To avoid misconception as to what I claim for the free antiseptic treatment of enteric fever, I will, before bringing this paper to a close, briefly state my case. The cure of any disease must be conditional—that is, we cannot truly estimate the value of any remedy we employ in disease unless on our commencing its exhibition the possibility of a cure exists. A fatal termination under any form of treatment is what, unfortunately, we have occasionally to expect, and for such an eventuality we must be prepared, even though our patient be at the time well under the influence of our pet remedy. Dr. Burney Yeo has very justly remarked “that occasionally fierce and destructive conflagrations occur which water will fail to extinguish. Yet none the less is water an extinguisher of fire.”

In connection with this observation I would say that more than once I have seen remedies tested in cases, obviously in a hopeless condition, and discredited because a miracle did not follow on their employment. I claim no “miraculous properties” for the antiseptic combination advocated in this paper, but I do maintain that its free use (in conjunction with the other items which, taken together, make up the antiseptic treatment of enteric fever) will reduce the mortality of that fever from 25 to 10 per cent., or even less. Surely that is no small gain!

CONCLUSIONS.

I will state my case in this way: Take 100 cases of enteric fever, as that fever is observed in India, and treat them expectantly with a well-arranged dietary and efficient nursing; from 70 to 80 of those cases will recover, leaving a mortality of from 20 to 30 per cent. Treat a second batch of 100 cases with the same dietetic and nursing arrangements, but instead of the expectant treatment, or rather in addition to it, employ the antiseptic system (which I may remind all readers of this paper does not solely consist in giving so many doses of an antiseptic mixture), and instead of a death-rate of from 20 to 30 per cent. we shall have something about 10 per cent. So much, and no more do I claim for the antiseptic treatment of enteric fever.

I will bring this paper to a close by stating explicitly and succinctly certain effects observed during the progress of the fever, which I ascribe to the method of treatment advocated in this paper:

1. Intellectual clearness of the patient, with very slight tendency to delirium or stupor.
2. Food invariably well assimilated.
3. Abdominal distension except to a very moderate extent, invariably absent.
4. Early cleaning and moistening of the tongue; dryness very rarely present.
5. Stools almost completely deodorised.
6. An offensive odour from breath or body of patient rarely observed.
7. Tendency to undue looseness of bowels rarely present.
8. Secondary complication of any kind never noticed.
9. The fever pursued its course “kindly,” with little distress to the patient.
10. Convalescence rapid and complete.

I was unable to satisfy myself that: (a) The duration of the fever was lessened; (b) that there was any marked and continuous lowering of the thermometric range; or (c) that an occasional tendency to relapse was removed.

A CASE OF PERNICIOUS BERI-BERI.

By A. JEFFERSON, M.D., B.S.LOND.,
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Now that the subject of tropical medicine is at last about to receive that attention which most assuredly it deserves, the following case of pernicious beri-beri seems worth recording. It also may not be amiss to recount the various stages passed through on the road to its diagnosis.

At the end of the year 1888 I went out to Manila, taking with me a fair experience of alcoholic paralysis and the knowledge that beri-beri was also a peripheral neuritis, which, however, sometimes assumed an oedematous form. Passing through Hong Kong I visited Mr. Ho Kai. From him I learnt that, owing to the efforts of our old fellow-student Mr. Takaki, beri-beri had been practically stamped

out of the Japanese navy, and that this change had apparently been effected by the introduction of a more nitrogenous dietary. Mr. Ho Kai also told me that a case of undoubted beri-beri had recently occurred in a member of the local British colony. Absolute immunity was not therefore to be obtained merely by the liberal indulgence in nitrogenous food, nor was the Britisher to be regarded as being exempt from the disease.

Arriving in Manila, and calling on Mr. Donelan, I was surprised when, speaking of the cholera epidemic of 1882 in that city, he informed me that an even more fatal epidemic of beri-beri had immediately succeeded it—an epidemic which, as I afterwards learnt, according to the estimate of Roeniger,¹ caused the death of 20,000 people, the mortality reaching 60 per cent. In this epidemic no children were attacked, but the susceptibility of women after parturition was very marked. As a result of this conversation I now modified my mental picture of the disease and began to associate its symptoms with the paralysis of diphtheria (which I then regarded as a peripheral neuritis) rather than with the neuritis of alcohol.

The first few months of my residence were consumed in acquiring a superficial knowledge of Spanish, together with my licence to practise. At about the end of that period I was consulted by a young half-caste lady, the wife of a patient, as to an ingrowing toenail. For this I performed partial avulsion under a local anæsthetic; and three days afterwards, calling to see her, I found her walking about the house in comparative comfort. Two days later I received at night an urgent message to visit her, as she was dying. Arriving at the house they told me that two or three hours before she had been seized with shortness of breath, and had lain down on her bed complaining of what was apparently an acute attack of indigestion. I found her sitting up in bed suffering from well-marked orthopnoea; her face blanched, and covered with cold perspiration; her pulse was 140, and her temperature subnormal. With difficulty she was able to ejaculate that she felt intense substernal pain, and that she was dying. Hastily examining her chest, I found dulness and absence of breath sounds extending posteriorly and equilaterally up to the angles of her scapulæ; while above, and over the whole of her thorax anteriorly the lungs were filled with minute crepitations suggestive of oedema; the cardiac sounds were practically inaudible. By chance some of her urine was obtainable, and with this I drove off to my consulting room, in order to test the specimen, and to fetch such remedies as might be necessary.

Examination of the urine showed absence of albumen, so, taking with me an aspirator, and such drugs as suggested themselves, I returned to my patient, pondering over the pathological obscurity of her symptoms, and wondering at the fact that I should have heard, for the first time in my life, what appeared to be crepitations characteristic of oedema at the apices of the lungs. The general appearance of the patient, and the suddenness of her attack, suggested pulmonary embolism, while the physical signs pointed to a partial and equilateral thrombosis of the pulmonary veins—a condition impossible to more than momentarily contemplate. Returning to the bedside I examined her legs, and found some slight oedema of both; her condition was now yet more grave, and within two hours she was dead. I certified the case as one of heart disease, and though by no means prepared to be cross-examined as to this, I remained unshaken in my belief for a space of about a fortnight.

During this period I happened to visit an acquaintance of my late patient, an elderly half-caste woman, who, referring to the phenomenon of the sudden end of one so young and apparently healthy, remarked that the cause was “certainly beri-beri.” This observation my limited knowledge of Spanish most fortunately compelled me to let pass—for my association of beri-beri with the neuritis of diphtheria had not gone so far as to admit of the possibility of death in a few hours in a case of the former disease. On this same day, interviewing my late patient's mother, I learnt from her that her daughter had suffered from slight oedema of the legs for some days before her death. How long she did not know; nor did my operation throw any light upon this, for, refusing to take chloroform, she had, with characteristic native modesty, merely exposed to my

view the affected toenail. Some days afterwards, going round the wards of S. Juan de Dios, I was shown a marked case of ascites in a beri-beric patient, and a remark was made as to the rarity of such cases and the relative frequency of pleural and pericardiac effusions.

I now consulted other local sources of information, and, finding that beri-beri was sometimes fatal in a few hours, with just such symptoms as my patient had presented, I began to suspect that the diagnosis of the aged half-caste might indeed have been the correct one. Subsequent experience and further information as to a feebleness in the patient's legs for a couple of days before death (obtained through the medium of her husband, who was away when the catastrophe occurred) later removed all doubt as to this.

At that time the only Philippine publication I could discover concerning beri-beri consisted of a contribution to the *Diario de Manila*, early in 1888, by Dr. Yglesias. In this he laid stress on three points: First, that the disease was of bacterial origin, the micro-organism being found in mud and commonly obtaining ingress, as in tetanus, by means of cuticular lesions; secondly, he described the symptoms as being due to a neuritis; and, lastly, stated that amongst them the chief characteristic was a myositis. At this time Dr. Miciano had only recently commenced his work in connection with this disease at the Hospital of S. Juan, into the wards of which I have a note that there entered in a little over five months, in the year 1887-88, 228 beri-beri patients, of whom 55 died.

But though there was then very little published in Manila in reference to beri-beri one found the local Spaniard, thanks chiefly to the writings of Lacerda and other South American observers, quite proficient in its symptomatology and not ignorant of its bibliography and of such work as we have done in the elucidation of the disease. Thus, in a series of articles as to the etiology of beri-beri published by Dr. Maceras in 1893, not only were the publications of Anderson and Simmons commented on, but a further reference was made to the work of Lind (1757), Clark (1792), W. Hunter (1804), Rogers (1808), and Marshall (1822).

Thanks to Dr. Patrick Manson and Allbutt's *System of Medicine*, our knowledge of beri-beri has been now brought up to date. Owing to Dr. Manson's able advocacy tropical medicine generally will no longer be treated with insular indifference. In time we may even see established at home a society modelled on the lines of the Colonial Society of Germany. In any case, however, one can foresee that the path of the British practitioner in the tropics is soon to be rendered much more smooth and pleasant to start upon. It is in order to possibly emphasise the necessity for this that I venture to publish this case.

REFERENCE.

¹ Quoted in the *Correspondencia Médica de Manila*, March, 1894.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

THE WET PACK IN BERI-BERI.

My only excuse for sending you the following notes is the success which attended the wet-pack treatment. I had not tried it in previous cases.

On September 12th, 1897, S. H., a fireman, complained of pain in the legs, difficulty in walking, fullness about the abdomen, and shortness of breath. He had some œdema over the crest of each tibia, and there was some effusion into the abdominal cavity, and œdema in the lumbar region. The temperature was normal, and the pulse 120. He was vomiting food. The urine was small in quantity and of a high colour, but contained no albumen.

The patient was isolated; and was ordered milk, beef tea, and lime juice, and the following mixture: Pot. nit. ʒj; tinct. scillæ ʒss; tinct. digitalis ʒviij; liq. ammon. acet. ʒss; aq. ad ʒj. Cap. ʒj tertiis horis. On September 13th the temperature was normal, the pulse 120, and vomiting had ceased. On September 14th the morning temperature was normal, and the pulse 125. He was vomiting everything. The urine was scanty and high-coloured, but contained no

albumen. He was ordered milk and soda water. The evening temperature was 100° F. He was put in a hot wet pack, and left in it for about half an hour. He perspired freely, and his temperature was normal soon afterwards. The next morning he felt better, and had not vomited. The temperature was 100° F., the pulse 130, rather incompressible; the urine scanty. The kidneys were dry-cupped, and a poultice applied afterwards. The evening temperature was 102° F., and the patient was put in hot wet pack again, and perspired freely. On September 16th the morning temperature was 98.8° F., the pulse 130. The œdema did not appear to be less. The evening temperature was 101.2° F. He was again put in the wet pack. On September 17th he felt much better, although there was a good deal of œdema still. The tension of the pulse was much less, and he was passing a fair quantity of urine. The temperature was 98.6° F., and the pulse 120. The patient made a good recovery, and resumed duty on October 1st. The œdema had all disappeared.

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HYPERPYREXIA IN ACUTE CROUPOUS PNEUMONIA.

On April 5th I was called to see W. M., a young man 28 years of age, of distinctly nervous temperament. He was suffering from croupous pneumonia of the lower lobe of the left lung, evidently consequent on an attack of influenza. The disease ran its ordinary course till the morning of April 10th (the sixth day of the pneumonia), when after a restless night my patient became drowsy, and when I saw him at 3 P.M. he was quite unconscious, breathing stertorously, and was just recovering from a convulsive seizure. I found his temperature to be 108.6°. I had no other thermometer at the time with which to check this result, but I subsequently compared the thermometer I used with three others, and found them all to correspond. The pulse was about 130, and rapidly failed, so that my patient died in half an hour without regaining consciousness. The highest temperatures in acute pneumonia that I can find recorded in the authorities at my disposal are 109.4° (fourteenth day) and 108.9° (seventh day).

Fochabers, N.B.

S. C. IRONSIDE, M.B., C.M.

A CASE OF INTRAUTERINE PULMONARY RESPIRATION.¹

I was called to attend Mrs. F. on the morning of April 17th in her fourth confinement. I had attended her in each of her previous pregnancies. She always had a fairly good time and required no instrumental aid. I arrived at the house at 9 A.M. and found she had been ill with pains for the greater part of the night, but had slept for some hours. She was awakened at 4 A.M. by the rupture of the membranes with a severe pain. The pains subsided to a great extent and were occurring every ten minutes when I saw her. The os was dilated about a third. It was a head presentation, but I could feel the apex of the child's left ear by sweeping my finger round the os. I returned to my own house, and came back to the patient at 10.30 A.M. Sitting by the bedside during the interval between the pains, I heard a distinct sobbing, smothered cry. I immediately examined and found the os very little more dilated, not quite half. I asked Mrs. F., "What noise was that?" She replied, "It is the child crying, and it has been crying at intervals since yesterday evening." I called the nurse, and she heard the sound; in fact, it could have been heard from any part of the room. It was just like a child sobbing under the bedclothes. I examined with the stethoscope and found it most distinct just where the mother pointed out to me as the place from whence she felt the sounds proceeding—namely, a point midway between the umbilicus and the iliac spine of the mother's right side. This was where I heard the fetal heart beat. The os was soft and dilatable. I examined the child's heart every few minutes, and I was prepared to use instruments if any failure manifested itself in the fetus. The cry was heard a number of times, and about 12 o'clock, when I gave a drachm of liquid extract of ergot, Mrs. F. remarked that "baby had gone to sleep." No further cry came till 12.30, when a couple of strong pains following each other rapidly expelled the

¹ Read at the Spring Meeting of the North of Ireland Branch, British Medical Association.