duval, who have shewn that the presence of the amino-acids is essential for the artificial cultivation of the bacillus, I was able to compare these bacilli with others artificially cultivated. They were very similar. In some, though slight differences were recognisable, such differences were less than those observed in comparing different bacilli of the same slide. Careful comparison left little doubt in my mind that the organisms from the bug and those from the cultures were identical; it seems to me probable that the bug actually died of acute leprosy.

Spiker!

I have recently endeavoured to demonstrate the transmission of the bacillus by fed bugs to healthy tissues. Bacilliferous insects were applied to shaven areas on guin

guinea

guinea-pigs, rabbits and monkeys, and to the clean, unaffected, arms of "arrested" maculo-anaesthetic lepers. Small portions of tissue surrounding the bite were excised immediately the insect left its mark and at varying times afterwards, and sections cut and stained. The results have, in the case of the lower animals, been uniformly negative. On one anaesthetic leper a small papule appeared at the site of the bite. This papule was excised but no bacilli were found in it; a definite bacillus was, however, found a considerable number of sections away from the edge of the papule, but whether it had been injected by the insect or had been deposited there previously or independently it was impossible to decide.

On consideration it will be apparent that one would be sanguine to expect any marked result from the application of these leper-fed bugs. Guinea-pigs and rabbits are uninoculable with leprosy, and leprosy bacilli, so far as my investigations have gone, rapidly disappear if injected into their tissues. As regards maculo-anaesthetic lepers, it seems to me that during most of the time they suffer from the disease they are partially immune and only their peripheral nervous system affords a suitable soil for the growth of the bacillus; - if the disease be indeed "arrested" even this partial susceptibility disappears

Reviewing the foregoing data, I venture to submit the following conclusions:-

(1) That considering the enormous numbers of lepra bacilli in the infiltrated or ulcerated skin and nasal mucosa of an active "tubercular" leper and the ingestion of bacilli by certain insects, direct contact and transmission by flies, fleas, mosquitoes or other insects are possible modes of spread of the disease; but such infection, if it ever does take place, is accidental and exceptional.

That

(d) That having found acid-fast bacilli answering as far as our imperfect tests permit to the characteristics of lepra bacilli in a considerable proportion (about 30 per cent. of specimens of acanthia lectularia up to 16 days after feeding on lepers, there is reason to believe that this species of insect constitutes a very important agent in the spreading of leprosy.

Unfortunately this view can be proven with finality only by the application of bugs previously fed on lepers to the persons of healthy individuals with the resultant development of the disease, unless, indeed, it be found, as there is some reason to hope, that certain monkeys and anthropoid apes are susceptible of inoculation with leprosy.

In connection with the foregoing conclusions, I quote in brief a few details, obtained without leading question or suggestion, of the early history of a patient - an European of intelligence and with a clear memory of the onset of the disease - H.M.Number 1039, aged at the time eleven years (whose father had been removed as a leper to this Asylum some time previously and who the son used frequently to visit) observed for the first time in the year 1901 a small red papule under his chin. It enlarged slowly, becoming in area, to use his own simile, about the size of a sixpenny piece. It was indurated and resistant to treatment. It remained thus, slightly raised, hard and of a reddish hue for two complete years before any further symptoms appeared. In the year 1903 he noticed for the first time a bluish puffiness of his cheeks, later slight thickening of his eyebrows, and a diffuse but moderate swelling of the entire countenance. Towards the end of the year tubercles began to appear on his face. Thereafter,

tubercles

tubercles continued to appear at irregular intervals on his face. The glands of his neck became involved. From this time onwards the tubercles and other typical signs were of universal distribution and shewed a diffuse systemic infection.

The initial pimple, and later tubercle, on the chin would appear to have been the primary lesion, probably caused by the bite of a leprosy-infected bug.

In the communities where it is endemic leprosy is notoriously a disease of the dirtier classes. It is not, however, necessarily associated with poverty, except in so far as this is a concomitant of squalor. In South Africa, although leprosy is more prevalent among the lower strata of the population it is by no means confined to them. Of the cases in Europeans a considerable percentage is derived from families in which poverty and privation are unknown, but where the practice of the elements of domestic hygiene and cleanliness is in abeyance. Cases do occasionally occur in persons whose habits are, in general, cleanly, but on enquiry one sometimes finds that lapses from such habits have occurred, affording opportunity for the transmission of the infection by the means suggested. There is in this Asylum, for example, a patient who once held a position of responsibility in a crack English regiment. He had always been of scrupulously cleanly habits when circumstances permitted. He states, however, that under the exigencies of campaigning in India and later in South Africa he frequently slept and retained for temporary use when no others were available blankets belonging to and previously used by natives.

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ON ENTERIC FEVER IN SOUTH INDIA.

by

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With plates.

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ON ENTERIC FEVER IN SOUTH INDIA.

A Thesis for the Degree of M.D. Dunelm.

by Percy Edward Turner, M.D., B.S. Dunelm.

M.R.C.S.: L.R.C.P. Lond: D.P.H. Oxon.

My attention was arrested during a perusal of the "British Medical Journal." in 1907, by a passage(1) in the review of Major Ernest Roberts' "Enteric Fever in India and in other Tropical and Sub-Tropical Regions," as follows:-

> "Major Roberts does not regard the native
> "of India as the source of the disease
> "except when in contact with British troops...";

inasmuch as in that part of India with which I am personally most acquainted, the native state of Travancore, the opposite may in my experience be said, viz. that the native inhabitants are liable to this disease and that without contact with British troops (of whom there are none in the state) or other Europeans (of whom there are very few).

And in the consideration of this subject I propose, as far as evidences falling under more than one heading will allow, to put my thesis thus:-

I. That natives of India do suffer from Enteric Fever and that without contact with Europeans.

II. That the attacks are frequently quite typical, as shown

(a) by their clinical symptoms and progress,

(b) by bacteriological evidences

(c) by their ability to give rise to typical cases of the disease in Europeans.

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III But that in spite of the commonness of sources of infection, many natives appear not to contract the disease and that this is probably due to an acquired immunity.

1 - Many natives in India do suffer from Enteric Fever.

That Europeans in India suffer from this disease is only too well known whether through the writings of such authorities as Sir Patrick Manson, who says:-(2)

"Typhoid Fever .... is very common among them
"(young Soldiers and civilians) during the first
"two or three years after their arrival"

or by such statistics as the following, collected by E.Roberts:-(3)

"Enteric Fever among European troops in India,
1888 -1898, in mean ratios per mille;
Attack 23.6 Death 6.29 "

It is also very common amongst Indian born Europeans, but with this difference as compared with temperate climates that the incidence according to Rogers' statistics(4) is four times as great among children under 15 and four times as little among adults over 25 years of age; and he further concludes that the explanation of

"the comparative rarity among the large adult
" population of the Indian Army " -- 0.2 per
"mille only -- " is that this remarkably low
"age indidence also applies to the indigenous
"population".

This

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This is identical with the theory put forward with regard to Enteric Fever in Egypt but strongly opposed by Sandwith[5], who gives the following statistics, which it is interesting to compare with those given above for India for 1888-1902:-

"English Army in Egypt attacks of Enteric Fever per mille 25.0

"Egyptian " " " 2.0 "

and says:-

"I have often heard it stated that no adult "Egyptian suffers from this disease, and the "erroneous theory has been propounded that most "Egyptians have suffered from this fever as "children, thus procuring an acquired immunity "among the adults. I think I may dispose of "this theory quite shortly by stating that I have "performed, or assisted at, the post-mortem "examinations made upon several hundred children "under the age of five years .... but never in "one single case have we ever found pathological "evidence of Enteric Fever, while those cases "which I have carefully watched during life, "to elucidate this very question, have never dis- "played clinical symptoms of this disease, nor "answered to the serum test."

This same argument from negative post-mortem data was formerly used in connection with adult natives of India as witness Dr. Crombie[6] in 1893:-

The

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"The native of Calcutta is practically exempt
"from the disease ... the argument that cases
"occur but are not recognized must be very greatly
"weakened by the paucity of post-mortem records
"of Enteric ulceration of the bowels .....
" I am not aware that I have ever myself seen
"a case of Enteric Fever in a native of Bengal
" and my experience includes a period of seven
"years when... I made not less than 300 post-
"mortem examinations per annum";

but by 1899 positive evidence on the other side was forthcoming, as from A.Buchanan,(7) who recorded 25 cases in the Nagpur Jail since 1894 with several post-mortems, and in 1900 from Surgeon-Major Harris,(8) who had

"seen cases of typhoid fever amongst natives
"in Calcutta, and other places and verified the
"diagnosis in some cases post-mortem";

while in the same year according to the Madras Sanitary Report:-(9)

"Typhoid Fever is an acknowledged factor in the
"mortality of natives ... the Malabar district",

and this brings us near to that part of India mentioned in the introduction to this paper, to wit, Travancore.

This is a native State at the southernmost part of the Malabar coast, including indeed the very southernmost point, Cape Comorin, about twelve miles from which The Salvation Army has a Central Hospital, of which I have been for the last eight years in charge, the patients coming from all parts of South Travancore and beyond and from all classes of native society, the great majority being Hindus.

It

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It is a part of the country untouched by railways, has no British troops and, outside the capital, hardly any Europeans at all other than a few Missionaries of various Societies. If, therefore, Enteric Fever be found, and particularly in the villages, it may safely be considered to be indigenous and not derived from European sources.

II Evidences for the attacks being truly Enteric.

(a) Between January 1st 1901 and March 31st 1908, 45,705 new Out Patients attended the Hospital, and the general impression left upon my mind is that Enteric Fever is certainly found among them, more commonly amongst young people but also in their elders.

Out Patient observations are however necessarily incomplete, but I have been able also to find more certain evidence in 17 cases, in natives of India, who during the last three years of the period indicated were admitted to the wards as In-Patients with an undoubted diagnosis of Enteric, and omitting some others, doubtful cases, whose records for the sake of accuracy were headed only " ?Enteric".

The ages of these seventeen were as follows:- 9,6,12,35,8, 38,30,4,6,2,20,10,13,13,5,14,9, i.e. Thirteen (or fully three fourths) were under 15, one between 15 and 25 and three were over 25.

The clinical signs and symptoms commonly shown by these cases are the following:-

(i) Gradual, indefinite onset, commonly with headache. Occasionally in a malarial subject a malarial paroxysm is the first sign of an Enteric attack; but vigorous

treatment

treatment of the Malaria will generally allow the enteric course to be thereafter seen.

ii. Continued high temperature for some days. On this sign Rogers[10] says:- "The period of high continued fever is usually quite characteristic "and of great diagnostic value. By 'high continued " 'fever' I mean a temperature keeping persistently " above 101° F..... and not varying more than 2° F "for at least 48 hours."

iii. Gradual fall of temperature to normal, the total length of pyrexia being three or four weeks.

iv. Tumidity of abdomen - a valuable sign.

v. Constipation or diarrhoea.

The avarage Indian is very regular in his habits, usually"going out" at dawn each morning and sometimes at dusk each evening also.

vi. A pulse slow relatively to the pyrexia.

vii. General condition.

Some get into a truly "typhoid" condition, but the majority show a less re-action on similar lines.

viii. Liability to recrudescence or relapse, especially following indiscretions in diet.

ix. The Diazo re-action.

(There are two signs of value in temperate climes which are not usually available in India:-

1. The typhoid rash is not distinguishable in the darker-skinned; only occasionally in the comparatively fair skins of the higher castes are faint purplish spots visible.

2. Enlargement of the spleen is commonly not a trustworthy

trustworthy sign, being so frequently met with as a consequence of malaria, unless one happens to know from some examination of the patient shortly before the presumed typhoid attack that there was no previous enlargement of the organ.

Remarks on appended charts of some cases.

A. In a girl of 12; A severe attack of 31 days duration; a "typhoid" condition for some time, but ending in recovery.

B. In a man of 35 : shewing the typical low pulse rate.

C. In a man of 38 : typical loose stools.

D. In a girl of 13 : brought to the Hospital in an almost "typhoid" condition, with a history of 25 days fever; at the end of a week the evening temperature had got down to 38°C and the girl seemed much better, whereupon the parents, frightened at her low diet, took an opportunity to sit her up in bed and administer a good feed of pounded rice -- with a resultant recrudescence lasting 19 days.

E. In a boy of 12 ; not in the Hospital; a mild case lasting eighteen days only to a normal evening temperature but showing a temporary rise on the following day subsequent to a meal of rice and curry.

The only fatal case among the seventeen In-Patient cases was in a girl of 4, who apparently contracted the disease either from her mother or from a great-uncle, whose cases are mentioned later in connection with one method of transmission of the disease.

(b) Being far away from any bacteriological laboratory, we have hitherto not been able to confirm these clinical evidences by the serum test, but since 1901 there has been abundant proof forthcoming in other parts of India that the application of this means and diagnosis gives positive results. In that year R.H.Elliot (11) verified thirteen cases by it among natives in the General Hospital, Madras and in the following year Leonard Rogers (14) published the conclusion to which he had come that

> "So far are natives removed from being immune to
> "enteric that the disease is really quite common,
> "among them, a continuous series of cases having been
> "recognized by means of the serum test during the
> "last five months in the Medical College Hospital,
> "Calcutta";

whilein 1908 the last named writer says:-

> "During the last few years I have repeatedly obtained
> "positive serum tests for typhoid in high dilutions
> " of 1 in 100 or more":

so that I have no doubt but that when we have facilities for carrying out similiar observations in Travancore like results will be obtainable.

(c) It is generally accepted that these infectious diseases 'breed true', and therefore that any undoubted case implies a previous one as the source of infection.

Now in Europeans the diagnosis of a marked case is usually far less open to doubt than in an Indian and I therefore propose to mention here a series of cases in which

it seems certain that the infection came from Native sources, with the resultant deduction that the Natives of India do indeed suffer from true Enteric Fever.

About 1902 I was asked to attend some sick inmates of a Roman Catholic Convent about eighteen miles away and found that while one case was malarial the other two patients were typically enteric, of whom indeed one died. Now this Convent, which also includes within its walls an orphanage for native girls, is situated in a village entirely native with which there is daily communication through older women who do work in their homes which they have learnt in earlier years from the nuns and come to the Convent in connection therewith. Thepatients in question had not been away from their Community; whence then did they become infected but from some "native" case?

In 1904, one of the European Officers in charge of the Salvation Army work in his district was living in a village containing no other European inhabitant, with a native boy to attend him and prepare his meals after the Indian fashion. I saw him on September 1st when he complained of headache for three or four days - attributed to exposure to the sun - and some feeling of faintness and malise; there was nothing to cast doubts on the aetiology suggested and he returned to the village, no further news coming till the 7th, when his temperature the previous evening was said to have been 40.1°C! He was brought in to a room in my quarters on the following day, when a plentiful rose rash was evident, and passed through a severe attack of Enteric, the evening temperature

remaining

remaining at about 40° C. till September 26th and once reaching 41.2° C.

Again, in 1906 the Rev. Father C. was in charge of a native congregation off the main road to Trevandrum and lived in some rooms adjoining his church attended by a native servant. I was called to him when he had been ill about a week, had him removed to a place somewhat more accessible, and there he successfully came through a fairly severe attack of Enteric, (unfortunately only to die about a year later from Cholera).

Whence did these two Europeans become infected with the specific bacillus, but from some "native" source of infection in their respective surroundings?

The last case I have to mention under this heading is an even more striking one:-

I had been attending in his own home in a Caste village (the first European visitor thereto, I believe, in its history) about three miles from the Hospital the heir of a well-to-do Hindu family with a serious attack of what I considered to be Enteric Fever, and the patient had recovered to the very great joy and satisfaction of his family, who presently desired to show their gratitude by inviting as many as possible of the Hospital staff to a Feast after their own manner at which they might present a thank-offering, an invitation which was accepted. We never hesitate to take food freshly cooked in accordance with Caste rules and served after the Indian custom straight from the cooking vessels to a fresh Plantain -leaf, as, in the absence of

flies

flies, such food may be considered practically sterile, while the fresh leaf is much safer than a plate or dish washed in water from a doubtful source and wiped on a cloth by no means necessarily free from infection (e.g. Last year a high Government official and some of his family died from Cholera the infection of which was traced to the cloths used for wiping the plates etc. for the table having been washed at a pool to which Cholera-infected garments had just previously been taken !)

There are also three safe beverages in an Indian village, in order of safety -

1. Tender coconut water (absolutely safe)
2. Fresh conjee-water - i.e. the water in which the rice has been boiled - if no other water be added afterwards.
3. Coffee, which is always boiled, as is the milk, but may be infected through the sugar.

On this occasion the first named was provided ; but towards the end of the end of the meal one of the Hospital staff, a Swede, ~~aetat~~ aged 27, asking for a little more, was by accident given some of the ordinary drinking water of the house, of which he took a drink before discovering the mistake: the rest of the party drank only tender coconut water.

Twelve days later an attack of Enteric Fever of moderate severity manifested itself in the victim of this mischance; a result which, I suggest, incidentally

confirmed

confirmed the diagnosis of the original case in that house.

(III.) — Have many Natives an acquired Immunity? C + S Cases

In some interesting remarks last year by Lt - Col. D.B.Spencer,[13] he quotes the statistics for the British and native troops in India for fifteen years ending 31st December 1906, showing 21,929 cases with 5,481 deaths among the British (not counting Officers) and only 808 cases with 223 deaths among about twice the total strength of natives; and expresses the opinion that while

> "if the difference in figures of the incidence of
> "Typhoid in the British and Native communities were
> "slight, one might possibly admit a relative
> "immunity among Native troops, the result of their
> "having lived for generations amidst insanitary
> "surroundings in their village homes previous to
> "enlistment in the Army"

the actual difference is too great to be thus accounted for, it may be

> "that the excessive consumption of animal diet
> "by European troops in India, in a climate where
> "such food is generally unsuited, may possibly
> "explain the relative frequency of Enteric Fever
> "among European troops, as compared to the same
> "disease among Native troops, both living and
> "working side by side in our military cantonments,

year

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"year in year out, under precisely the same hygienic
"conditions".

This is followed up by Major Thompson,[14] who suggests that the enteric germ

"cannot gain a foothold in those not already predisposed
"to nurture it by ' the prepared ground' of an
"irritated intestinal canal superinduced by 'luxus
" 'consumption of proteids ' ".

That Europeans in India are apt to consume an excessive amount of animal food and that it is a climate where such food in excess is generally unsuited is pretty generally admitted, but that this habit, while unwise, is apt to set up "an irritated intestinal canal" is more open to question, and I do not quite see why it should ~~not~~ necessarily be easier to account for so large a difference (Colonel Spencer does not see any difficulty if the difference were but slight) by a (supposititious) condition of the intestines in the Europeans concerned, who are also liable to the disease in their own land, than by a condition of acquired immunity in the Natives of the country, where they are certainly liable to conditions favouring infection yet in many cases without becoming infected.

In support of this theory it is stated that the Ghurkahs are more nearly akin to Europeans in their meat-eating and other habits and that the Ghurkah troops are also intermediate between the other native troops and the British in their liability to contract enteric.

There

There however seems to me to be a fallacy underlying these wide statements of diet in that it seems to be implied that the natives of India are all practically vegetarian, which, in South India at least, is certainly far from being the case in civil life, where only the Brahmins and the élite of the upper castes are vegetarian from principle and the very poor from their poverty.

Curious to relate, too, out of ten cases of Typhoid fever in the persons of natives of India, reported by G Lamb[57] in 1902, six out of the ten were in Brahmins !

I would suggest that some instructive statistics might be compiled by those who have the opportunity, if the entire figures were worked out for different classes of Indians in the same regiments or cantonments : for instance, in one there may be Mahonedams who are flesh eaters, and Brahmins, who are strict vegetarians. One ought, according to the diet theory, to find a very marked disproportion between the incidence on the two classes of otherwise almost identical enviroment and habits.

If then it may be accepted that some natives of India do suffer from Enteric, it only remains to consider their habits and surroundings in relation to hygiene to arrive at the conclusion that the means of infection must be almost universal in the country, and so to come to the question "Why do they not all contract it?"

Some observers, indeed, have swung from the early belief that Indians are not liable to the disease at all to the opposite extreme as in the "Remarks on the apparent immunity of Asiatics from Enteric Fever " by F.W.Clarke[58] in which he quotes from his own report for 1897 :-

"It

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"It is interesting to note the small number
"of cases of Enteric Fever which occurred among
"the Chinese during the year (in Hong Kong)
"an experience which accords with the apparent
"immunity of the native population of India
"from this cause, while the circumstances connected
"with these cases appear to suggest that the
"same explanation of the apparent immunity may
"apply to both races -viz. that they are so fully
"exposed to the infection throughout the whole
"period of their existance that they almost always
"contract the disease in infancy or early childhood,
"when if they recover the disease will have been
"practically unnoticed, (- Whatever may be the
case with the Chinese, according to Dr. Clark,
my own fairly close acquaintanceship with Indians
of widely different grades does not incline
me to accept for a moment that many parents,
except possibly of the most aboriginal and
backward classes are to be found so utterly
lacking in observation and in care for their
children as this would seem to imply. P.F.T. -)
"while if they succumb the death will be attributed
"to Diarrhoea, convulsions, or some other symptom,"
which may be compared with the statement attributed by Dr. Andrew Duncan (?) to Elliot of Madras that "all natives suffer from it at some time or other".

Sometimes indeed the infection does seem to be particularly virulent and a number of people together or in quick succession contract the disease, as in the following instance:-

In

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In January 1907 a Sudra woman, aetat 30, was admitted on the sixth day of an attack, but stayed in Hospital six days only and then was removed back to her village. She was a member of a large Hindu joint family and a little later there were the following additional cases in the house, uncle about 60 and three children, boys of 8 and 6 and a girl of 4. As the house was a particularly spacious one, at the family's urgent request I sent out one of my senior students for a few days to take charge of the elder patient, with the result that in about a fortnight he also was attacked, in spite of great care in the matter of antiseptics, drinking water etc. I attribute this unusual number of cases in one house to two factors:

1. The attacke were accompanied by diarrhoea,
2. The place simply swarmed with flies, from which it was almost impossible to keep food free even while it was being eaten.

During the times when flies are prevalent they must be a very powerful means of infection, as is the case with Cholera, which is seldom absent from us in the dry season, although varying considerably in extent from year to year. Generally speaking it starts towards the end of the year with a few sporadic cases, but, unless some big festival intervene, not to am epidemic extent until some fishing village on the coast is affected. In these fishing villages large quantities of fish are sun-dried, and the plague of flies about them is great in consequence: apparently when a case of Cholera appears in such a place the half-dried

fish

fish is quickly infected by means of the flies and then one hears of a sudden outbreak of cases in the inland parts to which the fish has been taken.

But generally speaking, although there may be one or more distinct foci of enteric infection present in a community there is no epidemic of cases.

Take, for instance, the orphanage in the same compound as the nunnery referred to above; most of the orphans had been taken in as babies and lived there all their lives, but there was no epidemic among them, although two of the European nuns had somehow contracted the disease in spite of sanitary habits very different to those of Indian children or adults either.

Then, again, my European assistant was apparently infected by a single drink of the water habitually used by all members of that Sudra household, rather a large one, including three generations and well known to me so that I canbe practically certain that no other case of the disease occurred among them and that the younger ones at least had not suffered similarly at an earlier date.

Yet consider the native habits with regard only to water. The orthodox Hindu must bathe every morning. If there be a river near he will probably go thither, but usually he must be content with a neighbouring "tank" (a more or less artificial lake or reservoir) in which he will bathe himself, wash his clothes, and then rinse his mouth. To the same water, when tired of walking, he will come to bathe his feet, to it the herdman brings his buffaloes

and

and oxen, to it the dhobi brings his bundles of soiled clothes for washing. But none of these uses will be considered to make the fluid unfit for use in the daily careful toilet of the teeth. The said teeth generally do the care bestowed upon them great credit, but what of the digestive tract at the same time inoculated with innumerable bacteria?

Next, consider the "jungle", the pieces of waste land to which the whole population resorts each morning for the offices of nature, the dejecta being left upon the surface of the ground to the drying of the sun and the wonderful operation of the white ants, by which a definite faecal mass can hardly be recognized as such in perhaps twenty four hours and can easily therefore be carried on the feet of some other comer in the next day or two to the tank at the time of the morning bath, apart from being blown about as dust by the wind after desiccation under a tropical sun.

It may perhaps be questioned

(a) Are infected people likely to go out to the "jungle" ?

(b) Would not any parasitic bacteria soon be destroyed by the further action of that tropical sun?

In answer to (a) may be mentioned, firstly, the now generally accepted existence of "typhoid carriers", who, having recovered from an attack of the disease, continue for a long time intermittently to discharge virulent bacteria from the bowel; and, secondly, the following specific instance of another character, reported by T.P. Woodhouse (17) :-

"Four

"Four orderlies, who had been employed exclusively
"in nursing Enteric Fever cases.... were all in
"perfect health: none had been inoculated nor had
"had enteric fever. They lived together in a
"separate tent. On examination, two out of
"the four were found to be enteric bacillus -
"carriers: in both the faeces were infectious".

Woodhouse asks, "Why did not the two infected men convey
"the disease to their comrades who had lived
"for some months in such close relations to them?"
and suggests in answer that either they were immune or that the bacillus of these chronic carriers is not of an active, highly infective character.

The latter hypothesis hardly seems to be tenable when one remembers that these bacilli had presumably been caught from the actively infectious cases which these orderlies had been nursing: while it may further queried, "Why did not the carriers themselves get Enteric Fever ?" to which I would suggest the answer, "Because they both had acquired an Active Immunity."

In connection with question (b) the experiments in Kasauli of W.S. and L.W. Harrison (19) are of great importance:-

"Contaminated dust was exposed to the sun daily
in June from 10 a.m. to 4 p.m. the test thermometer
giving an average temperature of 53°C: yet
living typhoid bacilli were recovered after 77½
hours during 23 hours of which the direct rays
of the sun had been acting."

It would appear therefore that in spite of the exposure to a tropical sun typhoid bacilli may survive in infected dejecta for several days at least.

Lastly

Lastly, how often, remembering Horton-Smith's discovery of the Bacillus Typhosus in urine may not the very vicinity of the houses be specifically contaminated by the prevalent habit of micturition there during the dark hours by nearly everybody, including even the sick when anyhow possible.

I am led to the conclusion, therefore that in South India we may well suppose that

"widespread prevalence of the virus.....
"or possibly of the virus of other allied filth
"diseases"

which Roberts[10] postulated as a necessary precedent condition for the production of Acquired Personal Immunity through

"the constant ingestion of imbibition of doses
"of the virus which do not avail to induce an
"attack,"

and that this form of Acquired Immunity is the great reason why the native population has not to be divided up —— to make an adaptation of the old pre-vaccination era Variola census classification, —— into,

1. Those who have previously had Enteric
2. Those who have just had Enteric.
3. "Those who have to have their" Enteric!

ON ENTERIC FEVER IN SOUTH INDIA.

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ON ENTERIC FEVER IN SOUTH INDIA.

Postscript, April 1911.

To bring the Statistics given in the foregoing paper up to date I am now able to add the numbers and age-classes of those admitted to the Hospital for Enteric during a second period of three years, viz, up to March 31 st, of this year.

It will be seen that the total number of admissions is very much greater than in the previous period, but this will in part have been accounted for by recent additions to the Hospital buildings, more than doubling the previous accommodation for In-Patients.

Ages:-16,16,42, 2, 1,17,36,28,57, 9,16, 9,20, 3,20, 4,
8, 4,10,20,10,12,1½,30,35,30,18, 5, 6,27,14,
8,12,35,11,12,30,19,31,26,31, 5.

Of these, 20, or almost half, fall into the under 15 category
9 are between 15 and 25, and
13, or not quite one third, are over 25 years of age, giving an average of 17. 7 years of age compared with one of 13. 7 in the cases admitted during the first period examined.

This might be put in as an original article but leaving out the words original article

REPORT

ON AN INVESTIGATION OF CEREBRO-SPINAL FEVER IN THE NORTHERN TERRITORIES OF THE GOLD COAST IN 1908.

By

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REPORT ON AN INVESTIGATION OF CEREBRO-SPINAL FEVER IN THE NORTHERN TERRITORIES OF THE GOLD COAST IN 1908.

General History.

Cerebro-spinal Meningitis has been met with in natives of West Africa in different periods and places, and in the early part of 1905 formed a severe epidemic in Northern Nigeria which was reported on by Drs. Twomey and Davidson. The disease was supposed by the natives to have been introduced from the North-east but "not in the memory of living man," and serious outbreaks have occurred there at intervals during the last fifty years. Two Europeans died from the disease in Northern Nigeria in 1905.

In the early part of 1906, was first noticed a marked increase in the number of deaths occurring amongst natives of the Lobi-Dargati and Issalla-Grunshi country in the North-west and Western parts of the Northern Territories of the Gold Coast. The increased mortality was confined to the Harmattan season and ceased at the onset of the rains; Dr. Collier, the Medical Officer then stationed at Wa, diagnosed the disease as Cerebro-Spinal Meningitis and found the epidemic in a very severe form at Tizza, a town about 14 miles south of Lorha, and in the surrounding neighbourhood. With the commencement of the Harmattan of 1907 the disease recurred in the same districts producing an enormous mortality and many villages, spared the previous year, suffered most severely, in some cases whole villages being destroyed by it. Dr. Palmer who visited the infected neighbourhood in April of that year estimated the number of deaths as 8,000 or more; he found that the epidemic had apparently started at Ulu, a Dargati town about 20 miles east of Lorha, from which it had spread to a slight extent east as far as Tumu, in Issalla country, but that the main spread was to the West and South-west, reaching Wa and crossing the Black Volta river into French territory. It again ceased at the beginning of the rainy season.

History of Present Epidemic.

About the end of October, 1907, a suspicious case was reported from Lorha and I was detailed to proceed from Accra to the Northern Territories to investigate the disease should it recur in the Harmattan season of 1908. Leaving Accra on November 13th, 1907, and travelling *via* Kumasi to the northern Territories, I passed North through the western side to the affected area. The disease was unknown in the villages on the road up as far as Wekyan, three days north of Bole, but at Wekyan the chief said there had been five deaths in the preceding three weeks, and I saw one boy who was convalescent from Cerebro-Spinal Meningitis to which the other deaths were attributed. No further cases were seen, although the disease was known at many of the villages, until arriving at Lorha on January 3rd, where it has just broken out in the neighbourhood. I therefore made my headquarters at Lorha during the Harmattan, travelling from there about the Lobi-Dargati and Issalla countries.

Shortly after this, cases appeared at Golu, twenty miles north-west of Tumu and the disease was more or less present in other towns of the district west of Tumu. From this primary area the infection spread mainly to the west and south west and to a less extent to the north-west; thus, there was little or no disease east of Tumu, and in the north, in French Territory, which I visited in February, M. Bouchot, the Administrateur Resident at Leo, told me the disease was unknown. To the north-west however many villages became infected during February and March, notably Lambussie, Kokolobu and Tantua, the latter being almost wiped out in February while south of Lorha many villages along the main and indirect routes to Wa where similarly affected. Wa itself remained quite free of Cerebro-Spinal Meningitis but in February had an epidemic of pneumonia and later, several deaths from epidemic diarrhœa.

In Bole, during February, two deaths occurred in a Dargati caravan which had travelled south from the district between Lorha and Tumu and immediately after, the infection broke out in the town causing eleven deaths, but early isolation was enforced and it did not spread.

At Beri, a village near the Black Volta river on the main route between Kintampo and Bole, a sporadic case occurred in a man visiting the camp of the West African Frontier Force, then on manoeuvres in the district, but, so far as I can ascertain, no other cases occurred there or amongst the West African Frontier Force and this marks the most southerly extension of the infection.

In French Territory, to the West of the Northern Territories, Cerebro-Spinal Meningitis appears to have been very severe. Dr. Bargy, of the French Government Medical Service, with whom I discussed the question early in March, stated that the epidemic was marked to the west of the Black Volta and was spreading considerably to the south-west of the country; I could not ascertain that any special means had been adopted to combat the disease.

In the Northern Territories, therefore, Cerebro-Spinal Meningitis has increased, as regards the area affected, during each ensuing epidemic of the last three years so that it has appeared this present season markedly in the more populous north-west districts, and scattered at intervals along the western border as far as the boundary between the Northern Territories and Ashanti, covering an area roughly of over 10,000 square miles.

It is very difficult to form anything like a correct estimate of the total number of deaths caused by it. No count is kept by the natives as a general rule after about ten or twenty deaths have occurred in the village and a rough figure is all that can be obtained such as "the deaths were as the leaves of a tree," and when pressed, they will name a number which represents in their minds an indefinite large quantity such as "Two hundred" or "Fifty," but probably has no absolute relation to the real number. There can be no doubt that the mortality was extremely high in many places, especially in villages not previously attacked; "funeral customs," so dear to the native heart, were perforce abandoned in some villages owing to the rapidity with which deaths occurred, and the number of towns deserted in the north-west districts, during this and the preceding epidemics, bears testimony to its ravages. Then again, it must be remembered that the districts round Lorha are only now being opened up, and information as to the amount of sickness in many of the smaller villages is very difficult to obtain, so it is, I think, probable that the epidemic was present in places of which we had no distinct knowledge. In villages affected by previous epidemics the native opinion was that the disease was less severe this year, but on the other hand in villages visited by the disease for the first time, it proved very fatal.

The case mortality was very high in the height of the Harmattan, probably at least 80 per cent., but, as in previous years, was lessened towards the end of April when the epidemic ceased. On the whole I believe the fatalities were less than in 1907, and probably did not exceed 6000. Had the disease succeeded in gaining ground in the hitherto unaffected districts south of Wa there can be no doubt that a terrible mortality must have ensued.

No cases have occurred among the white residents of the Northern Territories, nor were any native officials attacked during the epidemic.

It is I think of very considerable interest to note that very careful enquiry among the chiefs and headmen of the villages I visited showed that Cerebro-Spinal Meningitis was entirely unknown in the district before 1906, and not within the recollection of any of them, had any such epidemic, or even isolated cases of sickness presenting the same symptoms, occurred to their knowledge.

Etiology.

Cerebro-Spinal Meningitis occurs in epidemic form in the Harmattan or dry season which lasts usually from November to May and is followed by tornadoes and the rainy season. The Harmattan wind blows steadily from the north-east until towards the end when it shifts round to the west and south-west, and is extremely dry and dusty; the country is undulating, with low scrub, grass and scattered trees in some places, or in other parts the small trees are closer together and form an "orchard country."

Vegetation is thicker along the water-courses, but there is no dense forest country as in Ashanti, consequently the relative humidity of the air is very low during the Harmattan, following as it does the warm moist air of the rainy season. The soil in the villages is light and dry and is largely composed of light earth mixed with ashes dumped there from the wood fires of many generations; this of course contributes to the dust of the Harmattan. These conditions react on the natives, producing catarrhal conditions and Pharyngitis, Laryngitis, Bronchitis and Pneumonia are very common in the early Harmattan; pneumococci and non-Gram staining cocci are easily found in the naso-pharynx and a ready ingress for the Meningococcus is probably formed.

The inhabitants are in the main an agricultural people, living on a vegetarian diet varied by occasional orgies of "bush meat." Their villages are composed of flat roofed mud houses or compounds separated from one another by distances varying from about thirty to two hundred or more yards, so that a village may easily occupy an area of ~~four~~ or ~~five~~ square miles; the individual compounds consist of an irregular collection of small chambers communicating by low openings in the mud walls or connected by low, dark and devious passages, after the nature of a rabbit warren; a small hole in the outer wall, and frequently one in the flat roof form the only communication with the exterior or a small yard enclosed by a wall. Fowls enter freely and fully into the family life and sheep and goats are herded inside at nights for protection from wild animals, the ventilation is naturally poor and the crowded conditions which obtain in the compounds render the dissemination of infection an easy matter.

As is usual in Cerebro-spinal fevers, the young of both sexes are mainly affected; as far as I could ascertain, all ages from early infancy to young adults of about 25 years appeared equally liable, the very old and weak also suffered considerably while the infection was less common amongst the middle aged and, when occurring, ran a chronic but less severe course. No attempt at isolation is made by the natives under ordinary conditions. Funeral customs, held immediately after death, consist of singing and

dancing without intermission for three days and nights during which visiting from adjacent compounds and villages is common: the corpse occupies a post of honour during these proceedings and is possibly a source of infection for all concerned.

The conditions and mode of life therefore are such as to give every facility for the transference of this disease if, as is generally believed, the germ of Cerebro-Spinal Meningitis is conveyed in the nasal mucus of those people who have been in close contact with the sick. It is almost certain that the infection is so conveyed from place to place, although these "contacts," bearing the infective germ on them, may not themselves develop the disease. Caravans are constantly passing north and south during the dry season and as the traders in many cases sleep in the native villages they probably form an important means of spreading the infection; certainly it was by this means that Bole became attacked.

The period of incubation is not known and I have found no reliable data in this epidemic on which to base an opinion.

In Northern Nigeria cattle are said to have been attacked and in the Northern Territories the epidemic of 1907 is said by the natives of the Lorha district to have been preceded by a great mortality amongst the fowls of some of the villages; nothing of the kind was however noticeable during this present season.

There is a concensus of native opinion that the disease only appears during the dry season but I think it probable it is endemic in the country, cases during the rains being atypical and less severe, but sufficient to produce a recurrence under the more trying conditions of the Harmattan.

Bacteriology. — Cerebro-spinal fever has been recognized in Europe for over a hundred years and bacteriological investigation has proved that the disease may be associated with at least ten different organisms, including the *Diplococcus pneumonia, Streptococcus pyogenes, Staphyllococcus pyogenes, Diplococcus intracellularis meningitidis* (*Micrococcus meningitidis cerebrospinalis*). *Bacillus typhi abdominalis. Bacillus pestis, etc., etc.* Recent research has however tended to show that primary or idiopathic cerebro-spinal fever is produced for the main part by the *Diplococcus intracellularis meningitidis*, but that in a few cases the causative agent is the *Diplococcus pneumoniæ.*

It was therefore of some importance to ascertain which of these distinct causes was responsible for the present epidemic. The procedure adopted included microscopic examination and culture and isolation of the organisms found in the spinal fluid of patients suffering from the disease. In six separate cases in which spinal puncture was performed the resulting material was examined microscopically, and received also directly into tubes containing nutritive culture material which was incubated at a temperature of 37°C.

For the microscopic examination different stains were used but the most important for diagnosis was the staining of the *whole* film by Gram's method and, when dried after the decolorising process, the staining of the one *half* of the film by dilute Fuchsin. Most known cocci (*Including Diplococcus pneumoniæ*) retain an intense violet colour after treatment by Gram, but some few, including the Gonococcus, the *Diplococcus intracellularis meningitidis* and *Micrococcus catarrhalis*, remain colourless at the end of the process; these latter may be stained red by Fuchsin and the method adopted of staining the whole slide by Gram and subsequently one half of the slide by Fuchsin makes the contrast more marked and the recognition of doubtful cocci easier.

The culture material used principally was of two kinds—Glycerine-Agar and a mixture of Agar, Peptone, and Ascitic fluid the latter being, from its more nutritive composition, preferable to the former for diagnostic purposes as micro-organisms grow on it more rapidly and luxuriantly.

The following results were obtained.

Case I.—A girl at 13 years approximately, at Nyari four miles west of Lorha. Spinal fluid limpid, colourless, flowed freely; about one fluid ounce removed. Microscopically (Slide No. 2), stained with Gram and Fuchsin; lymphocytes only present and extremely few, no poly-morphonuclear leucocytes, and no cocci; a centrifuged specimen showed no cocci. Inoculations were made on Ascitic-Agar and Glycerine Agar tubes which were incubated at 37 C.; colonies of Staphylococcus epidermidis albus appeared in 12 hours in the former together with gross contaminations by Bacilli; the glycerine-agar tube remained sterile.

Spinal puncture was again performed four days later but proved a "dry tap."

Case II.—Girl at 18 years approximately; brought to Lorha from Jatore, about 2½ miles to the east. Spinal fluid clear, flowed freely; one ounce removed. Microscopically (Slide No. 8); film stained with Gram and Fuchsin, polymorphonuclears and lymphocytes present and, after prolonged search, a few intracellular de-Grammed diplococci stained red are to be seen. Slide 10, stained with Loeffler's Methylene Blue shows also after prolonged search intracellular diplococci. Inoculations of Culture tube showed only Staphylococci and contaminations with Bacilli.

A second spinal puncture was made two days later and nine drams of turbid flocculent fluid removed. Films examined microscopically showed the same appearance as before (Slide No. 12.) Inoculations were made on 4 Ascitic Agar and 1 Glycerine-Agar tubes and after 12 hours incubation at 37 C. small discrete colonies of Staphylococci appeared in each tube; the surface of the culture material was then re-inoculated with the spinal fluid which lay in the bottom of the tube and the whole replaced in the incubator; twenty-four hours later appeared a flat translucent, pale bluish or grey growth, consisting of minute colonies, discrete at the edges of the mass of growth. In some of the tubes the new growth surrounds the previous growth of Staphylococci which are discrete round colonies about 3 m/m. in diameter, with smooth edges, raised centrally and more opaque. The growth appeared in both the Ascitic-Agar and the Glycerine-Agar tubes but was thinner and less luxuriant in the latter; it is entirely confined to the surface of the culture material; see culture tube A Microscopically (Slide Nos. 14 and 15) the growth consists of cocci decolorized with Gram and stained red with Fuchsin, they present some variation in size, some being about the size of Staphylococci, and others smaller, more oval in shape and arranged markedly in pairs or in tetrads; no capsule is present. A film stained with Leoffler's Meth. Blue (Slide 16) shows cocci in the same formation, *i.e.*, as diplococci or in tetrads.

Case III.—Male adult at 20 years approximately at Dikpe two and a half miles south-west of Lorha. Spinal fluid thick and purulent, flowing slowly drop by drop; six drams removed. Microscopically it contained enormous quantities of degenerating polymorphonuclears and a few lymphocytes; stained by Loefflers Meth. Blue diplococci are seen in many of the leucocytes

(Slide 18); stained with Gram and Fuchsin (Slide 19) and with Gram and Eosin (Slides 20-23) I cannot definitely recognize any diplococci.

One Glycerine-Agar and three Ascitic Agar tubes were inoculated with the spinal fluid; all except one Ascitic-Agar tube were incubated at 37 C. and in thirty-six hours each of these tubes showed a pure culture resembling in all respects the growth described under Case II. The remaining Ascitic Agar tube, which had been incubated at a lower temperature (an average of 25°-30°C) showed no growth after forty-eight hours when it was placed in the warm incubator, and in twenty-four hours a pure culture identical with the others appeared. Films of these cultures stained with Gram and Fuchsin (Slide 24) show small de-Grammed cocci stained red by the Fuchsin; they are slightly oval in shape and frequently occur in pairs (diplococci) with the opposed sides slightly flattened and the long axes parallel; in size they are about 1.6 *m* in length by .8 *m* in widtli. Tetrads also occur and microscopically as well as macroscopically, the organism is identical with that obtained from Case II. (see culture tubes B and C.)

Case IV.—Boy at 11 years approximately at Tumu sixty miles east of Lorha. Spinal fluid clear, limpid, under considerable pressure in spinal canal, flowing at first in a jet of fair force; one ounce was removed. Microscopically (Slide 26): film stained with Gram and Fuchsin, very few small lymphocytes present, no diplococci definitely seen. An Ascitic-Agar and a Glycerine-Agar tube were inoculated with the spinal fluid and incubated at 37 C, in forty-eight hours appeared the flat translucent bluish-white culture described in the preceding two cases, slight on the Glycerine-Agar but well marked on the Ascitic-Agar which was however contaminated with a fungus growth (Mucor.) and with Staphylococci. Specimens were stained with Gram and Fuchsin and examined microscopically; Slide 28. shows small paired cocci faintly stained red, and larger paired cocci more deeply stained with red. Hyphœ of the fungus are also present and stained dark blue with the Gram, contrasting vividly with the red cocci on the half of the slide stained with the Fuchsin. Similarly stained cocci without fungus growth are also seen in Slide 29.

Case V.—Boy at 13 years approximately at Kokolobu thirty miles north of Lorha. Spinal fluid clear, extremely low pressure, coming out drop by drop; one dram removed; films were made and an Ascitic-Agar tube inoculated. Microscopically (Slide 37) extremely few leucocytes to be seen; fibrin network marked and some very doubtful red-stained extracellular diplococci. I was unable to obtain a centrifuged specimen for examination as the puncture was performed two days journey from Lorha. The culture showed Staphylococci and Gram-staining Bacilli at first, and in forty-eight hours a thin growth of cocci of the same appearance as in the last three cases, and microscopically also decolorized with Gram.

Case VI.—Boy at 13 years approximately at Lorha. Spinal fluid yellowish green colour, turbid, four drams removed. Films for microscopic examination were made and seven Ascitic-Agar tubes inoculated with the fluid; stained with Gram and Fuchsin (Slides 43 and 44) many polymorphonuclear leucocytes and lymphocytes are seen, some few containing de-Grammed diplococci stained red; some extracellular diplococci are also to be seen. Slides 45 and 46 are stained with Loeffler's Meth: Blue and show similar white corpuscles with extra and intracellular diplococci.

Culture experiments.—After 24 hours incubation at 37° C. all seven Ascitic-Agar tubes presented pure, luxuriant growths of precisely those characters above described, viz:—a flat translucent bluish-white growth of colonies varying in size from minute "pin-point" spots up to about 3 m.m. in diameter, with smooth edges, usually discrete but closely

opposed where the growth is thickest. In a culture some days old the colour appears very faintly yellow. Films of these cultures were made and examined microscopically (Slides 47 and 48); stained with Gram and Fuchsin they are seen to consist of diplococci which have not retained the Gram stain but are coloured red with the Fuchsin. In size and appearance they resemble those described above under Cases II. and III. (see culture tubes D and E.)

Blood cultivations were attempted in four cases but the cocci were not obtained in any case.

Thus, in five out of six cases, or in 83 per cent. of patients suffering from Cerebro-Spinal Meningitis, an identical organism was isolated and grown in culture. These six cases were far removed from one another, one being four days march east of Lorha, another being two days north of Lorha, so they may, I think, be taken as representative of the epidemic in the Northern Territories and probably other parts of West Africa, and the organism isolated be regarded as the causative element.

The characters of this organism, as far as I was able to investigate them are as follows. It is a Diplococcus consisting typically of two cocci of somewhat oval shape, length about 1·5 ~~m.M.~~ width about ·8 ~~m.~~ opposed with their long axes parallel and the sides in opposition slightly flattened. In the spinal fluid it may be extra—or intracellular and is sometimes encapsuled, but a capsule is not always visible; single cocci are also seen; it is non-motile and there is no spore formation. In specimens taken from a culture, the cocci occur in pairs and frequently in tetrads, but larger forms also occur singly and in pairs. It stains well with aniline dyes, but presents the rare characteristic that it does not stain by Gram's method, which at once distinguishes it from Staphylococci, Streptococci and the Diplococcus pneumoniœ. It is ærobic and requires a nutritious medium for its satisfactory culture; on Ascitic-Agar the cultures, as a rule, were not visible until after 24 hours incubation at 37° C; in 36 to 48 hours they present the appearance of small discrete colonies varying in size from minute specks to about ·5 m.m. diameter; in older cultures the colonies may attain the diameter of 2 m.m. or over; they are flat, roughly circular with smooth edges, translucent, and of a very faint bluish-white tint as seen by transmitted light, in older cultures this bluish-white tint is lost somewhat and replaced by a duller faintly yellow colour but no pigment is formed; in the thickest part of the growth the colonies become closely opposed by their edges but do not fuse into one apparently smooth surface. On glycerine-agar the culture presents the same characteristics to a less degree; the growth is considerably slower and the colonies are smaller. The optimum temperature is about body temperature, *i.e.*, 37° C; below 30° C growth is very much delayed. The culture material is not liquefied by the growth, and no gas is formed.

The above characteristics agree, practically, entirely with the main characteristics of Weichselbaum's *Diplococcus intracellularis meningitidis*, otherwise known as *Micrococcus meningitidis, cerebrospinalis*, or more shortly as the *Meningococcus*, which has been shown to be the cause of the recent epidemics in Ireland, Scotland and the Continent and from the above experiments appears to be the cause of epidemic Cerebro-Spinal Miningitis in West Africa.

There are other properties of the Meningococcus of Weichselbaum which are of minor importance to the bacteriologist but of great importance in dealing with the disease it produces, for instance, its vitality; its power of resisting dessication; its pathogenicity on animals; its power of agglutinat-

ing with the serum of persons suffering from Cerebro-spinal Meningitis; its thermal death; its resistance to disinfectants; its method of transference from one person to another; its power of producing immunity all of which have been, and are still being, investigated, but about which there is still considerable doubt.

With the Meningococcus isolated from the cases described I was anxious to work out some of these minor characters, but I found the conditions under which the work had to be conducted in the Harmattan season in the Northern Territories were most unfavourable to such investigation and it was more immediately profitable to spend my time endeavouring to stop the spread of the disease. Experiments which I was however able to perform tended to show that the vitality of the meningococcus is not great that it is easily destroyed by a comparatively low temperature and by dessication, as cultures spread on cloth and dried at the ordinary temperature in daylight (not in direct sunlight) for 24 hours, failed to survive.

As before stated, it has been practically proved by investigators in the epidemics of Europe that the meningococcus lives, and is transported, in the mucus of the naso-pharynx of "contacts" or persons who have been in close association with the disease, and the germ has occasionally been recovered from such sites. Lingelsheim at Beulen in Germany claims to have found the germ in the naso-pharynx of 20 per cent. of cases of Cerebro-Spinal Meningitis and in 9 per cent. of contacts and normal persons, while Goodwin and von Sholley, in 1906, recovered the germ in 50 per cent. cases of Cerebro-Spinal Meningitis during the first two weeks of the sickness, and in 10 per cent. of the contacts; they emphasize the importance of isolating cases during the early weeks of the disease. The habits of the native render it easy enough for the nasal mucus of one person to be transferred directly or indirectly to another or inhaled when excreted and dried on the earthen floors or on foul clothes and the recipient may both contract the disease and convey it to other parts of the country.

While travelling about in different parts of the Northern Territories, I made frequent examinations of the nasal mucus both of patients and of contacts, but although I was much impressed by the extensive and varied flora of the native nose I was unable to isolate and recognize any meningococci. Gram-negative cocci were present in abundance but, in culture, formed the heavy mucinous grey growths separated as Type I. by Dunham in his work on this subject.

All bacteriological work had to performed under very primitive conditions; my "laboratory" at Lorha was a small room in the temporary native hospital, a mud building with a mud and cowdung floor and a grass roof, white ants made constant attacks on boxes and apparatus from below, "borers" in the roof constantly dropped fine sawdust over everything and the irritating dust of the strong Harmattan wind found its way everywhere in the absence of any kind of door or windows. Spinal puncture had also to be done under equally unfavourable surroundings, mostly with the patient on the floor in the native house or compound, with the wind blowing straw and ashes about, the family fowls in close proximity and usually a large group of interested but septic natives. An unfortunate habit of the native is that when sick, the body is anointed with a "Medicine," some filthy greasy composition in which shea-butter forms the vehicle for the active principle, apparently soot, and this, combined with their infrequent habits of washing, renders the task of making the skin aseptic at the spinal puncture peculiarly difficult; dirt is engrained throughout the epidermis and even after a scrubbing and cleansing sufficiently thorough to expose the dermal papillæ, gross

dirt could easily be seen on rubbing with cotton wool. These conditions had to be made the best of, but I mention them as accounting for the contamination of bacilli and fungus which appeared in some of the cultures described, while the difficulties of travelling and of life in a native village made it impossible to attempt sub-cultures.

No post-mortem examinations could be made nor material obtained from a body after death, on account of native feeling on the subject. One such attempt by Dr. Cope of Tumu had to be hastily abandoned owing to the hostile attitude adopted by the natives and it was judged advisable not to interfere with native prejudices on the subject to too great an extent.

Clinical Symptoms.

In almost all cases the onset is sudden, the disease attacking persons apparently in good health. Intense headache at first general, but becoming more localised in the occipital region is commonly the earliest sign and this is soon followed by stiffness of the neck and more or less retraction of the head. Vomiting is commonly an early symptom and convulsions are frequent in infants; the temperature is usually raised but may be subnormal at this stage and the pulse has usually shown increased frequency, I have never met with the marked slowing of the pulse generally associated with this disease.

Cases fall into the classification usually adopted of I. Malignant: II. Ordinary: III. Atypical.

I. *Malignant.*—In this, the acute fulminant form, a person, apparently in perfect health in the morning, is suddenly attacked with headache, possibly vomiting, stiffness of the neck, more or less retraction of the head, rapidly followed by delirium, spasmodic contractions of arms, legs or other parts, unconsciousness, coma and death in less than 12 hours. This form of the disease is by no means uncommon and although it was never my fortune to meet with such a case, native descriptions are precise and to be met with in almost every village I visited where the infection had occurred, especially in the earlier stages of the epidemic and during its height; whether the temperature is high or low I cannot tell, but the described course of the disease suggests that death occours from hyperpyrexia. No eruption appears to occur. It is obvious that these symptoms agree very closely with those of Siriasis or "sunstroke," but apart from the fact that these cases occurred mostly before the hottest time of the year (March and April) the dry climate of the Harmattan, the comparatively open country about 500 miles from the coast line, and the altitude (about 1,000 feet above sea-level) render it improbable that Siriasis is the cause, while the presence of the cerebro-spinal epidemic leaves little doubt that the symptoms are due to the latter.

II. *Ordinary*—Onset is sudden, with increasing headache, frequently vomiting, pain and stiffness in the neck and back, followed by retraction of the head; the temperature is variable up to about 103° F. but I have never found any very high temperature at this stage, and the pulse rate has been moderately accelerated; during the whole course of the disease the temperature is erratic and presents nothing characteristic. Pain extends along the spine and intentional movements are avoided, photophobia is common. The patient is irritable and fretful, lies on one side and resents any interference; orthotonus is more common than opisthotonus and, although the latter occurs in slow spasms, particularly in infants and young children, I have never seen it so pronounced as in cases of Posterior Basic Meningitis amongst infants in England. Tonic contractions of the muscles of the limbs are common in young patients. Kernig's sign is present in practically

all the acute cases shortly after the onset but in many milder cases, especially in the older patients, it could not be elicited and, very frequently, no knee-jerk could be obtained although in some cases it was greatly exaggerated. Herpes occasionally occurs, particularly near the lips and alœ nasi; I have never succeeded in finding any purpuric eruptions although it is possible they may be present but invisible on account of the dark negro skin. Amongst ocular symptoms, conjunctivitis is common and probably caused more by dirt than the specific coccus; ptosis is less common; strabismus, divergent and convergent, is frequent, it is evidently of spasmodic origin, the eyeballs moving in jerky movements occasionally giving rise to conjugate deviation; the pupils are frequently unequal; no ophthalmoscopic examinations were made. Delirium is common in the acute cases, at first noisy but in cases going to the bad, soon subsiding into low muttering with intervals of apathy and unconsciousness. Food, usually consisting of a mixture of ground corn (gari) and water, is taken very badly, and in an illness of any length emaciation is extreme and accompanied by deep and extensive bed-sores from lying on the ground, or on thin reed mats. The cardiac action is correspondingly weak and rapid, sometimes irregular but cardiac murmurs are extremely rare. The respiration is shallow and irregular, Cheyne-Stokes breathing occurs in the later stages and there is frequently marked carination of the abdomen. The urine presents nothing characteristic.

This, the "ordinary" type, presents itself in acute, sub-acute or chronic modifications, running a corresponding course; the acute variety is common amongst ages ranging from early infancy to young adults and may terminate fatally in from 3 to 7 days or afterwards settle down to a sub-acute course; the primary sub-acute variety may appear at any age but is more common among full adults, it is of course less fatal than the acute and appears to last for three or four weeks and be followed by a tedious convalescence; it merges into the chronic form which includes cases amongst the more elderly people, causing a fairly large mortality; the symptoms of meningitis in this variety may be but ill-marked—persistent headache, some stiffness of neck, and marked debility and emaciation being the most prominent signs.

In fatal cases delirium deepens into coma from which the patient can only at first be roused with difficulty, and which may last over twenty-four hours before death ensues.

III. *Atypical.*—In this class I include those cases in which the more characteristic symptoms are marked by complications. Those that I saw included periarthritis and pneumonia.

Periarthritis was present in some subacute forms and affected particularly the larger joints of upper and lower limbs, producing swelling, tenderness and hyperœsthesia.

Broncho-pneumonia was present in a subacute case, a boy aged 11 years at Tumu, in whom was headache, stiffness of the neck, slight retraction of head, orthotonus with pain down the spine extending to the buttocks, weakness and dragging of feet. The constitutional disturbance was but slight and the temperature frequently subnormal in the morning and about 101° F. at night. There was diminished movement, dulness, slight tubular breathing and sibili at the left apex in front, behind and in the axilla; the pulse respiration ratio was about 2.6 1, but there was very little cough and no sputum could be obtained. Spinal puncture was performed (Case IV.) with some relief to the spinal symptoms and the boy eventually recovered. Two others cases complicated by pneumonia were subsequently treated by Dr. Cope at Tumu.

In this connection I must refer to the epidemic of pneumonia which occurred about the same time at Wa, four days south of Lorha, which was characterized by extreme severity, short duration, and high mortality. I did not see any of these cases, as I was in Tumu at the time, but the possibility of their being due to the meningococcus must not be forgotten. Several isolated cases of apparently the same nature, with death on the first day of illness, were about this time also reported from villages round Lorha in which Cerebro-Spinal Meningitis was known to be present.

Treatment.

The use of drugs is practically confined to the alleviation of symptoms and I could not discover that any drug had any effect on the course of the disease. An early calomel purge, and counter-irritation in the form of Liq. Iodi. or Liq. Epispasticus over the cervical spine gave some relief to the headache; cardiac tonics and stimulants are advisable early, and Phenacetin and Caffein are of some temporary use if the temperature is unduly high. Alteratives, such as Mercury and Potassium Iodide, are perfectly useless and the only drug which gave any considerable relief was Opium, in the form of Liq. Morphinae or Chlorodyne.

Spinal puncture in the lumbar region, and the removal of as much spinal fluid as will flow away of itself without aspiration probably relieves cerebral pressure and certainly relieved symptoms for a time in some cases; it is almost unnecessary to say that the greatest care must be taken to render the site of the puncture as far as possible aseptic, but is is a process which entails much labour and heart-weariness. The steps of this small operation need not be described, I think, but the needle used should be about 3½ inches long for comfortable use on an adult, and about 2½ inches for a small child, and they should be of good calibre. I have usually found that the needle penetrates the spinal canal at a depth of about 2½ inches in the adult and about 1 to 1½ inch in the infant but this naturally depends on the muscular development; fluid generally flows freely as soon as the needle enters the canal but, should it not, a stylet may be passed down the needle to clear it of any possible clot; it is frequently clear and limpid in severe cases of the disease.

Antimeningitis serums have not been tried on any cases in West Africa; their preparation and use require the utmost care and skill and conditions of surgical cleanliness which are at present unattainable so far from the Coast as the infected districts of the Northern Territories. In Europe at least three separate kinds of such serum have been tried and discarded as of no avail, but recently a serum has been prepared by Flexner and Jobling of the Rockefeller Institute for Medical Research, New York, which has been tried in the Belfast epidemic apparently with good results, the mortality being reduced from 72 per cent to about 25 per cent (British Medical Journal Febuary 15th, 1908). Judgment as to its proper value must be withheld until further results are known, but it is possible that a serum of this nature might be of considerable value in West Africa in any future epidemic, if it can possibly withstand the journey and climate.

Post-Mortem Appearances.

I can give no personal description of these as native feeling prevented any post-mortem examination being made.

Diagnosis.

The diagnosis is usually clear but may be doubtful in subacute or chronic forms in atypical cases. Constant headache, usually occipital, some stiffness of neck with possibly very slight retraction of head and tendency to orthotonus, and pain or hyperœsthesia over the spine are usually found. Kernig's sign is of great importance and pathognomonic of meningitis but I have seen undoubted cases in which it has been very

slightly marked or absent. The knee-jerk is frequently difficult to obtain in natives but if exaggerated in one or both legs is worthy of consideration in conjunction with other symptoms as confirmative evidence. Ocular symptoms, such as ptosis or momentary spasmodic squint, should be looked for. Spinal puncture and the recognition of the meningococcus would settle the diagnosis. The history of the case and the presence of others with more marked symptoms will probably help especially in such cases as infantile pneumonias.

The possibility of Siriasis being confounded with epidemic Cerebro-Spinal Meningitis has already being discussed.

Prophylaxis. The steps taken to limit as far as possible the spread of the epidemic were :—

1. Notification by the chiefs of the villages to the nearest Medical Officer of all cases of sudden death and of any increase in the rate of mortality among their people.

2. Isolation hospitals, temporary or permanent, were erected at stations in the infected districts, in suitable places, roughly south-west of the European settlement, that is to leeward having regard to the prevailing direction of the Harmattan wind, and about 400 yards away. As far as could be done, all cases of meningitis were isolated as early as possible but it was a very unpopular measure with the natives and a great difficulty was the nursing and feeding of patients as there were no trained nurses or dressers in the station. Disinfection of the hospital is sufficiently easily and completely performed at the end of the epidemic by firing the grass roof which falls into the mud building.

Isolation was preached to the chiefs and headmen of the infected villages and in one or two cases they were prevailed upon to build an outlying grass hut for the reception of patients but the objection raised, with some reason, was that the patients so isolated ran risk of attack at night from wild beasts ; generally the utmost that could be done was to collect the sick together in one compound, or one part of a compound, from which the younger people were excluded as far as possible. The probable methods of spread of the epidemic were pointed out and the rudiments of sanitation instilled into their primitive minds.

3. Disinfection of infected houses with burning sulphur was carried out where possible.

4. Examination of suspicious cases of sickness among caravans.

5. Funeral customs, with the consequent intervisiting, were forbidden or restrained as far as possible.

6. House-to-house visiting on any large scale could not possibly be carried out on account of the large areas covered by the separate villages but, as far as could be done, all cases of sickness were examined.

7. The public market was closed where considered necessary, as in Tumu, having regard to the people from infected villages who frequented it.

8. Station carriers and labourers were not taken from villages where sickness was present.

9. Zongas have been erected in many cases by the District Com-

missioners

as halting places

missioners to serve as halting places for travelling caravans, thus avoiding the necessity of traders sleeping in the native villages.

10. I found it advisable at the time of the Chief Commissioner's tour of the Northern Territories to suggest that, owing to the prevalence of the epidemic at that time in the Lorha and Tumu districts, it would be inadvisable for the chiefs or headmen of the villages to travel to these stations for the purpose of meeting the Chief Commissioner as it would in many cases entail their sleeping at infected villages on the road, and so possibly spread the infection. This suggestion was acted upon and any large congregation of chiefs at Lorha and Tumu was avoided.

11. I also suggested to the Chief Commissioner, while at Lorha, that it would be unwise, while the epidemic was on in the Harmattan season, to allow any gang of natives from the infected districts to be taken down country for work at the mines in Ashanti and the Colony, owing to the grave risk of introducing the infection to those parts. The Chief Commissioner agreed with the suggestion and no men were sent.

In conclusion I wish to acknowledge the willing assistance I received from all Medical Officers in the districts, particularly Dr. Lunn, Dr. Mayer, and Dr. Cope and also from Major H. Walker-Leigh, the District Commissioner of Lorha, who gave me every assistance in his power.

I wish also to lay stress on the fact that severe epidemics of Cerebro-Spinal Meningitis such as have occurred in West Africa for certainly the last three years, cannot be considered apart from the grave epidemics of the same disease which have occurred so recently in many parts of Europe, including the United Kingdom. Amongst the civilized peoples of Europe every step can be taken to arrest the progress of the disease, but it must be admitted that it is at least possible that the disease in West Africa, similar as it is in cause and effect, may act as a "feeder" to that in Europe and so tend to prolong its presence there. There is direct or indirect communication by means of caravans, which may convey the infection, between West Africa and many parts of North Africa from which the disease may be carried by ships to the sea-ports of Europe, where it is frequently found the epidemic starts. Whether the infection is also air-borne has not been determined, but the direction of its spread in West Africa—roughly that of the prevailing wind—suggests that it may be to some extent, although its main mode of conveyance is by "contacts," as described.

It is therefore, I submit, of the utmost importance that every precaution possible should be adopted to arrest the disease in West Africa as far as can be done. I have described the steps taken for this purpose during the present epidemic, and I suggest that in any future outbreak they be rigorously enforced, together with any other means which may appear advisable—such, for instance, as nasal disinfection, a measure which is however entirely impracticable at present among natives.

Review (24)

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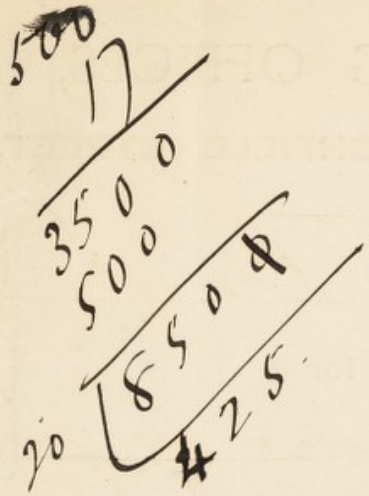

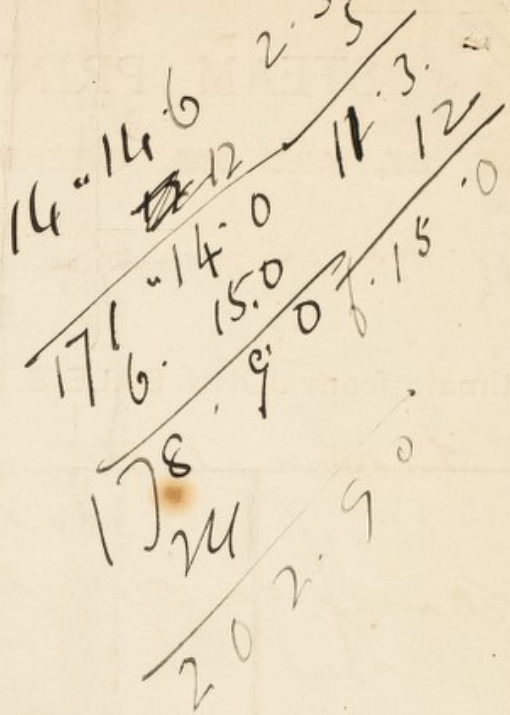

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