Collected papers on tropical medicine

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1729
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COLLECTANEA: Tropical Medicine.

1) BRADDON (W.L.). The Beri-beri piracy. 1911.
2) BRONN (A.). Trypanosomiasis in N.E. Rhodesia. 1911.
3) BRUCE (W.L.). Zambesi ulcer. 1911.
4) BRUNWIN (A.D.). Santonin treatment of Dysentery. 1908.
5) DUNCAN (A.). Amoebic dysentery. 1908.
6) JONES (A.W.). Bilharzia of the large intestine. 1910.
7) McCULLOCH (H.D.). Roentgen rays in Malaria. 1911.
8) MALARIA. Notes on cases. 1911.
9) SMITHSON (O.C.). Mossman fever. 1910.

Correspondence Bourg

89186

1729/1

Mount Beryl.

Seremban, F.M.S.

11th April 1911.

To the Editor of the Journal of

Tropical Medicine and Hygiene.

The Beri-beri Piracy

Sir.

If Science be the exact expression of facts accurately ascertained, then the leader which appeared in your columns on "Tropical Research", in your issue of March 15th last, in its reference to Research done in ~~regard to~~ Beri-beri deviates so far from the scientific normal that perhaps you will not resent my criticising a passage in it which, as a statement, is inaccurate, and (what I feel you would care for more) in its implication is unjust.

You refer to Drs. Fraser and Stanton as having "done excellent work as regards the part that rice plays in the spread of Beri-beri" What have they done?

It will be news to you and to many of your readers no doubt to learn that Drs. Fraser and Stanton have, so far, contributed not a single new or original independent observation of any facts determining whether it be the origin or "the spread" of Beri-beri.

It is true that after I had published evidence establishing the position of Beri-beri as a disease (not due to infection (as commonly believed) but ~~as a~~ the result merely of eating a certain sort of rice, and when I had obtained from the local Government an opportunity to demonstrate by crucial experiment the truth of that view, Drs. Fraser and Stanton were associated with me by the Government in the experiment ~~by the Government and~~ ordered to report on the results obtained.

— the Durian Tipus Enquiry —

That experiment was (as it had been hoped it might be) completely successful.

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demonstrated under conditions of control,

It ~~proved~~ once more, what I had already proved ~~////~~ by evidence (at least as it happens here) far more extensive, that in the production of Beri-beri the one + only essential causal factor is the consumption of uncured rice.

Being successful Drs. Fraser and Stantin reported on the experiment in that sense to Government -- but they presented the results to the public as entirely their own!

Nowhere in the text of the two publications which they made nor in numerous public repetitions of them afterwards, of the results, did they refer to the present writer by whom both officially and in fact the observations in part at least were actually made, as having either initiated or contributed to the result!!

It is true that Drs. Fraser and Stanton have done some excellent work on the chemical differences which exist between rice which does, and rice which does not produce Beri-beri; and in confirmation of Eijkman's well known observations on the production of polyneuritis from rice in birds.

But as regards the practical, the epidemiological aspects of the Beri-beri question they have (outside the unamiable and astonishing piracy of my results just mentioned) added nothing new or original to the issue.

When you say therefore in your Editorial " it is no exageration to say that the following up of Dr. Braddon's original idea (!) by these two men (!) may in time save thousands and thousands of lives" etc. the ~~implication~~ suggestion that my part was merely to have furnished an idea, a surmise, which these two men had the greater merit of translating from regions of ~~supposed fancy~~ pure theory into fact, of practical application, is an implication which does great (although I am sure unintentional) injustice to myself, and ~~even~~ also to others.

For, in the first place, that Beri-beri arose in some way from rice eating was never claimed by me to be my own original idea. Van Dieren + others are in print to the contrary.

But I did claim and still do claim, that I first brought forward evidence which appeared irrefragable + has indeed never been contested which proved that theory beyond all possible doubt.

Moreover the remedy, the simple, inexpensive, but wholly efficient remedy, by which Beri-beri may be everywhere completely ~~xxxxfixxx~~ prevented was first indicated by me. That remedy is merely the substitution, wherever rice forms the staple of diet, of the cured for uncured sort of grain.

The remedy was recommended by, and it was ~~It has been~~ adopted by the local Government of the Strait Settlements and the Federated Malay States in all their public institutions, before ever Drs Fraser or Stanton appeared upon the scene. Its success was such that Beri-beri, long the scourge of Jails and the Asylums, which it formerly had converted into death-traps and shambles disappeared from them instantaneously, and completely, and for the first time in their history, merely on the substitution of cured for uncured rice.

In the hospitals too, among cases already admitted for Beri-beri, the death-rate formerly in many cases reaching such apalling figures as 30, 40. and even 45%, was reduced by the same simple means to rates of no more than 10% or even 5%!

Your hope, that "thousand and thousands of lives may in time be saved" is not exaggerated-- they have already been saved, are annually being saved by the discovery, which is not, however, the work of Drs. Fraser and Stanton.

I am
Sir,
your obedient servant

W. Lennard Braddon

3

1729/2 O.C. 89186

O.C. Trypanosomiasis in North Eastern Rhodesia.

by Alexander Brown, M.B., Ch.B., Serenje.
~~and John Rennie, D.Sc. Aberdeen.~~

The following record of two cases of quite recent occurrence of human trypanosomiasis from the Luangwa valley, N. E. Rhodesia supplies further evidence of the rapid spread of this disease in this region.

(Muemye)

Case 1.– Mwemaye sent to Hospital at Serenje for examination having left of his own accord a proscribed area in the Luangwa valley, N.E. Rhodesia. The patient declares himself perfectly well and strong and regarded the examination in a humourous light. He looked healthy, although somewhat thin, with individual muscles rather flabby. He explained his condition as due to hunger.

There was no evidence of heart or of lung disease. Respiration normal

Pulse 80, thin and feeble.

Temperature 96° F.

Glands in neck, axillae, and groin were all enlarged. Those on the left side of the neck

were palpable but not dunctunable. Those on the right side were much larger. One in particular was larger than the others freely movable and very soft as if it were suppurating.

Both tonsils were suppurating

Microscopic Examination. – A little juice was taken from the enlarged glands and numerous trypanosomes were found. Some fields were without, in others 3, 4, and 5.

The blood was then examined and several trypanosomes were at once found in the first fresh specimen. These appeared slender with free flagellum and were exceedingly active.

Noticeable features are the apparent health of the man. He bolted, after examination but has since been found. There is no doubt as to the feeble pulse or low temperature. Thermometer was tested on the dispensary boy and found quite accurate.

The only tsetse fly occurring near Mwemye's, ~~Mwenzi's~~ home is G. morsitans and he has never been further from it than Fort Jameson. There are infected areas 50-60 miles both north of his district – Petauke and Nawalia, but he has never been near either of these.

Case 2.– Kawimbi. Sent to Hospital, Serenje, because he was ill, and in the habit of falling asleep.

When ~~seen~~ first (January 1911) he had been ill for more than a month. He left his home on the Muchinga mountains and went down to the Luangwa valley four months ago. He travelled south to the Sasare mines, went east to Fort Jameson, then to Blantyre in Nyasaland. On leaving Blantyre to return to Fort Jameson he began to feel ill. He passed through Fort Jameson on his way west to Serenje on the Muchinga mountains. Shortly after he was brought to Dr A. Brown (Serenje).

The patient looked ill and had certain characteristic signs suggestive of Trypanosomiasis. He complained of pain in the head and body. There was slight wasting of the lower extremities. He had a dull and heavy look and turning his head from time to time would close his eyes as if tired. The intelligence of his answers showed that the dull look was not habitual to him. His speech was thick; his tongue had a fine tremor. Anaemia was pronounced. There was no oedema. Pulse 80, small and thin. Temperature 96°.2 F.

The glands on the left side of the neck were slightly enlarged, those on the right side very much ~~larger~~ so. There was no throat or other condition which might

have accounted for them as in Case 1.

Microscopic examination.—The fresh blood films showed numerous trypanosomes, with extremely active movements.

Comparative features of interest in these two cases are the low temperature, feeble pulse, and enlarged glands of right side which were common to both, whilst in ~~case~~ No1, who showed no signs of suffering, the parasites were more numerous than in No2 who was very ill.

On March 16th, Mwemye (Case1), still maintained that he felt quite well; Kawimbi (Case 2) is going steadily down hill at this date.

Note on the Trypanosomes

(by J. Mennie D.Sc., Aberdeen)

~~The~~ An examination of a number of blood films ~~of the two cases~~ stained with Giemsa from these two patients has confirmed the presence of Trypanosomes in both ~~of these~~ cases. ~~as~~ In neither are the parasites numerous, although it is correct that they are more numerous in the blood from the apparently healthy man than from the sick. Roughly they

may be estimated in the proportion of about 3 to 1.
In Mwenye's case apparently dimorphic forms occur, slender and stumpy.
In both the kinetonucleus is large and the free flagellum long. In some stumpy forms the latter appears short but whether in such cases the terminal portion is simply obscured is not certain. In Kawimbi's case the parasites seem more of an intermediate type as regards shape, but all seem to possess the long free flagellum.

The species may be either T. *Sambiense* or T. *brucei*. ~~It would~~ The very distinct character of a long free flagellum appears to differentiate ~~the~~ the forms seen in both cases from T. *rhodesiense* n. sp. ~~which~~ in which, judging from the figures of this species reproduced in Bull. Sl. S. Bureau No. 22. p 399, an extremely short flagellum or its entire absence appears to be a feature.

1729/3 (1.)

89186

Zambesi Ulcer

A short description of an African disease.

Since coming to the Zambesi delta I have been struck by the number of cases one meets of an ulcer which I do not remember to have seen before nor to have found described in any publication.

The main points about it are:

(1) ~~That~~ It is met with with rare exceptions in one part of the body – below the knee.

(2) ~~that~~ It is usually single rarely double and more rarely in the form of two or perhaps three small ulcers on the same leg.*

(3) ~~that~~ It does not spread but exhibits immediate sloughing of the area attacked remains a week or more and then heals by granulation.

(4) ~~That~~ It produces no constitutional disturbance nor enlargement of the lymphatic glands.

(5) It is invariably associated with the

(1.) Situation. The variety of appearance anywhere but on the foot or lower two thirds of the leg is striking and should throw some light on the manner of transmission

The disease chiefly attacks field workers and they certainly get more cuts and scratches about the legs than elsewhere, but there are other breaches of surface and if the process were a direct contagion one would expect to find a proportion of cases where the lesion was on the face, trunk or arms owing to the custom these people have of sleeping packed together, wearing one anothers clothing and rolling practically naked on the ground where the discharge must often have contaminated the earth the mats and so on.

(2.) Description. In its typical form it is a single shallow, punched out, round or oval ulcer about the size of a florin, with a slightly undermined edge, a soft base and a flat purple coloured floor. A few show a fungating mass, others vary greatly in size — from

a hole in which a large pea would fit to a patch extending more than half round the leg — others, but they are rare, are multiple and then the individual ulcers are small.

3 Progress. I have not seen the onset but there appears to be a tender oedematous local swelling one day and the next a slough in the middle of it. This disintegrates and the cavity left — which is only skin deep but looks deeper on account of the raised edge like that of the ankle hole of a Cline's splint, fills up with a purple gelatinous material resembling the blood stained mucus in a dysentery stool. In a smear it looks like sputum and is almost entirely free from bacteria except the two mentioned.

The swelling gradually subsides giving a flatter appearance, the colour becomes bright red, and granulation finishes the process of repair. The active stage is sometimes over in four or five days, more usually ten to fifteen but may

last two or three weeks, the longer stage being the period of granulation which is slower than that where an equal area of skin is destroyed by injury.

When a toe is attacked it frequently heals without terminating the disease the result being an alarming swelling of the foot that breaks through somewhere else or causes the toe to break down again. Some cases would give the impression of spreading if not seen every day as after remaining unchanged with an unhealthy edge for days they suddenly enlarge by the sloughing of a ring round them after which the larger ulcer so formed heals.

3. Effect Although the neighbouring glands are not affected in the ordinary way a mass in the crural region may be infected with or without an ulcer, forming what is locally known as a "boil" and either subsiding again or suppurating

Some Observations on the Santonin Treatment of Dysentery.

By. A. D. Brunwin. M.A., M.B., BC (Cantab) Late ~~Medical~~ Resident Medical Officer, Colonial Hospital, Suva, Fiji.

and Hygiene

In the Journal of Tropical Medicine, of November 1st 1907, Dr. D. J. Drake of Teypoor, Assam, describes a method of treating dysentery by means of yellow santonin.

From the statistics published this line of treatment appears in his hands to have given far better results than either ipecacuanha, salines or bismuth.

On one estate the average number of days in hospital of dysenteric patients was 6·58 and the mortality 3 %, when treated by santonin; of those otherwise treated the average stay was 13·1 with 13 deaths. On another estate the difference in favour of santonin was even more marked.

The disease treated appears to have been the bacillary form of dysentery. There is no suggestion as to how the drug acts but presumably it may be looked upon as an intestinal disinfectant.

(2)

The method recommended was to give 5 grains of yellow santonin three times a day on alternate days, in 2 drams of olive oil.

The description of this method of treatment led me to try it for ~~one~~ nearly 4 months at the Colonial Hospital, Suva, during my appointment of resident medical officer there; for the preceding 2 months I had used either ipecacuanha or salines, and occasionally bismuth. I am so far satisfied with the santonin treatment as to use it in preference to any others, although my statistics on the subject do not show any marked difference between this and other methods.

Dysentery in Fiji is almost confined to a period commencing at the end of September and ending early in May. There is generally no marked rise of temperature, and the disease is very rarely followed by tropical liver abscess; and I believe that amoebae, dysenteric have never been found in the faeces.

From the course and symptoms of the disease it appears that practically all cases of

dysentery in Fiji belong to the bacillary type.

My statistics of cases treated are roughly as follows:-

Of 66 patients treated by ipecacuanha, salines, and bismuth the average stay in hospital was 11·26 days with 4 deaths.

Of 62 patients treated with santonin, the average stay in hospital was 11·11 days with 3 deaths.

Though these statistics are slightly in favour of the santonin treatment, yet I believe its actual value is greater than would be deduced from these alone. It is, in the first place, difficult to say exactly when a case of dysentery is cured; depending on whether it is considered that the patient has recovered when the blood and mucus have disappeared from the motions; or not until he is actually discharged from the hospital.

For the most part I have taken the number of days that the patient was in hospital, except where the patient was suffering from an intercurrent or subsequent illness. Again, the length

(4)

of stay in hospital depends on the ~~later~~ prospect of the patient on leaving, whether he can rest, or will have to work immediately. I often insist on the latter class staying in hospital longer than is absolutely necessary for the above reason.

Also, the lines of treatment have not, in many cases been consistently followed, as various symptoms may call for a change of drugs given; this was more often the case in the tentative stage of santonin treatment, as some apprehension was felt as to its effect on the patients' general condition, and possibly distrust as to its anti-dysenteric properties.

The above reasons will therefore cause some errors in the statistics, particularly as no special notes of the cases were taken at the time.

Even though there were no difference in the length of treatment and mortality I should distinctly prefer santonin to either the ipecacuanha or

(5)

saline treatment. The former is very depressing and unpleasant for most people which is obviously undesirable in those already suffering acutely; while the constant evacuations and anal irritation caused by frequent doses of salines, also increase the patient's distress.

Santonin appears to be free from these defects, and is at the same time quite as useful, if not more so, in combating the disease.

In all cases I have given it as recommended by Dr Drake, namely 5 grains in olive oil three times a day on alternate days, and have always used yellow santonin.

This is continued in every case until the faeces become free from blood and mucus and normal in colour. This generally happens within a few days, and I usually given bismuth and salol for two or three days to follow.

6

The treatment may be combined with washing out the rectum but I have never found this had any advantage in acute cases.
The treatment with santonin is usually preceded by a statim dose of sodium sulphate, 4 drams.
Of course the patient is kept at rest as far as possible; the diet being milk only.
I have seen no bad results from the administration of santonin as described above. There has not even been any complaint of yellow vision, and no cardiac depression except in those severe cases in which it occurs as part of the disease. Even in these cases santonin did not seem to accentuate the depression in any way.
Of the three dysenteric patients who died while having santonin treatment, one was a European with congenital cardiac disease; another was a Solomon Islander with a very

acute attacks who could not be persuaded to stay in bed. For the other death during santonin and for the 4 deaths under other treatment, no special reason could be given except failure of the drugs to control the disease

Besides being more comfortable for the patient, the santonin treatment has the advantage that it is easy to administer, and the patient requires less constant attention than in treatment by ipecacuanha, salines, or enemata. This is a distinct advantage where skilled nursing is not in proportion to the number of patients or is entirely lacking, as in many districts in the tropics.

Two patients under santonin treatment developed acute non-suppurative arthritis towards the termination of the disease. One of these, a European, had been for years a constant sufferer from sub-acute & chronic rheumatism,

the other was a Solomon Islander who had had no joint trouble previously. These were the only complications noticed in any of the cases.

To sum up; I am distinctly in favour of yellow santonin for the treatment of dysentery, both on account of it being more comfortable for the patient to take, and more easy to administer than other forms of treatment. ~~Statis~~ My statistics also show a slightly shorter course and lower mortality; but I believe the ~~course~~ duration of the acute symptoms to be still shorter than the statistics would appear to indicate.

Some Observations on the Treatment

of Dysentery by Santonin.

1 O.C. 1729/5 89186

Amoebic Dysentery

by

Andrew Duncan M.D. B.S. (Lond) F.R.C.P. F.R.C.S.

J. S. F. aet 19, a lieutenant in the Royal Navy consulted me on Sept 16 for amoebic dysentery from which he had been suffering for 5 months. During this time he had exhibited the features of the disease as described by Drs Councilman and ~~Laffleur~~ Lafleur in their classical paper in the John Hopkins Report for 1891; ~~namely the motions had been constantly~~ no solid motion had passed for the first months, the dejecta being of a light brownish or greenish character, sometimes with blood, but more often the latter was absent. ~~He was~~ Exacerbations followed by alleviations of the symptoms occurred as the disease progressed – on ~~Ttrans~~ he was in sick quarters on H.M.S Alexandria. After six weeks July 1 had elapsed, no improvement having set in, he was transferred to the hospital at Port Said, where he stayed for three weeks, and was then invalided home as the symptoms had by this time practically disappeared. On the day of his landing in England he again had a relapse, and was admitted into the Naval hospital at Plymouth for a week, when the symptoms once more ~~abated~~ abated. He reached his home on Sept 6th. The bowels now not having been opened for 48 hours, he was given some ipecacuanha, which brought on once more the loose motions with mucus. This looseness was absent during the day, but always came on at night, the bowels being opened about four times ~~each~~ each night.

2 He had not had a normal solid motion for four months when I first saw ~~home~~ him
He had been treated for amoebic dysentery, according to the plan advocated in
America, namely with rectal injections; ipecacuanha was not administered.

As the rectal injection had not stayed the progress of the disease, I
at first gave him izal, which drug has been strongly advocated in India
lately – this had no effect. After taking the izal for nine days, I began treating
him with 9 grain ~~large~~ doses of ipecacuanha three daily – The effect at once was most striking. The
motions gradually cleared until on Oct 10th he passed his first ~~stool~~ solid motion.
Since this date he has had no more loose motions but rather a tendency to
constipation. His personal appearance is very different to that present on his first
visit to me, being now that of a boy in perfect health.

I think this case worthy of record for the following ~~several~~ reasons. First, the immediate
cessation of the symptoms on the exhibition of ipecacuanha was most striking.
Secondly the case with the practice of other physicians in England would seem
to show that ~~the amoebic dysentery~~ the treatment of amoebic dysentery varies
with the country in which the disease occurs, in the same way as the
treatment of bacterial dysentery varies with the country in which it occurs. Thus
ipecacuanha which is so successful with regard to the latter, as is well known,
in India, was of ~~no~~ scarcely any avail in South Africa – Sir Frederick Treves
has informed me that it was given up, and the cases treated by saline. Again
in South Africa monsonia ovata gave remarkably good results under Dr
Maberley: it was found to have no effect at the Branch Seamen's Hospital
Albert Docks. In Malay lavage of the intestines was found to have
superior efficacy over ipecacuanha in

patients.

Returning now to the treatment of the Amoebic variety of the affection. The experience of American Physicians has been until recently against ~~the~~ any value to be obtained from ipecacuanha. Dr Strong in his very comprehensive article in Professor Sir Wm Osler's System of Medicine states that local treatment by rectal injection of quinine and irrigations gives by far the most efficacious results: in the last Edition ~~of Osler's own text~~ of the Principles and Practice of Medicine by Osler, ipecacuanha is not mentioned in the treatment of this form of dysentery, but the Strong, he holds quinine injections to be the best. In the before mentioned article of the John Hopkins Reports, quinine injections appear to have been followed by the most satisfactory results. Sir Patrick Manson on the contrary now holds ipecacuanha to be a specific for amoebic dysentery; in the early editions of his work on Tropical Diseases, it would appear that he was not decided whether amoebae really ~~were the cause~~ were the secondary cause of the disease. And in this ~~[illegible]~~ view, I for a long time agreed with ~~his first views~~ him. During the whole of my service in India, I never saw a single case of amoebic dysentery, that is to say ~~dysentery~~ dysentery showing a long course and with the ulceration similar to those described in the J. John Hopkins Reports ^ — Colonel Buchanan, that indefatigable investigator ~~into~~ into Tropical Diseases in India, ~~[illegible]~~. During the last [illegible] held that amoebae were only concomitants. There have been however one or two cases reported in the Indian [illegible] of success obtained by ipecacuanha. How are we to explain

^ nor have I ever seen the peculiar [illegible] appearances that have been described

4

these different results? The only explanation which seems to me is that the type of the disease as seen in America differs from that seen in England –

Lastly the term Amoebic or Tropical dysentery has always seemed to me to be a misnomer, at any rate as regards India – The most frequent form of the disease, in fact according to my experience, the ~~only~~ only form of the disease, seen in the United Provinces and in the Punjab has been bacillary dysentery. Professor Leonard Rogers states that this is the most ~~frequent~~ frequent form in his experience – ~~[illegible]~~ I do not know whether this be the case in other tropical regions, but if so, it would seem that Tropical dysentery is more closely associated with bacteria than with amoebae as a cause

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Bilharzia of Large Intestine

In the interesting article by Mr. Owen Richards on Bilharzia of the large intestine contained in your issue for March 15th a note is given of a case treated by appendicostomy. As the application of this operation to the condition under discussion has no doubt been up to now but rarely employed, the following somewhat sketchy notes of a case of my own may be of interest.

The patient, a male Egyptian aged about forty-five, was admitted on the medical side of the Government Hospital in the Summer of 1907 under the care of Dr. Betts. He complained of frequent bloody stools with abdominal pain, the latter symptom being the one from which he was most anxious to obtain relief. Ova were detected in the rectum & Bilharzia ova in the stools. No tumour could be detected in the abdomen. Medical treatment failing to afford him any relief he was transferred to my wards & on June 30th I performed an appendicostomy. Recovery from the operation was rapid & through the fistula we were subsequently able to irrigate the large bowel with Silver Nitrate (1 in 4,000), Normal saline and

other solutions. The case was presented later at the Société Medicale d'Alexandrie to show the technical advantages of the operation apart altogether from the question of the efficiency of its application to Bilharzial disease At first there was no improvement in the number and character of the stools whilst his ~~weight went~~ he lost slightly in weight. We were with difficulty able to keep him under observation in hospital until Oct 9th but he then insisted on leaving and as he belonged to the class that live on charity ("at the door of Allah" as they say) I have since lost all trace of him. At the time he was discharged from hospital I made the following note:-

"Looks well - no pain - no blood nor pus in stools (examined by Dr Husseinein) - Stools are well formed & about three in 24 hours - He was extremely undiscreet in his diet. Weight 112 lbs only."

My idea in doing the operation was that by ameliorating the symptoms we might prolong the patients life & thus tide him over the date when the parent worm by dying might bring about a natural cure

In my case the appendix affords some difficulty, being very long, narrow & firmly fixed behind the caecum. Indeed half way through the operation I felt severe misgivings lest we I was not exposing my patient to a risk unjustified by the problematical nature of any possible benefit: If it can be proved therefore that a majority of these cases present unusually long and adherent appendices the operation itself will have to be regarded at any rate as a somewhat less innocent and harmless procedure than it is usually taught. When we consider that the ova are ciliated deeply to the mucous membrane one can scarcely hope for great benefit from topical applications even though they be applied by the medium of an appendicovalonic fistula. Of course in my case the parent-worm remains unbroken. One must also take into consideration that the class from which most of these patients come — namely the fellaheen — are not conspicuous for their intelligence, ~~in fact I can scarcely picture a fellaha reclining on his divan~~ and

I doubt whether one could explain satisfactorily to a fellaha how he might to take advantage of the fistula: & indeed cannot picture them reclining on their divans, irrigating their colon with a douche can suspended from the roof of their mud hut.

In short - whilst admitting that theoretically there is something to be said in favour of the operation, I cannot think it is likely to prove of much practical benefit. For my part the case would have to be very specially selected that would tempt me to repeat the procedure.

A Webb Jones F.R.C.S.
Surgeon & Gynaecologist to
Govt Hosp
Alexandria
Egypt.

O.C.

1729/7

8186

Curative influence of Roentgen Rays in Malaria.
By H.D. Mc Culloch M.B., M.S.(Glas)

The orginal article under the above title, by Lt Col Bruce Skinner M.V.O. R.A.M.C and Lt H.W Carson R.A.M.C, which appeared in the British Medical Journal of the 25th Feby last (p431) is one, that not only opens up a new and important field, in the early treatment of those essentially blood infections, in which the spleen, the liver, & to a lesser extent the pancreas, is involved, but it also throws interesting light, upon those infective processes which ~~are~~ the result of infections, which are not intravascular & of the blood primarily, in which the lymphatic glands, which are distributed in so many parts of the body, become involved. As examples of the former, we have septic endocarditis & malaria, & of the latter Tuberculosis and syphillis.

In 1906 I made a contribution to the Lancet, entitled "Observations on the induction of auto vaccination by x ray inadiations of the lymphatic glands in Tuberculosis, & other glandular infections, as revealed by the opsonic chart."

That work received corroboration at the hands of Dr Lawrence & Crane of the United States in Tuberculosis, and with Messrs Paoli & Nuncioni of Italy in primary syphillis.

Now the physiological relationship of the spleen & the bone marrow on the one hand, and that of the lymphatic glands on the other, have been well known in connection with the reactive processes which are termed inflammatory. There is deposited in these various glands, masses of neoplastic cells, which increase the bulk of these glands, greatly hampering their physeiological functions, particularly where the microbic invasion is sudden & extensive, as in the early

Summons 241

Early Stages of malarial fever during the period of sporulation. The resulting conditions are variously termed Splenitis, hepa-titis and adenitis &c.

In dealing with these conditions we are concerned with relieving distressing symptoms & the elimination of the cause, which, up to the present time, we have been able to achieve with more or less success. But of the resulting fibrositis and induration, this has been left to time & nature, to deal with. The repression of fibrositis, or the regulation of these reactive processes, in the early stages, has not been possible, beyond what has been achieved by fomentations poultices & packs, and a recourse in the later stages to arsenic & the iodides. The value of the time honoured & judicious use of quinine, when administered by subcutaneous injection for preference, as a malarial parasiticide is not disputed, but in dealing with these conditions & their sequelae, it will be admitted, that much remains to be done, that it has not hitherto been possible to do.

Medicinal fibrolytic, alterative & resolvent remedies, have been tedious, irksome & disappointing in the majority of cases.

In the x rays, we now have a means of achieving these much to be desired ends, with absolute safety to the patient, & in a short time.

No doubt, what occurs after recovery from these fevers, is a crippling of portions of these glands, & a compensating hypertrophy in those directions where the gland parenchyma has escaped the constricting effects of fibrositis, just as when one kidney is surgically removed, its fellow undergoes functional hypertrophy to meet the needs of the body, this being a true hypertrophy & not a fibrositis.

In 1907-08 three cases of splenic & hepatic hypertrophy, the result of malaria in Englishmen, came under my treatment by the X Rays, and in one, who was able to meet the extra cost, a series of blood examinations were made previous to, during & after the treatment. This was a patient of my friend Mr Neil Macgillycuddy of Bournemouth, who sent him to me at my suggestion. His anaemia was his chief complaint, though there was marked enlargement of both spleen & liver. His colour index soon rose from a little above half the normal, to 88%, & this improvement, synchronized with a marked reduction in the size of his spleen & liver. The blood examinations were made for me by the Bournemouth Borough Bacteriologist, & by the Clinical Research Association. In all three cases the results were very gratifying.
I happened to visit London shortly after these experiences, & mentioned them to Mr James Cantlie & Sir Patrick Manson. The former lent me his ear, & I was pleased to hear from him, that his friend Dr Ironside Bruce had also had similar success with enlarged spleens, I cannot recall whether he referred to leucocythemic spleens as much excellent x ray work in this connexion had already been done. But they are two very divergent pathological states, that of leucocythaemia being more comparable to the status lymphaticus, while that of malaria is a pseudo-reactive hypertrophy, and one that is far more amenable to X Ray Treatment.
The authors of the paper above referred to, unfortunately, neither enter into the x ray technique adopted by them, nor do they seem to have made any blood examinations, but to me their remarkable experiences are very convincing, since my experience with the X Rays, which began in India with oriental cases in 1899.

The authors conclude their paper, by stating that they also have five cases of chronic enlargement of the spleen after malaria & that in these cases they are not able to speak yet with any certainty. Such however are the cases which I have dealt with & they afforded the most gratifying results.

I regret I am unable to accept the suggestions of the authors, in regard to the essential therapeutics of the x rays, which they compare with the effect of heat, with which there is not the remotest analogy. In the x rays we have, what has been termed, a 'fourth state of matter', a most potent molecular agency, which influences cells according to their individual molecular complexity and their relative instability. The more recently & rapidly developing cells are more influenced in regard to their resolution, than are those that have become 'fixed' & more relatively stable. Spermatozoa, the lymphocyte and the neurone, are some of our most complex cells, but micro-organisms, even of a pathogenic kind, are not so influenced, because of their lesser molecular complexity & relative stability.

It may be asked, how then do you account for the X Ray burn or the X Ray cancer, so called? It is well known that that the rapid abstraction of heat by the application to the surface of a piece of CO_2 snow for a few minutes, instead of a few seconds, will also cause a burn or frost bite, which is followed by extensive necrosis, just as the injudicious use of too prolonged & unfiltered x rays will cause, similar changes without any appreciable trace of heat reaching the skin.

H. D. W. Cullough.

1729/8

89186

1911

May 1st Mr Hardie Philips – a tall thin lanky youth, very anaemic – age about 22 years, has been a few months in the country, takes 5 grs Quinine daily, does not know what preparation as he brought them from home, in the bungalow goes about with sarong & bare legs, legs up to knees covered with scratches, mosquito bites, has never had Malaria,

8th Fever, Temp 11 am 103° { Quin Hydrochlor grs XV, Aspirin 5 grs, Hot drink.
3 pm 101
5 " 102
7 " 103 Quin Hyd grs XV
10 " 101

9th " " 8 am 99°2
11 am Normal
5 pm 100° Quin Hyd grs XV
9 pm 101·4

10th 8 am 100° Quin Hyd grs V
12 101 ~~Homels Haematogen & milk~~.
4 pm 103·6. { Haemoglobinuria, Calcium Chloride grs XX, Sing 4 hourly, Quin Bihydrochl grs XV
5 pm 102. Champagne, egg flips.
7 pm 101·4 Brands Essence of Chicken
11 pm 101° milk ad lib – Urine Solid albumin. Haematogen & milk –

11th 3 am 101° Same treatment –
5·30 am 99·6 albumin 50% solids

May
11th Temp.

9 am	100°	Calcis chloride gr x every 4 hours.
12 noon.	100°·2	Tinc hydrochlor gtt v 3 times daily
4 pm	100°·2	
10 pm	99°·8	
12th 6 am	Normal	
12 noon	do	

13th all day normal. and remained normal until I took him to Kuala Lumpur Hospital for nursing on the 17th by which time his urine had cleared, only a slight trace of albumin being present - as there seemed every chance of his Kidneys suffering no permanent damage, I advised Harrison & Crosfield, to keep him there till he was strong enough to travel alone, then as it was May to send him home for the summer, before cold weather came on, have him examined in England, and if Kidneys all right send him out again, if possible putting him on a healthier estate - As you know I have in West Africa seen many cases of Blackwater Fever, with my Calcium Chloride & treatment I have never yet had a death. and I assure you this was a typical case of Haemoglobinuric Fever, cut-short by early application of what I consider the correct treatment - vide also Castellani & Chalmers -

4

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Mr Marshall - Manager - Ayer Angat Estate -
Age about 32 - anaemic, enlarged Spleen, heart weak & flabby -
has frequent attacks of fever. takes Quinine occasionally
Jennally for a week after each attack, then forgets it -
has been about 6 years out here, without home leave -
Last attack previously - June 11th to 21st Quin bihydrochlor
by intra muscular injection - 3 days running, then by mouth
10 gr daily -

Present attack
7th July Called 10. am temp 103°. vomiting, 2 Hyd. gr XV injection -
he telephoned 4 pm. very bad, Blackwater, at 4.30 I
found him collapsed, vomiting, rigors, heart's action very bad
Injected, Strychnine & Digitalin, at once, hot bottles, blankets
Calcium Chlor gr XXX in Chloroform water every 4 hours, and
2 injections Quin bihyd during the night, feeding, champagne
brandy, Chicken essence, ice, Haematogen from the start -
Temp Kept varying. 104° to 106°. urine Solid -

8th Temp still 105° inject Quinine gr XV at 4 am, but temp
kept up till 7 pm when it dropped to 100°. very bad night,
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9th At 6 am, temp 99°.2 better, taking food well, 10 pm. 100°.2

10th better night slept at intervals, has stopped vomiting & shivering.
temp Normal all day. takes nourishment freely every half hour
Urine clearing. pulse improving -

11th & 12th gaining strength - Temp Keeping normal, takes food well

13th still improving — urine clearing.

14th motored him to Station & took him up to K L Hospital -
his kidneys will want watching as this was a more severe
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25 Left F. M. S for Singapore en route to England

1729/8

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1729/9
Mossman Fever
Oliver Smithson.
89186

T.M. O.C.

1

Mossman Fever
by Oliver Smithson FRCS Ed.

A peculiar fever, which, to the best of my knowledge has never been described, is common in some parts of North Queensland.

It occurs mainly in the Mossman district which lies a few miles north of Port Douglas. For this reason I have given to it the name of Mossman Fever. Locally it is known as Filarial Fever — why, I do not know.

The disease begins somewhat suddenly & the patient complains of headache & a feeling of general malaise.

Slight shivering is common but actual rigors seldom occur.

The temperature within a few hours rises to 102° 103° or even 105°. The pulse rate is increased but not as a rule in proportion to the temperature.

The tongue quickly becomes coated with a thick moist white fur. In fact its appearance is characteristic. The breath becomes exceedingly foul.

Vomiting is occasionally troublesome & some patients complain bitterly of pain in the epigastrium

Within a day or two of the commencement of the attack the posterior or subscapular group of axillary glands is found to be enlarged & tender. The enlargement is not great; the glands seldom being bigger than an ordinary marble. They are not painful unless pressed upon, the patient is usually unaware of their existence until his attention is called to them.

In a few cases the superficial inguinal glands are enlarged — but this is exceptional.

The duration of the disease is generally from ten to fourteen days. During this period the patient's condition is unchanged, the temperature being from 101° to 104° at night & dropping a degree or two in the morning.

At the end of ten days or so the temperature drops more or less rapidly to a very short time the patient is quite well again.

The death rate is very low. Few fatal cases having occurred.

In these cases the patients all fell rapidly into a 'typhoid' condition. There was no delirium the patients were always rational; but were dull & lethargic. They

(2)

could be roused by speaking sharply to them, but immediately lapsed into a semi conscious doze.

In these cases the pulse was rapid & very weak & although the temperature dropped for a time the pulse rate could not be reduced & there was a rapid rise of temperature just preceding death.

No drug seems to have much influence in this disease. I tried quinine, salicylate of soda & salicylate of quinine & found them useless. A Simple Saline mixture seemed to do more good than anything — at least the patients ~~appeared~~ to appreciate it more than anything else, while ~~antifebrin~~ phenacetin certainly relieves the headache more than does antifebrin or antipyrin

I examined the blood of seven patients suffering from this disease. In a few instances I came across the Filaria Sanguinis hominis, but beyond this the blood appeared normal in every instance.

The chief industry in Mauritius is the growing of sugar cane.

(20)

This fever attacks the cane cutters almost exclusively. The farmers themselves & those men employed in the crushing mills almost always escape.
The inhabitants are inclined to blame the mosquito in connection with this fever, but I cannot think the mosquito is a factor to be reckoned with. Neither do I consider the disease to be infectious.
In the Port Douglas hospital one ward is set apart for this fever although mosquitoes abound, patients in the other wards seldom or never contract the disease.
Another fact worthy of mention — Mareeba is about 6 miles north of Port Douglas, & by air across the bay & about 14 miles by rail. In many instances I took a patient's temperature just before entering the train at Mareeba, & again an hour afterwards on admission to the Port Douglas hospital. In the vast majority of cases I found the temperature had fallen one or two degrees — occasionally it fell to normal, but the temperature rose always rise the next day. The medical officer of the Port Douglas hospital tells me

he has often noticed the same thing.
Although Port Douglas is so near Thursday Island the disease is unknown at the former place.
Another peculiar feature is this — cases admitted from certain Sugar Farms are nearly always of a mild type whilst those admitted from other Farms are of a severe type. In fact Some Farms have earned a most undesirable reputation in this respect.

Dr Clark whom was formerly in charge of the Mount Molloy District Hospital, which is about 20 miles west of Port Douglas, says a similar fever occurs there & is known locally as "Scrub" fever. Mount Molloy is over the Coastal range & is a mining district. Going further inland to Chillagoe, Mungana & the Mitchell River district the disease is not known — at least during a residence of nearly a year there, I never saw a case.

It seems possible that the fever may be conveyed by the bite of some insect found amongst the Sugar Cane, as the disease is confined almost exclusively to the Cane cutters, though the occurrence of the disease

in a mining district like Mount Morgan
& the absence of lymphangitis seem to
against this view.

(6)

AT HOME:
9—10.30
2—3
6—7.30

TELEPHONE 2623.

"Hanover Villa,"
Stanley Street, East,
Brisbane.

Sept. 22. 1910

Dear Sir

For the last few years I have been travelling about Cape York Peninsula I have come across a fever which I have never seen described, & which the the local medical men do not seem to have taken the trouble to investigate.

I enclose some notes on same & some characteristic temperature charts.

Should you think the notes worth publishing in your paper, I would be glad to receive a copy

Yours truly

Oliver Smithson

Addressed to the Editor of the Tropical Journal