BERI-BERI IN THE MESOPOTAMIAN FORCE

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THE occurrence of a number of cases clinically resembling beri-beri in the Mesopotamian Force from 1915 to 1919 gave rise at the time to much investigation and discussion.

Entire agreement was not always reached as to whether the diagnosis of beri-beri was correct. It was admitted by all observers that there was no clinical difference between the text-book description of the disease and the cases seen in the field ; but because in some instances no defect could be found in the diet some observers considered the disease could not be beri-beri. Others classed the disease as beri-beri, and considered it likely that some other cause than food deficiency was here operative. It will be understood, therefore, why an unknown number of cases amongst British troops was in the earlier days returned as 'peripheral neuritis', while exactly similar cases were later classed as 'beri-beri'. The total number of beri-beri cases is therefore somewhat larger than that here reported. Now that the campaign has come to an end there is opportunity to review the facts and to consider what the correct view of these cases should be. It may be stated here that the frequently simultaneous occurrence of signs of nerve degeneration in the lower limbs and of cardiac signs, which we may call 'cardio-neurotoxic syndrome', is the chief characteristic of the disease. The terms 'cardio-neurotoxic syndrome' and 'beri-beri' will be used indiscriminately; that is to say, the latter word will not necessarily imply that a vitamine deficiency was the cause of the condition.

The cases returned as beri-beri during this campaign may be classed into three divisions: (A) British cases, occurring amongst the land forces. Of these there were 84 in 1917 and 51 in 1918. But, as already explained, some other cases were returned as 'peripheral neuritis'. (B) Chinese cases, amongst Chinese labourers on land. Of these there were 647 in 1917, 198 in 1918, and 76 up to the end of September, 1919. (C) Ship cases, occurring amongst Indian crews. There was a small epidemic in 1916 and another in 1918. There were practically no Indian cases on land.

It is the opinion of the writer that though the clinical picture is approximately the same in each of the three classes of cases, and though the pathological lesion is also in all probability approximately the same, yet that the etiology of each class is different. In the etiology of this cardio-neurotoxic syndrome there

appear to be two main predisposing causes, food deficiency and infection. The exciting cause may be fatigue, secondary infection, or other depressing factor. If this be correct, the syndrome may arise under entirely different circumstances, and the measures necessary for its prevention or treatment should accordingly be varied to suit the particular instance. Concerning these classes, the writer's views are:

(i) That the British cases were not due to a food deficiency; but that they were of infective origin.

(ii) That the Chinese cases have previously suffered from beri-beri in their own country, the result either of diet deficiency or infection; or else they have lived for a prolonged period in such a state of vitamine deficiency that illness, or fatigue, or other depressing cause may allow the ill effects of this long-standing deficiency to evince themselves.

In other words, that they were suffering from 'latent beri-beri' when they landed in Mesopotamia.

(iii) That the ship cases were examples of the orthodox primary beri-beri, arising from recent vitamine deficiency. This primary beri-beri can be readily prevented by attention to the diet; but once the syndrome has developed cure is sometimes tardy.

This conception of the human cases of beri-beri is still more comprehensible when compared with the results of animal experiments, such as McCarrison's observations on birds. In 1914 (1) McCarrison, by means of infection with *Bacillus suispestifer*, induced polyneuritis gallinarum in birds fed on abundance of mixed grains.

Birds that survived the bacillary infection for six days developed polyneuritis: these correspond to the British cases (Class A) in which the presence of an infection is suggested. McCarrison's male pigeons (2) that took on an average sixty-one days of totally avitaminic diet to acquire beri-beri correspond to the ship cases (Class C), where, as will be shown, five months of a partially avitaminic diet were sufficient to induce the disease in men.

McCarrison also found that with the combined factors of deficient diet and infection acting together the cardio-neurotoxic syndrome developed in birds with greater readiness than when either factor acted alone. It is suggested that the Chinese of Class B are analogous to these pigeons.

A. *British Cases of Beri-beri.*

It has often been proved that consumption for several weeks of a diet deficient in vitamins will cause in birds degeneration of certain peripheral nerves and, as McCarrison (2) has recently shown, widespread endocrine changes. The work of Eijkmann (3) and others is sufficient evidence that a diet of polished rice may cause beri-beri in men. Reason must be given therefore for holding the opinion that these cases amongst British soldiers, which appeared clinically so like beri-beri, were not due to food deficiency but to some infective cause. Opportunity

was afforded for studying the disease in epidemic form in a certain British regiment in Mesopotamia on two occasions, whilst the records of another British unit show that the disease also was endemic amongst them.

The regiment first referred to was encamped at Shaiba, about eight miles from Busra, in the latter half of 1916. By the end of the year sixty-six cases of so-called beri-beri had occurred, most of them in December. This epidemic was investigated by the Medical Advisory Committee (4) and by Colonels Wilcox and Melville, Consulting Physicians to the Force, and by Major Stevenson of the Base Central Laboratory. Reference will be made later to their reports.

In the beginning of the year 1917 the disease died out, and in April the regiment moved about 100 miles to Nasiriyeh. In December 1917 there was a similar epidemic in the regiment, then at Nasiriyeh: on this occasion there were only fourteen cases and the epidemic was investigated by Colonel Melville and Major Stevenson.

To return to the first or Shaiba epidemic, the Medical Advisory Committee in their report remark that beri-beri had occurred about the same time the previous year in other units. This, together with its recurrence in December 1917, suggests that this syndrome is more prevalent in the autumn.

A clinical account of a typical case of this disease will now be given, a sporadic case being selected because the subject was himself a medical officer and able to give an accurate and detailed account of his symptoms. The Shaiba cases will be referred to later.

Captain 'C.', a medical officer, aged 26, stationed in a camp on the line between Baghdad and Persia, suffered from diarrhoea in the first week of November 1917. For two or three days there was a rise of temperature to about 100° F. On recovery from the diarrhoea the patient noticed weakness of his legs, and for this complaint was admitted to hospital on November 10. At this time were noted tenderness of the calves and exaggeration of the knee-jerks, together with some palpitation on walking and slight swelling of the feet. With this clinical history the patient was evacuated to Baghdad.

The patient stated that his diet in camp, where he was medical officer to an Indian regiment, was particularly good. It appears to have been rich in anti-beri-beri vitamins, for he took porridge daily, dhal occasionally, and eggs often. He ate four fresh eggs daily, exclusive of those in puddings, &c. He ate rice but rarely. No one else in the regiment or in the camp seems to have been similarly attacked.

On arrival in Baghdad the knee-jerks were absent. There was some cardiac dilatation, the apex beat being in the nipple line. There was no tachycardia whilst the patient lay in bed. After five days in Baghdad the patient was evacuated down-stream and reached Amara a week later. On his arrival there the knee-jerks had returned.

On arrival in Busra in the first week of December patches of skin anaesthetic to light touch and to pin-prick were discovered on the tibial aspect of each leg extending nearly half-way from ankle to knee. The apex beat was now noted as

being outside the nipple line. The tenderness of the calves was still present. The patient was evacuated from Busra to India, and after convalescence and a few weeks' leave was fit to return to duty in Mesopotamia. During eighteen months' further duty in Mesopotamia he has shown no recurrence of the disease.

The Shaiba epidemic cases were similar to the above.

Reference need only be made to one in particular because of the patient's short residence in the country.

Pte. P., total residence in Mesopotamia five weeks, all at Shaiba. Onset of symptoms five days before admission, i. e. after one month's residence. This case was severe, the heart being much dilated, with systolic murmurs at the apex and tricuspid areas. There were also muscular weakness and hyperaesthesia of the legs. The knee-jerks were entirely absent.

I am indebted to Captain T. H. Oliver, R.A.M.C., for the report on Pte. P.'s case. Captain Oliver, in a report on fifty-four of the Shaiba cases, notes the following points:

'Dilatation of the heart, usually left-sided, was present in 80 per cent. of cases. Systolic murmurs were common at the apex, pulmonary and tricuspid areas. Tachycardia always resulted from slight exertion. There was some oedema over shins and ankles in about one-third of the cases. Frequently there was a slight evening temperature, especially at the beginning of the patients' stay in hospital. This is apart from the fact that a previous history of malaria was noted in nearly half the cases, a benign tertian parasite found in five cases and a malignant tertian in one case. The knee-jerks were usually normal or increased: they were absent in about one-fourth of the cases. Weakness of the leg muscles was a common early symptom, but sometimes only occurred later. Flabbiness of the calves was common. Anaesthesia was not common in this epidemic: hyperaesthesia was occasionally found. In no case was there any ocular or palatal palsy.'

In most of the sporadic cases which the writer has seen some anaesthesia has been present, occasionally extending up to the mid-thigh and at times of a stocking type. Usually, however, it is in patches and liable to occur about the ankles. Colonels Wilcox and Melville in their report on the Shaiba epidemic say: 'We are satisfied that in the cases examined by us the symptoms were such that the diagnosis of beri-beri was correct.'

The medical officer of the regiment wrote: 'Diarrhoea has been very common in the regiment, and I should say that over 50 per cent. of people coming to Shaiba get diarrhoea, which is presumably due to the water.' But he also remarked that no initial diarrhoea had been noted in the beri-beri cases and some had had no diarrhoea at any period. This is of importance because peripheral neuritis occasionally occurs as a complication of bacillary dysentery or of other enteritic conditions. Regarding the diet of this infected regiment at Shaiba the Medical Advisory Committee wrote as follows: 'Before the attack, troops had a full mixed diet, the only important deficiency being in fresh vegetables, this deficiency of course being equally common among troops at the front and in

many parts of Mesopotamia from which so far little or no beri-beri has been reported.' In December 1916, after 36 cases had occurred, they reported, 'A good and varied diet is said to be given, which includes fresh vegetables. Marmite is also being issued.' Nevertheless, another 20 cases occurred before the end of the year.

The Consulting Physicians, on the other hand, considered that there was evidence of deficiency of anti-beri-beri vitamins in the food as consumed, not so much from deficiency in the rations as from the nature of the Shaiba water, which necessitated prolonged boiling of food articles and therefore caused destruction of the vitamins. They remarked also on the absence of oatmeal and of dhal in the dietary.

There must, however, have been numbers of other troops who had neither oatmeal nor dhal, since the whole British force were on the same rations: yet no other unit developed an epidemic such as this. Moreover the fact that next year the disease recurred in the same British unit in another place and under a satisfactory diet and water-supply also would suggest that the diet at Shaiba was not to blame.

There had previous to the arrival of this regiment been two Indian regiments at Shaiba. They occupied part of the same camp the British did and used the same water-supply: none of these Indians developed beri-beri. It has already been mentioned that there have been very few Indian cases on land throughout the campaign.

The services of the chemical analyst were called in to prove the absence of metallic poisons in the food and drinking water and the absence of arsenic in the hair of those affected.

Major Stevenson of the Central Laboratory in his investigation noted that the same regiment had had one or two similar cases at another camp during the previous spring: also that 7 of the men affected had been in Mesopotamia only between 42 and 48 days. These 7 cases had arrived in Mesopotamia from a dépôt in India, where they had been for over a year. The voyage from Bombay to Busra takes less than a week, so that the length of the period on board ship cannot be a factor in causing the disease. Some of the remaining cases had come direct from the United Kingdom to Mesopotamia and had been in the country several months. One of the 7 recent arrivals was the case Pte. P., mentioned above.

This is an important point because three months of a deficient diet has been mentioned (5) as the minimum period before the beri-beri syndrome will manifest itself: note the typical cases described below under C where five months of the deficient ship rations were necessary.

Major Stevenson also drew up a plan of the sixty tents occupied by the regiment and marked in the cases per tent. The essentials of his plan are reproduced here as Fig. 1. It will be seen that the machine-gun section had more than its share of cases: moreover some of the earliest cases came from this section.

Major Stevenson also calculated by the Laws of Probability what the tent

distribution of these cases would be, if among all the occupants of these sixty tents there were an equal chance of the disease occurring; as, for instance, would be the case were the whole regiment equally exposed to some virus. According to this calculation :

23	tents	would have	no cases
22	"	"	" 1 case
10.5	"	"	" 2 cases
3.3	"	"	" 3 cases
0.5	"	"	" 4 cases.

FIG. 1. Diagram of the sixty tents of the regiment at Shaiba in December 1916. Each black dot represents a case diagnosed as beri-beri at the time of the epidemic.

The actual figures were :

23	tents	had	no cases
24	"	"	1 case
9	"	"	2 cases
3	"	"	3 cases
1	"	"	4 cases.

These actual and calculated figures correspond so closely as to be strongly suggestive of an equal distribution. But that is only if we consider the whole sixty tents as one unit. It is possible, on the other hand, to regard the more heavily affected machine-gun section as a separate unit. In that case the machine-gun section might be considered as the starting-point of an infection that spread equally among the rest of the regiment. Major Stevenson also made very thorough inquiries into the diet of the regiment. There seem to have been no defects except that the amount of total solids in the Shaiba water was so high as

to necessitate prolonged cooking of stews. An ordinary stew seems to have taken five hours to be palatable, whilst dhal never became soft enough to eat. The oatmeal in bags was weevily and therefore unpopular, so latterly tinned oatmeal was supplied, and this was satisfactory. Practically no rice seems to have been eaten.

Major Stevenson did blood cultures on eleven cases of this type under aerobic and anaerobic conditions: they were negative. The dietary deficiencies mentioned were with some likelihood considered at first to be responsible for the disease; but Major Stevenson subsequently considered it probable that an infective agent was at work. The deficiency theory certainly receives a severe blow when we come to the recurrence of the disease in the same regiment a year later at Nasiriyeh.

Colonel Melville and Major Stevenson also investigated this second epidemic, and I have had the advantage of discussing it with Captain Ford, the officer then in medical charge of the regiment. There were fourteen cases this time, all of the same type as in the preceding year. Two of the cases had been in the country only nine and ten weeks respectively, and the latter of these two came direct from England, the former from a dépôt in India.

Neither of the investigating officers was able to find any deficiency in the dietary: in fact owing to the occurrence of the disease in the previous year special care was taken to see that the men's food was satisfactory. The oatmeal was now clean and the water good, and Captain Ford was satisfied there was nothing lacking. Certainly the battalion had whatever the rest of the Force was getting and in some cases more. There was very little beer available, and three of those affected were total abstainers. Eggs, notably rich in vitamins, were occasionally eaten: one of the patients ate four eggs a week. In addition, from November 1, the whole battalion had been on marmite, $\frac{1}{4}$ oz. per man twice a week.

Major Stevenson investigated more thoroughly eight of the cases which were selected by Colonel Melville as the most typical. Two of these eight cases were recrudescences from the previous year's attack: it is remarked below concerning the Chinese cases how prone those who have once suffered from beri-beri are to relapse. Major Stevenson also made a spot map of the distribution of these eight cases amongst the regimental huts: the total number of huts was about fifty. He found that:

2 cases occurred in 1 hut in 3 instances
and 1 case „ „ 1 „ 2 „

Each of the two recurrent cases had a fresh case in the same hut with him. He concluded that this was not a 'chance' distribution. It would seem, however, that the numbers are too small here to make a deduction of value.

Amongst the total fourteen cases there was a history of recent diarrhoea in two, and of dysentery in one case. The incidence of dysentery had been high during the preceding two months in all units in Nasiriyeh, and a little higher in

the beri-beri infected regiment than amongst other British units there. Stool cultures by Major Stevenson of his eight selected cases and of cases of dysentery in the battalion gave no information. Neither did the plating out of the next 100 consecutive cases of enteritis in the regiment. The differential leucocyte counts of seven affected cases were normal; blood cultures in the seven cases were sterile except for a few contaminations.

Three of the cases gave a recent history of tonsillitis. Swabs from the tonsils of eight tonsillitis cases (not with neuritis) in the battalion were also plated out: no diphtheroid organisms were found. This finding is of importance because about a year later there were three cases of post-diphtheritic palsy in this regiment after it had moved to a distant camp. The suggestion was then made that the epidemic of the two previous years had been diphtheritic, but this idea cannot be held because the paralyses were of a different type: there was no ocular or palatal palsy in the 1916 or 1917 epidemic. Also it would be incredible that sixty-six cases of post-diphtheritic palsy could occur in one year and fourteen in the next, all of the same type, and without any diphtheritic throats in the battalion. The investigation of this second or Nasiriyeh epidemic confirmed Major Stevenson in his opinion that the 'disease was not due to a food deficiency, but was a neuritis sequent to a toxic or infective factor'.

The recurrence at Nasiriyeh seems to have shaken Colonel Melville's original opinion regarding the Shaiba epidemic, since he reported: 'The presence of the disease again this year in a body of men, specially dieted and looked after—at the same time of the year and in another station—is rather striking and suggests some cause other than purely dietary.'

Another unit had also an undue number of cases with the same cardio-neurotoxic syndrome as described above. This unit was not a homogeneous one, being composed of men from numerous outside units, collected together at the Base and employed on clerical duties only. In this unit there has been no epidemic; but at various times during the two years ending with November 1918, fifteen cases have occurred out of a total of about 700 men. No case has occurred since November 1918. These men are sedentarily employed, and are on the same rations as the rest of the British troops at the Base. The occurrence is suggested of some source of infection in the unit during this period. Sporadic cases have also occurred in the Force.

Exclusive of the epidemic already described in the regiment, there were reported eighteen cases during 1917, and thirty-seven cases during 1918. At least four of these cases were officers, in only one of whom was there any suspicion of his having been on a shortage of anti-beri-beri vitamins at any time previously. One sees not rarely in Indian civil practice cases of leg polyneuritis that one can hardly explain by diet deficiency or other known cause. Sir John Rose Bradford, in a recent paper (6) on polyneuritis, writes of seeing occasionally similar cases on the Western front, which he regarded as milder manifestations of a specific infective febrile polyneuritis. The cases the writer has seen have also not uncommonly been associated with slight febrile disturbance; but he has only rarely

seen the more severe polyneuritis to which Sir John Rose Bradford refers as the standard type. The cardiac disturbance was not a frequent feature of his cases; so the infective agent, if any, in the Mesopotamian cases may not be the specific organism isolated from Sir John Rose Bradford's cases. Epidemics of similar polyneuritis have occurred in British regiments before, notably in the Royal Lancaster regiment at Lebong (7) in 1914, and in the King's Own Scottish Borderers at Aden (8) in 1907.

It seems, therefore, probable that a cardio-neurotoxic syndrome, apparently identical with that known as beri-beri, can occur apart from any deficiency of diet; and the probability is that it is a result of infection by some micro-organism. There is no evidence yet how infection occurs; the intestinal route is suggested by the occasional clinical association with enteritis as well as by the well-known post-mortem finding of duodenitis in both human and avian cases.

B. *Beri-beri amongst the Chinese in Mesopotamia.*

A Chinese Porter Corps from Singapore arrived in Busra in 1917. The corps came in two drafts, the first in January and the second in April. Cases of beri-beri occurred in both drafts on the voyage and some of them were landed in Bombay. Beri-beri became very severe in the beginning of April and remained so for the next three months. There were 615 cases and 29 deaths from beri-beri in this unit, whose total strength was only about 1,000. Of the remainder several showed early signs of the disease. The entire unit was therefore repatriated. The diet of this corps at first was practically the same as that mentioned below as the present diet, with the exception that there was no bread supplied, 12 oz. of meat were given daily instead of 4 oz., and 28 oz. of rice instead of 20 oz. This rice was of the polished Siam variety, and had been brought with the corps from Singapore because it was their usual staple diet. No immediate successful result was obtained from the use of a diet richer in vitamins. The history of this corps does not afford us sufficient data for deductions as to the origin of the disease. The diet may well have been defective; but beri-beri was evidently established in the corps when it sailed from Singapore. In this case the great majority of the cases occurred between the beginning of April and the end of June, which is contrary to the experience given below with the other Chinese labourers. But in the case of the Porter Corps there were exceptional factors. A possible recent deficiency in their diet is not denied, as it is in the case of the dock labourers. The Porter Corps had only just landed and had been on this possibly deficient diet during the voyage and for some weeks after landing. Moreover in the light of what is said below it may be stated here that the rainfall in the winter of 1916-17 was almost negligible and the following spring correspondingly hot and dry.

Other Chinese labourers have, however, been employed and have been under observation for longer periods.

Some thousands of skilled Chinese labourers, for the most part fitters, boiler-makers, riveters, hammermen, and carpenters, have been brought into Mesopotamia since the summer of 1917 for work in the dockyards and railway workshops. Since the spring of 1919 the repatriation of these men has begun and their number is now under one thousand. They have shown and continue to show several cases of beri-beri, 198 in 1918 and 76 cases up to the end of September 1919. Clinically this disease amongst them is indistinguishable from the cardio-neurotoxic syndrome above described as occurring amongst British soldiers.

The common complaint of the Chinaman with beri-beri is of the heaviness of his legs and of their weakness: often also he remarks on the flabbiness of his calves. On admission to hospital he usually has a temperature of about 100° F., and this pyrexia lasts three or four days. There is often tenderness of the calf muscles. The knee-jerks may be absent; but are more commonly exaggerated, and are often unequal. Anaesthesia in patches is often present on the legs; but this is difficult to elicit accurately in the Chinaman. Oedema over the lower parts of the shins is usually present, but soon clears with rest; this appears to have but little if any relation to the cardiac condition. There is sometimes dilatation of the left heart, and very often undue sensibility to slight exertion, a pulse of 80 accelerating to 120 or more on the patient's sitting up in bed. Cardiac bruits are often heard over mitral, tricuspid, and pulmonary areas. Examination with a differential stethoscope (Leyton's model) shows as a rule abnormality only if the examination be made in the first few days after admission. The ratio between the intensity of the sounds then sometimes indicates myocardial derangement, and in severe cases this sign may last for long.

As regards treatment rest in bed rapidly benefits all but the most acute cases: the exhibition of marmite or of other special foods seems without effect. Relapse on resuming work is very common.

A brief account of a typical case is given here.

A. Y., age 26, a boat-builder, six months in Mesopotamia, came to hospital on July 26, complaining of weakness and heaviness of his legs. He was of fair nutrition, and showed absence of knee-jerks, but had no calf-tenderness or apparent anaesthesia. There was slight oedema of the front of the lower legs. There was some dilatation of the left side of the heart, accompanied by a systolic murmur at the apex. The pulse-rate when sitting down was 116; after walking a few steps it was 146 per minute. The gait was high-stepping.

The illness was accompanied by slight pyrexia; the temperature rising to between 99° and 100° for four days, and then, after five days of being subnormal, returning to the normal.

The patient rapidly improved after a few days' rest in bed: he was, however, evacuated from the country.

Experience has shown us that cases of beri-beri, however mild, are so liable to a return of the condition, either on the resumption of hard work or from the

onset of any other disease, that it is advisable to repatriate any Chinese beri-beri case as soon as he is fit to travel. During a malaria epidemic in Busra some of the Chinese who were attacked with malaria reported sick with beri-beri symptoms: a similar combination with influenza was also sometimes seen. Any cause of debility that would produce its own train of symptoms in another subject might apparently, in one of these Chinese, produce beri-beri. An example of malaria with beri-beri is seen in the case of W. M. C., a Shanghai ship-plater, who had been in Busra eight months when he reported sick, complaining of weakness of the legs.

He showed a tertian fever with regular ague attacks, and a spleen reaching three fingers below the rib margin. The benign tertian parasite was found in the blood. He also showed signs of early beri-beri, with exaggeration of knee-jerks, oedema of the shins, and some anaesthesia of the legs. The gait was high-stepping on a broad base. From the size of the spleen he had doubtless been suffering from malaria for some time, and it is probable that the resultant debility had determined the onset of beri-beri.

A few more cases only need be referred to, to show after how brief an interval after arrival in Busra the neurotoxic syndrome may evince itself, much less than the several months of deficient diet considered necessary for the development of beri-beri in a previously healthy subject. Even if we include the period of the voyage from China the time is too short: some cases have actually been taken off the boat with the disease already manifest.

F. N. H., a moulder, reached Busra on October 6, 1918, and felt weakness of the legs seven days later: he was in hospital from October 17, and showed the usual syndrome.

S. Q. L. reached Busra on February 6, and began the disease on March 16, 1½ months after arrival.

Stevedore B. A. L. reached Busra on September 6, and developed beri-beri on October 12, five weeks after arrival.

Other cases show intervals of 26 days, 23 days, 25 days, and 7 days respectively between disembarkation at Busra and development of beri-beri. In the case of blacksmith Y. A. S. the interval is even shorter. He left Shanghai on August 21, arrived in Busra on October 7, and developed beri-beri the next day.

If it be true, as seems the case, that three months' vitaminic deficiency are necessary for the primary development of beri-beri, then these cases, if suffering from a primary attack, must have had the disease developing before they left China. Moreover the dietaries on the ship from Shanghai to Bombay, at the camps in Singapore and Bombay where they stay *en route*, on the ship from Bombay to Busra, and in the Mesopotamian Force after arrival in Busra must also have been deficient. This is a large assumption to make. The probable explanation is that these cases had all had beri-beri previously in China, or had lived for years on a partially avitaminic diet to the extent that any hard work or general debilitating cause would bring on the cardio-neurotoxic syndrome.

A study of the cases that have arisen after several months' residence in Mesopotamia makes a similar deduction probable. There seems to be no deficiency in the diet given to the Chinese in Mesopotamia. When the first cases of beri-beri occurred much thought was given to the matter, and in consultation with the Chinese headmen a diet as rich as possible in anti-beri-beri vitamins was arranged. Money was not spared in framing this diet: salted eggs and China tea were imported, and dried beans and peas were freely given. So generous is the allowance that the food of the Chinese labourer has become more costly than that of the British soldier. This is an important economic point and a fact that deducts greatly from the value of a Chinese force. The amount of each article of diet issued daily to each Chinaman is given here. The rice is of the unpolished variety.

Chinese daily ration for week ending August 9.

Quantity of each article in ounces.

	Sunday.	Monday.	Tuesday.	Wednesday.	Thursday.	Friday.	Saturday.	
Rice	20	20	20	20	20	20	20	
	Frozen	Fresh	Frozen	Fresh	Fish	Fresh	Frozen	
Meat	4	4	4	4	6	4	4	Usually beef: pork rarely available
Wholemeal bread	4	4	4	4	4	4	4	
China tea	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	
Jam	2	2	2	2	2	2	2	
Sugar	2	2	2	2	2	2	2	
Salt	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	
Green vege- tables	4	4	4	4	4	4	4	
Milk	2	2	2	2	2	2	2	
Salted eggs	—	2	—	—	2	—	2	Not by weight
Cooking oil	1	1	1	1	1	1	1	
Vinegar	1	1	1	1	1	1	1	

In some weeks potatoes are given in lieu of green vegetables; also peas or beans: and salt fish or bacon may be given in lieu of salted eggs.

In addition to the above the Chinaman spends his own liberal pay readily on the purchase of extras. He is especially fond of prawns, ducks, fowls, vegetables, pickles, and green peas, fresh or tinned, and supplements his already adequate rations with these. It does not seem therefore that any present or recent vitamin deficiency is the cause of the Chinese beri-beri in Mesopotamia. On a few occasions the number of cases occurring at one time has been of the nature of a mild epidemic and has led to special inquiry. There was a small outbreak at Amara in October and November 1918. The writer investigated this together with the local officers and could find no vitamin deficiency. The incidence of the disease was heaviest amongst the boiler-makers.

The cases that occurred in November appeared to be strangely grouped at one end of the Chinese living hut, vide Fig. 2.

A similar tendency to grouping in certain huts is shown in the cases that were occurring in the dockyard huts at Busra, also in November 1918, vide Fig. 3.

FIG. 2. Diagram of the beds in the Chinese hut at Amara in November 1918. Each bed marked in black represents where a man slept who developed beri-beri. 75 beds altogether: 8 cases. Not drawn to scale.

The diagrams are presented here because they appear of interest; but the writer cannot adequately explain them. The arrangement is not definite enough to warrant the deduction of an infective agent being at work. Moreover there were prepared an equal number of diagrams of other areas that show a 'scatter' or 'chance' distribution. Captain Mitra, I. M. S., in medical charge of the Busra dockyard, prepared the Busra diagrams, and further he made an interesting experiment to see if the disease tended to linger in the buildings themselves. He evacuated the most infected huts (shown in Fig. 3) of Chinese labourers, and then, without taking any sanitary measures with regard to the buildings or their surroundings, he put Indians into them. No case of beri-beri has occurred amongst these Indians in the course of a year.

The disease tends to occur more in the autumn; and this seems the case also with the British troops in Class A. The following statement shows approximately the average strength of Chinese in Mesopotamia, the exact number of beri-beri cases, and the morbidity percentage calculated therefrom in the course of thirteen months for which these figures are available:

	Chinese.	Chinese beri-beri cases.	Morbidity rate per cent.
1918			
September	5000	13	0.26
October	5000	33	0.66
November	5000	42	0.84
December	5000	13	0.26
1919			
January	5500	15	0.27
February	5700	8	0.14
March	6000	12	0.20
April	5000	10	0.20
May	4000	2	0.05
June	1800	4	0.22
July	1430	5	0.34
August	1260	8	0.63
September	1190	12	1.00

FIG. 3. Diagram of some of the huts of the Chinese labourers in the dockyard at Busra in November 1918. Not drawn to scale. Beds marked in black are those whose occupants became affected with beri-beri in the three months preceding. Cases whose bed cannot be located are marked by black dots in the centre of the hut.

Hut A	28 men	11 cases
" B	36 "	13 "
" C	42 "	3 "
" D	42 "	3 "
" E	40 "	6 "
" F	40 "	3 "
" G	36 "	7 "
" H	36 "	6 "

If one may make deductions from a statement of this kind, it would seem that external temperature has no influence on the disease. Though the incidence is at its minimum when the weather begins to grow hot, yet it commences to increase during the hottest month, and increases still more as the weather grows cool, to decrease suddenly in the coldest months. The only weather factor in Mesopotamia that corresponds with this change is the rain. Rain usually ceases in Mesopotamia in April, and after six rainless months the wet season begins in late November. In 1919 occasional falls of rain continued till May, and the rainless period in this case corresponds therefore with that during which there is a gradual increase in the beri-beri incidence.

Too much importance must not, however, be attached to limited observations of this nature.

The outbreaks referred to in October and November 1918 were in Amara and Busra; also in October a small outbreak occurred amongst the Chinese at Shaiba. This is the same Shaiba where the epidemic occurred in the British regiment in 1916, but the actual sites were nearly two miles apart. The writer investigated this Chinese outbreak on November 1. The distribution of cases amongst the dwelling huts was of the 'scatter' type. There were deficiencies in the diet in that the dried peas were heavily infected with a small beetle and almost uneatable, and the flour was white, i. e. not wholemeal; but there appeared to be no gross lack of vitamins. The rice, which forms the principal article of diet, was of the unpolished variety. The following extract from the writer's report at the time will serve here:

'The disease was said to have begun in Shaiba during June last, but it was getting worse latterly and about half the cases occurred during the last month. Besides the 17 cases I saw, since June 9 beri-beri cases had died, 8 had been sent to India, and 7 more were now in No. — I. G. H. at Busra.'

This makes a total of 41 cases in five months amongst a total Chinese population of 280; that is, a case incidence of 14.6 per cent. The 280 Chinese may for practical purposes be divided into three batches; 140 men from Shanghai, who arrived in Mesopotamia on May 3, 1918, 100 men from Chi-li, who arrived in Mesopotamia on June 20, 1918, 40 men from Canton and Singapore, who arrived in Mesopotamia on February 9, 1918.

The incidence of the disease has not been equal in these three batches; thus:

	Total No. of men.	No. of cases.	Percentage of cases to healthy.
Shanghai	140	22	15.7
Chi-li	100	4	4.0
Canton and Singapore	40	15	37.5

The men who have been in Shaiba the longest are thus seen to be the worst affected.

Neither is the occupation incidence equal:

	Total No.	No. of cases.	Percentage of beri-beri cases to healthy.
Boiler-makers	40	12	30.0
Fitters	150	14	9.3
Riveters	30	6	20.0
Hammermen	40	5	12.5
Turners	8	3	Numbers too small
Carpenters	2	1	
Tinsmiths	10	0	

From this it is seen that the boiler-makers have the highest incidence, a fact I also found in investigating a similar epidemic amongst the Chinese in Amara. The boiler-makers probably have the hardest work of any of the above. It may be added that after reduction of the working time from nine to seven hours daily, accompanied by rectification of the dietary deficiencies mentioned, there were no more cases of beri-beri at Shaiba.

The experience of the Mesopotamian Force with Chinese labour is not an isolated one in the war. Three French observers give an account (9) of 228 cases of beri-beri amongst the Indo-Chinese at Marseilles. They say that most of the cases developed before disembarkation or within a few days after. Another French medical officer in Algeria writes (10) of 251 cases of beri-beri amongst Annamites imported during the war. He attributes the outbreak to overcrowding on the transport, and could find no evidence of food insufficiency.

To sum up, the exciting cause of an attack of beri-beri in a Chinaman in Mesopotamia appears to have been fatigue, or the onset of any other illness or general debilitating factor. But there is a predisposing cause of long standing, and affecting much of the young manhood of Eastern Asia to an extent that detracts from its economic and military value. This cause appears to be latent beri-beri previously acquired in the man's own country. The ultimate cause of this latent beri-beri may be a vitaminic deficiency of long standing: it may be an infection like that which is thought to occur in the British cases.

C. *Ship Beri-beri in Mesopotamia.*

The ship beri-beri that has occurred in the Mesopotamian campaign, though clinically resembling the two forms previously described, has been apparently of a different origin and certainly much easier to deal with. We have here the classical disease due to a food deficiency, taking five months of this deficiency to evince itself and cured by remedying the defects in the diet. This may be regarded as 'primary beri-beri' in contrast to the latent or secondary form from which the Chinese suffered. There were two small outbreaks of this primary beri-beri amongst Indian crews on boats at the bar where the Shat-el-Arab flows into the Persian Gulf. The first of these was in December 1916 on the ship C. and was investigated by Lt.-Col. Melville, I. M. S., then Consulting Physician at the Base. There were four cases of beri-beri amongst an Indian crew of forty-two. Most of the crew had been on the ship for three years; some

had been there seven months, none for a less period. The ship C. had been on the bar for just over five months, and only since that period had there been difficulty with the rations. Fresh food, except fresh meat, was not common, since a month's rations were put on board at a time. The staple food was rice, and this was of the polished white variety. Atta (wholemeal flour) was supplied, but only rarely eaten. The dhal (pulse) ration, which might be expected to replace vitamins deficient elsewhere, was somewhat short, and the variety supplied was so difficult to soften in water that it was often discarded. Colonel Melville considered there was evidence of a vitamin shortage and that the beri-beri was due thereto. The diet was corrected and no further cases were reported. The second outbreak occurred in October 1918, on the ship S. M. also at the bar, and was investigated by the writer. As in the ship C. the clinical features were more cardiac than neurotoxic; dilatation of the heart and tachycardia with slight oedema of the shins were always present. In some cases the knee-jerks were exaggerated; in only one, who died, were they absent.

The crew of eighty-one men had been on the ship S. M. for six months, and there were altogether seventeen cases of beri-beri. Amongst the eight cases the writer saw personally, in four the syndrome had taken five months and in four others six months of service on the ship to appear.

The incidence of the disease amongst the crew was as follows:

	Total No. of men.	No. of beri-beri cases.	Morbidity percentage.	Deaths.
Europeans	21	Nil	—	—
<i>Indians.</i>				
Hindus	28	14	50.0	5
Mohammedans	20	3	15.0	—
Goanese	12	Nil	—	—
Total	81	17	21.0	5

The Goanese ate European rations: no one on European rations got beri-beri. The lower morbidity amongst the Mohammedans is probably because ten days previous to the medical inquiry the captain of the ship read that the disease was due to food deficiency, and then tried to put the Indian crew on European rations. The Hindus, however, refused to take them and kept to their former food. The Mohammedans had no scruples. Previously to that time both Hindus and Mohammedans had been on the same rations. These rations on examination, though abundant in quantity, were evidently deficient in anti-beri-beri vitamins. The rice was of the polished or Rangoon variety. The flour was white (i. e. not wholemeal); it was also weevily. The dhal, which might have supplied the defects of the rice, was very dirty, containing many moth eggs and maggots. These defects were remedied in various ways, and the captain of the ship wrote some months later that he had had no further cases of beri-beri.

These two outbreaks of ship beri-beri appear therefore to be due to food deficiency.

Clinically the resemblance between the above three classes is great. Any one case might have occurred in any of the three classes. When considered *in toto*, however, Class A may be said to have the nervous more in evidence than the cardiac symptoms; Class C has the cardiac symptoms more prominent; while Class B is intermediate between these two and usually has both nervous and cardiac symptoms. Oedema over the lower shins is present in only a proportion of the cases of Class A; it seems always to be present in Class C, and usual in Class B. Having regard to McCarrison's discovery of the extensive endocrine changes in birds fed on decorticated rice, the liability of the Chinese cases to recurrence as a result of an outside depressing factor is somewhat suggestive of a chronic endocrine deficiency. That endocrine changes occur has also been shown by Seishichi Ohno (11), who finds an hypertrophy of suprarenal medulla in beri-beri cadavers. This agrees with McCarrison's observations on underfed birds, and is also in accordance with post-mortem appearances the writer has seen in emaciated prisoners of war with so-called war oedema. Several Turkish soldiers, when captured by us in 1918, were in this condition, and some showed post-mortem suprarenal hypertrophy.

The idea that beri-beri is due to a food deficiency and to nothing else has of recent years been too dogmatically stated. Drs. Harriette Chick and Margaret Hume begin their paper (12) on 'The Distribution among Food-stuffs of the Substances required for the Prevention of Beri-beri and Scurvy', by saying, 'Sufficient evidence is now available to permit beri-beri . . . to be classed among the "deficiency" diseases due to a defective diet, without further comment.' Similarly a recent lecturer (13) states: 'Beri-beri is a deficiency disease, due to reliance upon polished rice': like statements have also been made by leading physiologists (14 and 2). The numerous medical officers who have worked in Mesopotamia during the last three years arrived in the country imbued with that teaching. Many of them have come in contact with British or Chinese cases and tried to reconcile their previous teaching with the facts as described here and with the conditions under which these cases have arisen. This they have been unable to do, and many have left Mesopotamia with the impression that the accepted theories as to the causation of beri-beri are incomplete.

This paper states the principal facts of a Mesopotamian experience, and attempts to reconcile them with previous clinical observations and animal experiments. There are still difficulties in our entire understanding of the subject; but the comparison of the various conditions under which beri-beri has arisen in the Mesopotamian campaign will help, the writer hopes, to throw some light on the origin of the disease, and to explain some of the apparent contradictions that medical officers have been faced with.

Summary.

The disease called beri-beri is a syndrome which may arise from various causes under different circumstances. One class of case is not due to a food deficiency; but appears to result from an infection. Other cases are due to a vitamine deficiency in the food: this takes a few months to operate in a previously healthy subject and may be called 'primary beri-beri'.

In yet another class the syndrome arises apparently from the effect of some depressing influence or secondary infection on a subject previously rendered susceptible to the disease. These cases may be considered to have been suffering from 'latent beri-beri'. In all classes the clinical appearance is approximately the same, there being differences only in the relative frequency of various manifestations in the three groups.

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 Lt.-Col. G. S. Buchanan, C.B., R.A.M.C.
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