almost the babe cries the ignorant mother feeds it—feeds it on a fluid rich in proteids, therefore some babes sometimes fall ill of enteric. The Ghurkas, in India, suffer six times as much as any other caste; they are meat eaters, so are Punjaubis, and strangest fact of all, as regards India, enteric fever only appears in jails which contain meat-eating prisoners in a meat-eating community in Madras Presidency.

The writer recognises other factors, therefore, fully; and it depends on our views of how best to diminish enteric whether we inoculate to increase the individual's resistance; disinfect to attempt the destruction of the specific germs; isolate, segregate, and quarantine, &c., to prevent its effective access; or by sanitation, &c. minimise its growth; or resort to scientific reform in our dietary, especially in the young who are suddenly exposed to an alien environment in India and elsewhere, and so preserve the health of our troops from the ravages of this pest of Western civilization. The moral was contained in Mr. J. M. Barrie's "Little Mary," ably expounded by the little lady who said, when reproached for concealing her Irish birth: "It's sinful to boast."

REPORT ON AN OUTBREAK OF MULTIPLE PERIPHERAL NEURITIS.

By Lieutenant-Colonel H. S. McGill.

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DURING September and October, 1907, a number of cases presenting symptoms of peripheral neuritis were admitted from a British Infantry Regiment stationed at Poona. The regiment was a fine body of men who had arrived in the station early in the year. Previous to these cases, ten with somewhat similar symptoms had been treated between March and August. The disease occurred in an epidemic form in September, causing 21 admissions—17 in the first two weeks—continued during October, when there were 20 cases, rapidly declining in November and December to 7 and 3 cases respectively, and ceasing entirely at the end of the year. Altogether there were 61 admissions which came from every company, but three furnished one-half of the cases. The disease was not limited to any particular age: 15 occurring in men of 20 to 25 years; 29 in those between 25 to 30 years; 13 amongst men of 30 to 35; and 4 in others over that age. None of them were married, and there were no cases amongst the women.

Symptoms.—Many stated that they first noticed the symptoms, swelling and pain in the legs, with shortness of breath, on the morning of reporting sick; others that they had suffered from them for a few days, with inability to walk any distance in comfort; whilst some admitted they had noticed the symptoms for several weeks, but did not think them serious enough to report sick.
The cases can be divided into four groups, according to the severity of the symptoms: First group, 12 cases, with only the following symptoms: tachycardia, slight dyspnœa, shin œdema and weakness of the legs. Second group, 23 cases having similar symptoms, with also tenderness of the calves. In addition 3 had slight numbness of fingers and legs, 6 slight foot drop, 10 soft mitral or pulmonary bruits, 2 Romberg’s symptom, several disordered gait, 16 loss of knee-jerk, and some others profuse sweating of hands and feet. Third group, 8 cases exhibited the following symptoms: tachycardia, dyspnœa, tremor of tongue and fingers, profuse sweating of hands and feet, weakness of legs, swelling and pain in calves, suffused face, restlessness, insomnia, occasional gastric irritation, loss of knee-jerk and shuffling gait. One had Romberg’s symptom, and another nystagmus or lateral movement of the head. Fourth group: 18 cases formed this group, all showing the following symptoms: tachycardia, dyspnœa, shin œdema, swelling and pain in the calves and weakness of the legs. In addition, 15 had loss of knee-jerks, 10 soft mitral or pulmonary systolic murmurs, 6 marked pulsation of the cervical vessels, 6 slight foot drop, 7 numbness of calves, 1 cramp in the legs, 3 Romberg’s symptom, and 1 anasarca of the extremities, abdomen and face. The gait was disordered in all, shuffling, waddling, unsteady, and in a few slightly high stepping. In none of the cases examined was the urine found to contain albumin. Out of the total number, 1 died and 2 were invalided; the remainder, 6 of whom suffered from relapses, recovered and returned to duty. The fatal case occurred during September in a man who had been nine days in hospital with tachycardia, slight dyspnœa, shin œdema, pain in the calves and weakness of legs, and who suddenly developed all the symptoms of acute cardiac insufficiency, i.e., intense orthopœa, great restlessness, profuse sweating, marked pulsation of cervical vessels, tumultuous action of the heart, and weak, very rapid, uncountable pulse—and died two hours later. The following conditions were found at the autopsy:—

Pericardium full of clear fluid; heart, weight 20 ounces, left ventricle hypertrophied, right dilated, valves healthy, but mitral and tricuspid orifices dilated. Lungs much congested and œdematous, especially at the bases. Liver weight 52 ounces, was much congested and fatty. Spleen weighed 7 ounces. Kidneys each weighed 5½ ounces, both were apparently normal. The stomach and duodenum looked healthy, showing no congestion or ulceration.

It is quite evident that we are here dealing with a series of cases exhibiting in varying degrees of severity the symptoms of multiple peripheral neuritis caused by some toxin, with a peculiar affinity for attacking the peripheral and vagus nerves. The toxins most likely to be implicated in the production of this pathological condition are those connected with: (a) chronic arsenical poisoning; (b) chronic alcoholic poisoning, and (c) eudemic neuritis or beri-beri. (a) Arsenic: this can...
be rejected as a possible cause, for none of the cases presented the characteristic ocular, gastric or cutaneous symptoms seen in chronic poisoning by that metal, nor was there drunk by the men of the regiment any sample of the beer, which would be the most likely vehicle for the introduction of arsenic into the system, found to contain, after most careful and independent analysis, more than the legal permissible minimum of $\frac{1}{60}$ grain of arsenic to the gallon. 

(b) Alcohol: the following facts show that alcohol taken in excess was an important factor in the causation of these cases:

(1) The History.—With few exceptions all the men admitted drank beer immoderately. None of them were teetotalers, and though some stated they only drank 2 to 3 pints a day, the greater number, certainly 50, confessed to having regularly drunk 6 to 8 pints daily, not a few of them admitting to as much as 2 gallons, whilst several drank 3 gallons whenever they could get it. The larger amounts seem enormous, but the men always adhered to their original statements. Now, what does the consumption of this amount of beer represent quo absolute alcohol? Careful analysis conducted by the Divisional Sanitary Officer and the Director Central Excise Laboratory, Kasauli, showed that all the samples of beer drunk by this regiment contained 5.6 to 5.7 vol. per cent. of absolute alcohol, so that a man who drank from 6 pints to 2 gallons of it consumed 6.7 to 17.0 ounces of 100 per cent. alcohol, whilst the 3-gallon men took nearly 26.5 ounces. In the opinion of the director of the Central Excise Laboratory "the consumption of such large amounts of alcohol daily would suffice to account for all the symptoms described."

(2) The Symptoms.—Anyone who has seen a case of multiple alcoholic neuritis, which is not very uncommon in the army, would at once recognise the similarity of many of its symptoms with those I have described in this series of cases. Bain, in his "Text-book of Medical Practice," gives a clinical picture of a case of a multiple alcoholic neuritis, in which he describes the occurrence of the following characteristic symptoms:

Formication or numbness in fingers and feet, pain in legs increased on movement, tenderness of the calves on pressure; foot drop, tremor of tongue and fingers, high stepping or waddling gait, tachycardia, dyspnœa and oedema along the shins, loss of knee-jerk, profuse sweating of hands and feet, anaesthesia of lower extremities rarely spreading to the trunk. Further, when speaking of the prognosis of these cases he remarks: "Alcoholic cases usually recover completely, although it may take months or years for them to do so. There is, however, a constant danger of sudden death in severe cases, an event that has been attributed to neuritis of the vagus leading to cardiac failure.” In support of this statement, I have notes of two cases from different corps that were carried into the station hospital with all the symptoms of acute cardiac insufficiency. Both died a few hours after admission. In each the pericardium contained
some fluid. The heart was enlarged, weight 16 ounces, right ventricle much dilated, left hypertrophied, valves healthy but mitral and tricuspid orifices dilated, tissue fatty and very flabby. Lungs much congested and edematous, liver fatty and enlarged, weight 72 and 76 ounces respectively. Spleen weighed 8 ounces, very dark coloured and soft. Kidneys weighed 7½ and 7 ounces, very fatty and congested. Stomach and duodenum congested but not ulcerated. Both these men were habitual heavy drinkers. One was of most active habits, the other never took any exercise. Neither had ever reported sick, and no evidence could be obtained of their ever having complained of feeling unwell before being carried to hospital. Osler also draws attention to a group of cases of dilatation and hypertrophy of the heart occurring in men who do very hard work and at the same time drink alcohol—chiefly beer—to excess. They report sick complaining of palpitation, shortness of breath, slight anasarca of lower extremities, pains in the legs, and later may develop symptoms of cardiac insufficiency. I may mention that almost all the men who suffered from this neuritis were of most active habits, regularly taking part in hockey, football, cross country races and gymnastics. I think it will be allowed that the evidence brought forward of the intemperate habits of a large majority of the sufferers gives colour to the suggestion that alcohol taken in excess had a good deal to answer for in the causation of this outbreak of multiple peripheral neuritis. There is, however, one feature of the outbreak that cannot be reconciled with the theory that it was entirely due to alcohol. I refer to the epidemic character it assumed during September and October. Closely as the cases described resemble alcoholic multiple neuritis, I have never known or heard of that disease appearing in an epidemic form. So it is evident that some other toxin was primarily or secondarily responsible for this outbreak. The most likely one in the East is the unknown toxin responsible for the occurrence of endemic neuritis or beri-beri. (c) Beri-beri. There is no doubt that the symptoms described also resemble those attributed to the toxin or germ of beri-beri, but if the outbreak were that disease it was in most cases of the larval type. Some had more severe symptoms; one, the fatal case, was pernicious, whilst others in group “No. 4,” with pulsation of cervical vessels, tachycardia, bruits, dyspnoea and general discomfort, were allied to that type. None belonged to the “dry or paralytic” form with marked paresis and muscular wasting, nor excepting one did any show the symptoms of the “wet or dropsical” type with general anasarca. According to Daniels this disease at the present day not infrequently occurs in the larval form, though there is always the danger that the case may suddenly develop acute cardiac symptoms. Assuming we are dealing with beri-beri, where did its germ or toxin come from and how did it enter the body? The modern views of the causation of beri-beri are the following: (1) The food theory: in this, certain kinds of food are believed to produce the disease owing to their being deficient in nitrogen or fat, which causes nitrogen or fat
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starvation; (b) or to the presence of a toxin or germ elaborated in decayed or imperfectly cured fish and rice. The want of nitrogen or fat is not now credited with causing this disease, though for a long time the Japanese and others believed in nitrogen starvation. Fish decayed or badly cured need not be considered. Certain kinds of rice are believed to contain injurious qualities which when taken into the alimentary canal produce neuritis. Braddon and Fletcher, who have both had a large experience of the disease in the Malay States, are convinced and bring considerable evidence in support of their opinion that eating rice regularly and in large quantities which has only been dried before husking, stale uncured white rice, will invariably sooner or later cause beri-beri in its consumers. This does not occur if the rice after drying is boiled or steamed, then husked and dried, i.e., cured brown rice. According to these authorities the husk of the dried white stale rice contains a fungus which penetrates the grain when it is decorticated and gives rise to the disease when the rice is eaten.

(2) The Germ Theory.—There are two views as to the path by which the infecting germ may gain admission to the body. Manson believes it to be a saprophyte that lives in the soil or surroundings of beri-beri localities—earth or place infection—and in the presence of heat, moisture, and a special soil, elaborates a toxin which being inhaled or swallowed by man causes neuritis. Wright also looks on it as a place infection, but attributes it to pollution by the faces of beri-beri cases; the germ residing in the earth or surroundings is taken in with soiled food, and after a short incubation in the alimentary canal causes a specific duodenitis followed by neuritis.

(3) The Parasitic Theory.—Hewlett suggests that beri-beri may be caused by a protozoon which he reports having isolated from the urine of a beri-beri patient. Manson and Daniels also offer the opinion that pediculi may act as carriers of infection, which presupposes an infecting protozoon. Whatever influence these accepted causes may have in certain outbreaks of beri-beri it is very certain that none of them were concerned in the one under consideration: (1) None of the cases ate much rice; (2) beri-beri had never occurred before in the lines, and is very uncommon amongst the natives of the place; (3) all excreta were disinfected and there was no chance of a specific pollution of the soil. Very little is really known about the cause of beri-beri, and though many instances have been recorded where probably it was due to eating diseased rice or to inhaling or swallowing a toxin given out from infected soil, I do not think that these causes can account for its occurrence in every instance. I am inclined to believe Sir Patrick Manson when he states: "It is quite possible that several kinds of peripheral neuritis, each with its special cause, may have been included under the term beri-beri, which after all may only be like the word dysentery, a name for a group of symptoms produced by several diseases of the same tissues or organs and not of one
special disease of these tissues or organs.” The symptoms I have described were, I believe, those of a disease belonging to the beri-beri group, not due to any of the accepted causes but to some toxin introduced not improbably in the beer, or produced by some fermentation in the alimentary tract caused by the beer. With very few exceptions all the men in the regiment drank beer, but it was evident that only those suffered who drank it to excess, a considerable quantity apparently having to be regularly consumed before neuritis appeared. Though some of the cases were true alcoholic multiple neuritis, which Daniels states is the disease most likely to be mistaken for beri-beri when the cause is present, as it certainly was in these cases, its occurrence as an epidemic at one period of the outbreak upsets the opinion that it was purely an alcoholic neuritis. It can only then be attributed to a beri-beri infection, not, however, originating from the commonly accepted causes but undoubtedly predisposed to, if not excited by, the excessive consumption of beer.

Treatment.—All the cases were kept apart as much as possible, and those whose symptoms required it were kept in bed—men with marked cardiac symptoms being most carefully watched. Soda sulphate 1 drachm was given daily, whilst the more severe cases were given a mixture of digitalis and strychnine. The diet was light and digestible. All the cases recovered and returned to duty after one to three months’ treatment and rest, excepting one who died and two who were invalided. The fatal case was treated with digitalin, strychnine, amyl nitrite, trinitrin, and venesection. When the troops went into camp the barrack rooms were cleaned and whitewashed, but these precautions were really not necessary as there was no possibility of any place infection.

NOTES FROM KORDOFAN ON TWO CASES OF FEVER ASSOCIATED WITH SPIROCHÈTES IN THE BLOOD.

By Captain A. B. Cummins.
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On November 5th, 1909, a large draft of men arrived in El Obeid, Kordofan Province, Anglo-Egyptian Sudan, from furlough in Egypt. These men belonged to the 3rd Battalion, which is at present stationed at El Obeid.

On November 11th two of these men reported sick with fever. The blood of both men was on the same day submitted to the routine examination for malarial parasites, and in both cases with negative results. On this occasion no spirochètes were noted in the blood of either of the men.