A CONTRIBUTION TO THE STUDY OF THE ETIOLOGY OF BERI-BERI.

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PREFACE.

A RECENT outbreak of beri-beri in the 83rd Wallajahabad Light Infantry has lent such valuable and interesting support to the view that this disease is directly connected with a diet of stale, raw, milled rice, that it appears well worthy of record.

Certain points favouring an intoxication theory of the etiology of beri-beri, with a supposititious causal organism flourishing in the foodstuff, as against the more recent hypothesis of a deficiency of some constituent of the grain, removed in the rice polishings, are noted.

Under a separate title, the relationship of beri-beri to epidemic dropsy is considered, as the outbreak in the 83rd Regiment provided cases which seem to link up these two diseases; and attribute them both to the same fundamental pathological factor.

For the sake of simplicity, the subject of this paper has been divided under eight heads.

(1) Recent views upon the etiology of beri-beri.
(2) The outbreak of beri-beri in the 83rd Regiment.
(3) The history of beri-beri epidemics in the 83rd Regiment.
(4) Native practice in the treatment of beri-beri.
(5) The two leading theories as to the etiology of beri-beri.
(6) The probable existence of beri-beri amongst non-rice-eating communities, with a possible explanation thereof.
(7) Clinical histories of the cases of beri-beri in the 83rd Regiment.
(8) Summarized diagnosis of the epidemic.

It is as well to start with a clear idea as to the difference between "raw" and "parboiled," "fresh" and "stale" rice.

Rice known as "raw" is the ordinary "white" rice of commerce. After being gathered as paddy from the fields, it is directly husked, without any preliminary boiling by a heavy milling process, which removes with the husk the whole of the red envelope of the grain. Raw rice may be imported from abroad (i.e., Rangoon, Siam, &c.), and is then known as "foreign" rice, or it may be of local production, i.e., "country" rice. In the latter case, it is probably sold soon after being gathered, and hence is likely to be "fresh" rice.
In the former case, it is probably milled and stored on a large scale and supplied as the demand requires, and hence is very probably "stale" rice.

Parboiled rice is rice which, having been garnered, is half-boiled before being husked. This process may be repeated two or three times. Subsequent to this parboiling, the husk is removed, usually by hand-pounding or more rarely in mills. The resulting grain is yellowish and waxy looking.

I.—RECENT VIEWS UPON THE ÄETIOLOGY OF BERI-BERI.

Before discussing the ætiology of the present outbreak of beri-beri in the 83rd Regiment, it will perhaps be as well to review briefly the recent opinions deduced from experiments on the causation of this disease.

Firstly, Leonard Braddon (Proc. Bom. Med. Congress, 1909) teaches that the cause of beri-beri is an intoxication resulting from the ingestion of a decorticated, raw, stale rice—this deduction he bases on three main facts.

(a) In the Malay Peninsula, one quarter of a million cases of beri-beri have been under observation, and these have occurred amongst rice-eating people only. None have arisen amongst non-rice-eaters.

Of the quarter million cases, 97.5 per cent were amongst the Chinese, who eat a raw, milled, stale rice. Another 1.5 per cent are furnished by other natives eating this same variety of rice. Tamils feeding on boiled rice, and Malays, feeding on fresh raw rice, absolutely escape.

(b) Dr. W. Fletcher’s feeding experiments on patients in the lunatic asylum at Kuala Lumpur, in Selangor, lasting two and a half years. One half of the patients fed on raw, milled rice developed beri-beri, whilst the other half fed on boiled rice totally escaped.

(c) Fraser’s, Stanton’s and Braddon’s experiments at Durian Tipus (Lancet, February and May, 1909), on coolies employed at road making. Only those coolies developed beri-beri who ate raw rice, those who ate boiled rice escaped. For further details of the carefully conducted and apparently conclusive experiments, reference may be made to the original articles.

Secondly, Schaumann (Indian Medical Gazette, November, 1909), experimenting with pigeons, rabbits, &c., concludes that nucleo-proteins are absolutely necessary for the organism, and that their absence gives rise to degeneration of nerve fibres, polyneuritis, &c.
In several cases of beri-beri developing on sailing-ships it was found that the foodstuffs were mouldy. Moulds have a great avidity for phosphorus and to obtain this they break up nucleoproteins.

He insists on the greater phosphorus content of parboiled (0.754 per cent) as compared with raw (e.g., Burma) rice (0.323 per cent), which has the entire pericarp removed during the process of husking—i.e., is heavily milled.

Thirdly, Fraser and Stanton (Proc. Lond. Soc. Trop. Med., February, 1910), report that they fed three groups of domestic fowls for five weeks on three varieties of rice. The first group fed on unmilled rice remained healthy, the second group fed on milled rice developed polyneuritis, the third group fed on milled rice plus the polishings removed from the rice during the milling process remained healthy. Fowls fed on parboiled rice likewise remained healthy. These experiments apparently show that it was the milling process which was at fault. An analysis of the percentage of phosphorus content varied inversely as their known beri-beri-producing tendency. Thus parboiled rice contained more phosphorus (0.469 per cent) than milled raw rice (0.277 per cent). Moreover the rice polishings removed in the milling process contained a relatively enormous amount of phosphorus (4.2 per cent). Fraser and Stanton therefore conclude that beri-beri is caused by deficiency of phosphorus in the diet. This phosphorus, in the case of rice, being removed with the pericarp of the seed in the polishings during the process of milling. It will be noticed that the phosphorus estimations of the two varieties of rice roughly agree with those obtained by Schaumann, parboiled rice containing about twice as much phosphorus as milled raw rice.

Fourthly.—At a meeting of the Far Eastern Association of Tropical Medicine, at Manila (Brit. Med. Journ., April 23, 1910, and Lancet, April 30, 1910), attended by the official delegates from the Governments of India, Hong Kong, Philippine Islands, Tsingtau (Imperial German Government) and Japan, the following resolution was passed: “That in the opinion of this Association sufficient evidence has now been produced in support of the view that beri-beri is associated with the continuous consumption of white (polished) rice as a staple article of diet, and the Association accordingly desires to bring this matter to the notice of the various Governments concerned.”

Fifthly.—The French School (Annales d’Hygiène et de Médecine
The Study of the Ætiology of Beri-Beri

Coloniales, No. 1, of 1910, and Le Caducée, February 19, 1910 holds that in a "red rice", freshly husked, and a more perfect preservation of milled rice, the deterioration of which is difficult to prevent in these latitudes . . . ." lies the prevention and cure of beri-beri. Dr. Thèze, writing of the 1906 (? 1909) epidemic at Poulo-Condore, says that it was a year of average beri-beri intensity. Of a total of 130 deaths at the penitentiary, 119 must be assigned to beri-beri. "From the commencement of the disease," he writes, "convinced according to the general opinion that beri-beri was contagious and due to a parasite, either a microbe or intestinal worm, we brought our attention to bear on the thorough disinfection of the convict prison; the walls, beds and floors were whitewashed weekly . . . . But new cases and deaths occurred and white rice was still the chief food of the convicts. Thus checked in our efforts, red (half-husked) rice was substituted for white (cleaned) rice on August 13. The epidemic reached its height on August 15, and then ended very abruptly on August 22, with 119 deaths. No new cases were reported from August 20, and the last death took place on the 22nd." He upholds the theory of deficient phosphoric food value, but seems to pay a little attention to the fact that the red rice used

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of rations distributed to Asiatic convicts during the year</th>
<th>Average of daily rations</th>
<th>Number of deaths due to beri-beri</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1909</td>
<td>234,716</td>
<td>615</td>
<td>80</td>
<td>From November 5, 1908, the red rice necessary for the Asiatic convicts was husked at the prison.</td>
</tr>
<tr>
<td>1900</td>
<td>239,356</td>
<td>628</td>
<td>78</td>
<td>the last four years were among the Chinese and natives from the interior, already attacked before being imprisoned.</td>
</tr>
<tr>
<td>1901</td>
<td>185,350</td>
<td>507</td>
<td>92</td>
<td></td>
</tr>
<tr>
<td>1902</td>
<td>169,661</td>
<td>464</td>
<td>85</td>
<td></td>
</tr>
<tr>
<td>1903</td>
<td>213,231</td>
<td>584</td>
<td>78</td>
<td></td>
</tr>
<tr>
<td>1904</td>
<td>272,315</td>
<td>746</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>1905</td>
<td>284,613</td>
<td>779</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>1906</td>
<td>313,091</td>
<td>860</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>1907</td>
<td>278,204</td>
<td>762</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

1 I am indebted to Major Clements, R.A.M.C., for these references, and also to Miss Rennick who translated Dr. Thèze's essay and the Reports of the Directors-General of Health of Indo-China and Cochinchina, for me.

2 The rice contractor at St. Thomas' Mount tells me that "red" rice commercially means invariably parboiled rice. Raw rice with its red envelope is, he says, never called "red" rice, but I do not know in which sense the writer uses the term.
was “freshly” husked, whilst the white rice was old and stale. The substitution of this freshly husked red for an old white rice, he says, “played a curative part far superior to all the antiseptic or anthelminthic treatments (thymol included) hitherto employed. The editorial adds, “It is the modification in the food of the invalids which explains the perfect cure of all these patients returned to their families, and for whom nothing was changed except the character of their rations.”

The remarkable table is given on the preceding page.

The French School undoubtedly attaches more importance to the heavy milling factor and the entire removal of the pericarp as the cause of beri-beri than to the factor of the staleness or freshness of the rice consumed. Indeed, Surgeon-Major Bréaudat, of the Pasteur Institute, of Saigon, has been permitted by the French Government to try the preventative and curative effect of the administration of rice polishings to the native prisoners and troops. The polishings must be the fresh produce, described in the husking mills under the name of rice flour, first quality. It must be sifted with care and should not contain any particles of husk which would irritate the digestive tube.

The following is the manner of prescribing it:

<table>
<thead>
<tr>
<th>Sifted rice bran</th>
<th>…</th>
<th>…</th>
<th>…</th>
<th>…</th>
<th>100 grm.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple syrup</td>
<td>…</td>
<td>…</td>
<td>…</td>
<td>…</td>
<td>60 &quot;</td>
</tr>
<tr>
<td>Essence of peppermint</td>
<td>…</td>
<td>…</td>
<td>…</td>
<td>1 c.c.</td>
<td></td>
</tr>
</tbody>
</table>

Make into a paste and divide into ten boluses. Dose, 2 to be taken with each meal, 6 (600 grm. bran) daily. The published results of four months up to October, 1909, certainly seem encouraging and further details will be awaited with interest.

Sixthly.—For the results of Fraser and Stanton’s most recent work (Lancet, December, 1910) announcing their renunciation of the phosphorus theory (see note at end of heading V.).

To sum up, during the last eighteen months, practically every investigator of the etiology of beri-beri has been adding the results of his work to our common knowledge, and we may now conclude that a constant diet of raw, heavily-milled rice, especially if the superadded factor of staleness be present, is likely to give rise to an outbreak of beri-beri amongst its consumers—whilst a diet of fresh country or parboiled rice will not do so.

II.—The Outbreak of Beri-beri in the 83rd Regiment.

The present epidemic broke out in the 83rd Regiment during the months of July to November, 1910. The first patient, who only
The Study of the Ätiology of Beri-Beri

complained of slight leg pains and of inability to march, I thought was a malingerer. I failed to diagnose the next few cases, which showed oedema of the legs, altered knee-jerks and various forms of paresthesia. When one of them, however, whilst under observation, suddenly developed peripheral neuritis, the diagnosis at once became clear, and the cause of the oedema noted in the early cases was immediately manifest.

The following are the chief aetiological points in connexion with the outbreak.

(1) Of the twelve cases, nine were recruits obtaining their rice direct from the Mohammedan recruit mess, one was a private, feeding at the same mess, whilst the remaining two were buying rice from the Bengali Bazaar, which was of the same variety as that supplied to the Mohammedan recruit mess.

(2) All the patients were Mohammedans; and all, as noted above, were eating the same variety of rice.

(3) The rice on which all these beri-beri patients were feeding had the following characters:
   (a) It was raw rice. (Not parboiled rice.)
   (b) It was stale rice. (Not rice fresh from the fields.)
   (c) It was milled rice. (Husks not removed by hand pounding.)
   (d) It was Bezwada rice.

Now this is exactly the very type of rice, described by Leonard Braddon and by his co-workers, as being the aetiological factor in the production of beri-beri.

It may here be noted that the rice was neither mouldy nor weevily—and was repeatedly passed by myself as good rice fit for food.

(4) None of the Hindus or native Christians of our Regiment, who were using a parboiled rice (even though this was milled and from Bezwada), developed beri-beri, nor did any of our non-rice-eating Mohammedans fall victims to this disease (with one single exception, vide below, (5)). All our beri-beri cases had this one factor in common, namely, they were consuming a raw, stale and milled Bezwada rice.

(5) One patient, Pte. Abdul Nairn, had eaten atta all his life, save on certain festival occasions when he would take some rice. On returning from furlough on June 5, 1910, he found himself moneyless. In order to obtain food, he indented on the Mohammedan recruit mess, which advanced him rations and deducted the cost thereof later from his pay. For the first time in his life, then, he fed on a staple rice diet, which, moreover, happened to be stale,
raw and milled. He continued to do this for four and a half months, when he developed beri-beri.

(6) On the ninth case appearing amongst the Mohammedan recruits, and not before, one change was made, and one change only. This change was to replace the raw, stale, milled, Bezwada rice supplied to their mess by a fresh (though still raw) hand-pounded, local rice, with the addition of a phosphorus pill twice weekly to their diet. No disinfection or earth-turning operations were instituted. There has not been a case of beri-beri amongst the recruits since.

(7) The nine recruit cases were all, naturally, in the same barracks, but the cases were not sleeping side by side; there is no evidence to support a contact theory as a cause of this epidemic. No precautions whatever have been taken in connexion with the disinfection of barracks or beds, nor have patients in hospital been isolated, yet no contact cases have been recorded.

(8) There are no grounds on which to support a theory of poisoning by arsenic, oxalates or fish—indeed, none of the patients as far as could be discovered had partaken of the two former at all, nor of the latter for many months. Ashmed's carbon dioxide poisoning theory will not hold, for the military open-air life of the recruits and their roomy, well-ventilated barracks preclude the possibility of over-crowding. Deficiency of nitrogen, fat or vegetables, cannot explain the outbreak, which affected Mohammedans alone and not the Hindus or native Christians, as the only difference in their diets was the difference between raw and parboiled rice.

(9) As to the many suggested and described protozoal, helminthological and bacterial causes for beri-beri, no recognized abnormal animal or vegetable organism was discovered in film examinations of the blood, urine or faces of any of our patients. The total red and white corpuscle as also the differential blood-counts were normal, whilst blood cultivations from the patients' median basilic vein all remained sterile.

As a result, then, of a careful study of the aetiology of this epidemic, I believe that the direct cause of its outbreak was a diet of raw, stale, milled rice, and this in spite of the fact that it was consumed with a variety of foodstuffs. (The daily recruit diet consists of rice 24 oz., mutton 4 oz., dall 2 oz.)

III.—History of Beri-beri Epidemics in the 83rd Regiment.

The history of beri-beri in the 83rd Regiment is intensely interesting. The regimental records show that since 1899 there have
been four recognized small outbreaks of this disease. One occurred at Secunderabad in 1899-1900, one at Rangoon in 1902, one at Cannanore in 1908, and the present one at St. Thomas' Mount in 1910. There were probably many other cases of beri-beri, occurring both during the epidemic, and also in the intervening years, which were diagnosed and returned as multiple neuritis or oedema of the legs, or which only attended the hospital and were shown as detained.

Thus during the years 1905-1909 the following number of cases were signed up in the annual regimental return of sick and wounded as suffering from these groups of symptoms.

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases of multiple neuritis or neuritis</th>
<th>Cases of oedema</th>
</tr>
</thead>
<tbody>
<tr>
<td>1905</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>1906</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>1907</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>1908</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>1909</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

And again, in regard to the 1908 Cannanore outbreak, Lieutenant-Colonel J. A. Burton, I.M.S., says casually in a report that several slight cases of oedema were only detained, or attended the hospital without being shown in the records.

It would seem probable, therefore, that during the past few years many cases of beri-beri, in addition to the described epidemics, have existed in the regiment without being returned as such, and indeed the 83rd have probably suffered from the disease more or less constantly since 1900.

Now the point to be noted in the aetiology of these small epidemics in this regiment is, that during the first three outbreaks Hindus, native Christians and Mohammedans alike were all attacked by the disease, but during the present 1910 outbreak Mohammedans, and Mohammedans only, have suffered. Not a single case has occurred among the Hindus or amongst native Christians, who in former epidemics were both equally affected.

Why was this?

The tale told me by the native officers in charge of the various messes at the different stations is very interesting. These officers, moreover, knew nothing of the value of the information they were imparting, nor were any leading questions put to them. They said that at Secunderabad, in 1900, boiled rice was hard to obtain. All the Mohammedans and the majority of the native Christians and Hindus therefore, were eating raw rice. In 1901, the regiment left for Rangoon, where raw rice only was supplied throughout the messes. In 1904 they went to Bellary; but, having
formed the habit of eating raw rice in Rangoon, they continued it, and also at Cannanore, where they arrived in March, 1908. At this station the raw rice they used came by steamers. During these periods then, Mohammedans, Hindus and native Christians alike were feeding on raw rice, and cases of beri-beri appeared indiscriminately amongst all these groups. But during the Cannanore epidemic of August and September, 1908, Lieutenant-Colonel J. A. Burton, I.M.S., reported that the raw rice consumed by the sepoys was not only stale and mouldy, but also full of weevils, and had an offensive odour, and advised that it should be changed. Parboiled rice was, therefore, issued to the Hindus and native Christians, whilst the Mohammedans who refused parboiled rice were supplied with a fresher and better variety of raw rice. Since that change not one case of beri-beri, of peripheral neuritis, or of oedema of the legs has occurred amongst the Hindus or native Christians, but the present epidemic has broken out amongst the Mohammedans who alone were feeding on raw rice.

As mentioned elsewhere, the raw rice which caused this epidemic was a stale, raw, milled, Bezwada rice. Immediately it was changed in November for a local, fresh, raw rice from Madura, Tanjore, &c., beri-beri ceased among the Mohammedans and not one case has arisen since.

The history of beri-beri in the 83rd Regiment then, seems to support in a remarkable way the theory that on a diet of parboiled rice beri-beri will not occur, whilst on a diet of raw rice, and especially if this rice be stale and milled, beri-beri may at any moment break out.

IV.—Native Practice in the Treatment of Beri-beri.

When the village hakim or native elder is called upon to treat that form of oedema which we know as wet beri-beri, he seeks out, they say, two leaves, the one a large five-lobed leaf which grows over walls and trees, and the other a small leaf growing wild on almost any maidan. From these two leaves he prepares a decoction, and administers it to the patient as a diuretic. In addition he makes one alteration in the diet. The patient is told not to eat rice, but to prepare chapatis from wheaten flour.

The treatment of the native elder, which has probably been founded on experience, supports the theory that rice is a causative agent in the production of beri-beri.
The Study of the Aetiology of Beri-Beri

V.—The Two Leading Theories as to the Aetiology of Beri-Beri.

A study of the experiments made recently on the aetiology of this disease, the experience of the present outbreak in the 83rd Wallajabad Light Infantry, the history of past epidemics in the regiment, and, to a less extent, the support lent to the theory by the practice of the native elders, seem to leave no doubt whatever that at least one cause for an outbreak of beri-beri is a diet consisting of raw, milled, stale rice.

As to which of these three factors is the most important it is somewhat difficult to decide. That the rice should be raw is probably a sine qua non. If boiled rice be the staple diet then no epidemic of beri-beri will break out, or if it was ever found to do so then the other two factors would probably be discovered to exist in an accentuated form.

The influence of heavily milling the rice seems from Fraser’s experiments to be of decided importance. By this process of polishing the red pericarp is in great part removed from the grain and the resulting highly polished white rice, when forming a staple diet, may at any moment apparently give rise to an outbreak of beri-beri.

We may here note that a possible reason why a boiled rice diet will not produce such an outbreak, whilst a raw rice one will (although both rices have been subject to an equally heavy milling), is because parboiling considerably toughens the pericarp and thus renders it far less liable to be removed completely in the milling process.

The effect of this process on the rice, if we accept Fraser’s and Stanton’s and Schaumann’s experiments, is the loss of phosphorus. Thus on this theory beri-beri becomes a disease of metabolism due in fact to a continued starvation of the individual as far as the phosphoric element in his diet is concerned. Now phosphorus, we know, is an essential food of the nervous tissues and, in its comparative absence from a given diet, one would naturally expect a degeneration of the nerve elements to follow, which is indeed the fundamental pathological change in every case of beri-beri (Yamagima, Wright, Durck).

As to the quality of staleness of the rice—it is apparently a fact that rice, although raw, provided it is freshly garnered from the fields, and lightly hand pounded, will not produce an outbreak

1 See note at the end of this heading.
of beri-beri. But if the rice is raw and stale (and not stored with such care as is taken at the Presidency Jail, Calcutta), then such an outbreak is probable. The quality of staleness then has probably some important influence in the determination of the onset of an epidemic of beri-beri.

Moulds too, as Schaumann points out, thrive on phosphorus and to this end greedily break up nucleo-proteins, which in rice abound in the pericarp. Thus we can readily understand how any mouldy rice of whatever type may give rise to beri-beri—just as any heavily milled rice would—by a removal from it through the agency of the moulds of the natural phosphorus-containing nucleo-protein element.¹

Indeed these recent researches seem to be really tending to explain and support the old protein starvation theory, but with the further addition of scientific reason and accurate detail.

If this theory of phosphorus starvation proves to be the true one, we may possibly find that those puzzling epidemics amongst non-rice-eating communities, which have been reported from time to time, may be due to the fact that the given diet in each individual outbreak was deficient in nucleo-protein (phosphorus) food value.

But so far we are only touching the very threshold of the aetiology of beri-beri. The point of absorbing interest is this—is beri-beri a disease of metabolism only, or is it due to an infection?

So far in this paper it has been written of as a disease of metabolism. What can be said in support of a theory of infection?

Firstly, that if beri-beri is an infection then the germ does not probably live in the patient’s body. For contact cases do not seem to occur, inoculation experiments are unsuccessful, blood cultivations by many observers are futile and the cellular blood elements remained undisturbed. The infection then if it exists, is probably not a direct germ infection.

Secondly, the periodicity of the disease, and its occurrence in epidemic form, and the frequency with which it selects healthy young adults as its victims, do not favour a theory of disordered metabolism.

Thirdly, of our twelve cases of beri-beri, three whilst under observation developed a definite high fever lasting for an average of five days in each case.

¹ See note at the end of this heading.
Now there were absolutely no symptoms of the onset of any independent pathological process to explain this pyrexia. In each case it commenced and terminated abruptly. Daily blood examinations and cellular counts failed to elicit any sign of malaria or the presence of other abnormal processes. There was no similar fever amongst any other patients in the hospital wards, nor indeed in the station. No physical signs were present anywhere in the patients’ body to explain its occurrence.

But in each case, coincidentally with the rise in temperature, there was a most decided increase in the patient’s beri-beri symptoms. Two of the cases on the second day of the fever suddenly developed a most marked peripheral neuritis with wrist and ankle drop and paralysis of the gluteal and spinal muscles, so that they were unable to raise themselves in bed. The third case was more fortunate. His symptoms of leg oedema and all altered sensations became increased, without the onset of any sign of severe neuritis.

Now, although one is taught that in beri-beri there is no definite high fever, yet three of our cases developed it in a most decided manner. I do not believe that this temperature could be explained by any discoverable fresh morbid process. I am forced to believe that the pyrexia was the manifestation of some acute condition in the patients’ bodies, which was a few hours later also responsible for a most severe accentuation in the patients’ beri-beri symptoms.

If this be so, no simple theory of abnormal metabolism alone is sufficient to explain the aetiology of beri-beri. This theory must be replaced by one of poisoning by the products of germ life, or the two may be combined.

To harmonize the facts supporting these two theories is indeed a difficult task, which however must be attempted. That beri-beri is an intoxication is held by two such authorities as Sir Patrick Manson and Leonard Braddon. The former is inclined to believe that the germ grows in the patient’s environment, in the rooms, the soil or the ship. He advances strong arguments for an intoxication theory, and then asks: Why should patients begin to mend so rapidly after they leave the locality? To me there is little evidence to support a place infection theory, compared with that surrounding an infection of food-stuffs, and the answer to Sir Patrick Manson’s question appears to be: Because on leaving the locality the patient obtains a fresh and non-infected diet.

Leonard Braddon believes that the germ lives on the outside of
the grain, and produces a toxin, which when ingested with the rice gives rise to beri-beri. Parboiling rice directly after it has been gathered kills the germ, if present, and hence no toxin is produced, and no beri-beri outbreak will occur. With raw rice on the other hand the germ may continue to thrive. The staler the rice, the more the toxin produced, and the greater the risk of contracting beri-beri. This theory, however, does not explain the supposed heavy milling factor in the production of beri-beri, nor why rice polishings, together with a heavy milled rice diet, will not give rise to beri-beri-like symptoms in fowls. It must be remembered, however, that the evidence upon which the heavy milling of rice as an important factor in the production of beri-beri depends is in the main laboratory evidence. And, moreover, this laboratory evidence rests on the production of multiple neuritis in fowls—a disease which has in the past led two former observers astray, and may of course be one quite different from beri-beri. And one is almost tempted to forsake for the time being such testimony in the face of other overwhelming observed facts collected by men from actual clinical experience with beri-beri patients.

That the probable growth of some germ in the rice is the causative factor in the production of beri-beri seems further to be supported by the freedom which the Presidency Jail, Calcutta, enjoys from an outbreak of this disease. Since 1905, stale, raw, milled Burma rice has been supplied to the prisoners, but this rice is stored with great care, one seer of slaked lime being added per maund of rice to preserve it. This powdered slaked lime is washed away before the rice is issued as food. One at least can say this, that although the rice is just the type we would expect to give rise to an epidemic of beri-beri, yet by the addition of a substance, during storage, which inhibits germ life, the prisoners remain free from beri-beri. On the other hand, if this disease is due to the removal of some essential food constituent during the heavy milling process, which the particular rice in question has been subjected to, how can the addition of slaked lime during storage give the prisoners the immunity to beri-beri they possess?

To sum up, beri-beri may be a disease of metabolism or a toxemia. The view that beri-beri is an intoxication with the causative germ multiplying in a food-stuff medium under atmospheric

1 Maurer and Trentlein produced a condition resembling beri-beri in fowls, polyneuritis gallinorum, by giving them oxalic acid in their food, and on these experiments based their oxalic acid theory.
conditions of high temperature and moisture is the one which would certainly appear most probable. The food-stuff which the germ commonly infects is rice. Parboiling the rice kills the germ and stops the toxin production. Infected raw rice, if stale, will contain much toxin and will give rise to an epidemic. The influence of heavy milling in connexion with this theory had best be left untouched until further investigation has cleared the ground to a greater extent than is at present the case.

But, however difficult all this lore on the subject of beri-beri may be, we have one clear practical fact before us—parboiled rice will not give rise to beri-beri, while a rice stored without precautions, milled and stale, will bring about an outbreak of this disease.

**Note.**

Since this paper was written, Fraser and Stanton have published a further valuable research on the etiology of beri-beri (Lancet, 1910, vol. ii. p. 1,755). Although still holding that this disease is brought about by a white rice which has had the pericarp too thoroughly removed from it by a heavy milling process, and that the polishings contain the essential for the prevention of beri-beri, yet they have satisfied themselves that this essential is not phosphorus, but some as yet unnamed precipitation principle which they have separated.

This important result would appear to shake the foundation of the metabolism theory to a greater extent than ever, and hence indirectly lends additional weight to the intoxication hypothesis.

VI.—**The Probable Existence of Beri-Beri Amongst Non-Rice-Eating Communities, with a Possible Explanation Thereof.**

No paper on the etiology of beri-beri would be complete without a brief reference to its apparent existence amongst practically non-rice-eating communities.

The reference must of necessity be brief, for but little is known of this part of the subject, and my own practical experience of it is unfortunately nil.

Now there are two questions to which an answer must be attempted:—

**Firstly:** Does it exist?

**Secondly:** If so, is any possible explanation forthcoming?

To the first question then, until it is disproved, a guarded reply in the affirmative may be given; for there are on record many out-
breaks of beri-beri, reported by competent medical officers, amongst British regiments and European crews of ships and amongst isolated individuals, where stale rice has apparently been a negligible factor. There is, too, the history of the famous outbreak at the Richmond Asylum, Dublin.

Lieutenant-Colonel McGill, R.A.M.C. (Proc. Bom. Med. Congress, 1909), reported sixty-one cases of shin oedema and tachycardia, associated with pain and weakness of the legs, which occurred in a British infantry regiment at Poona in 1907. The diagnosis lay between alcoholic neuritis and beri-beri. But although the men were alcoholics, yet the outbreak appeared in epidemic form. So that although the cases were possibly (and perhaps probably) cases of alcoholic peripheral neuritis with heart trouble, yet one cannot utterly ignore the possibility of a diagnosis of beri-beri being the true one.

Major Clements, R.A.M.C., tells me of an outbreak which he watched in a British battery at Mandalay in 1901, in which the symptoms described still more closely resembled those of beri-beri. All were alcoholics, and yet other British troops within a few miles, equally alcoholic, were unaffected. The outbreak, too, was of an epidemic character, some twenty cases occurring amongst some eighty men within a few weeks in the hot damp season.

The Annual Reports of the Sanitary Commissioner with the Government of India, too, since 1900, have yearly shown some outbreaks returned as beri-beri amongst the British troops of the Indian Army, but no satisfactory explanation of any epidemic has yet been forthcoming.

In the face of these and similar reports then, it would be fool-hardy, without practical experience, to deny the existence of beri-beri, where its presence could not be explained by the stale, raw rice theory.

Secondly, granted for the present that beri-beri may exist amongst non-rice-eaters, is there any possibility of connecting up these puzzling outbreaks and those undoubted epidemics amongst rice-eaters which are certainly due to the ingestion of a stale, raw rice, and probably to a toxin the result of the growth of an organism therein?

I believe that this possibility exists, and it is somewhat supported by an outbreak of beri-beri which Captain Gharpurey, I.M.S., tells me he observed whilst in Aden, in 1908. He was then in charge of the 113th Infantry, and amongst them some six cases presenting signs of leg oedema, altered knee-jerks, tachycardia, partial anaesthesia...
and marked tenderness of the calf muscles occurred. At the same
time in this hot, damp climate, twenty-eight cases presenting
similar signs were reported from the British troops. Now of the
several native cases, some two took a small amount of rice
daily, whilst the remainder were atta feeders alone. Now the
quantity of rice consumed by the latter and by the British garrison
was negligible. None of these natives took alcohol, and the
medical officer at Aden reported that the incidence of the disease
was not greater in the case of the British troops among beer
drinkers than among others (Report of the Sanitary Commissioner
with the Government of India, 1908). But Captain Gharpurey
noticed this most important point. The atta supplied to the native
troops was mouldy, stale and weevily. The same atta was baked
into wheaten loaves and supplied to the British troops. This atta
flour had been imported from India, and stored in large quantities
for some time in the Aden godowns, and hence was consumed at an
uncertain date after being prepared from the fresh wheat grain. No
satisfactory cause for the outbreak has been brought forward.

The similarity between this epidemic of beri-beri, possibly due to
stale weevily wheat flour, and those outbreaks which we know are
due to stale rice is apparent, and at once suggests the possibility of
a far broader view of the aetiology of this disease.

Might beri-beri not be a toxæmia produced by a specific germ
infection of various stale grains (e.g., rice, wheat), and, perchance, of
even other still more varied food-stuffs? This suggestion at present,
is, of course, merely a bye-thought, which however would seem
worthy of attention by those who have to deal with outbreaks of a
beri-beri-like disease amongst non-rice-consumers.

VII.—CLINICAL HISTORIES OF THE CASES OF BERI-BERI IN THE
83RD WALLAJABAD LIGHT INFANTRY.

The chief clinical facts brought out by the 83rd Wallajabad
Light Infantry patients collectively were as follows:—

Of the twelve patients, eight evidenced only such comparatively
slight signs of their disease as œdema, various forms of paraesthesia
and altered knee-jerks. One was a picture of exaggerated muscular
inco-ordination, with the gait and bearing of a case of pseudo-
hypertrophic muscular paralysis. Three, after first being under
observation for œdema, paraesthesia, and loss of knee-jerks,
developed marked peripheral neuritis, two of these cases being of
the severest possible kind.

The more important symptoms grouped under the heading of
the separate systems were:—
H. Stott 247

(1) Nervous System.—(a) Hyperæsthesia definitely existed in two cases.

(b) Paræsthesia, such as sensations of weakness and heaviness of limbs, numbness, tingling and dragging pains, was present in every case.

(c) Feather Anæsthesia.—In ten this was slight and affected practically the lower extremities only. One had complete feather anaesthesia of the whole body excluding the dorsal aspect of the head and back. One had no anaesthesia at all.

(d) Peripheral Neuritis.—Three cases showed definite severe peripheral neuritis—two of these developed it suddenly (with pyrexia) in the middle of the night, whilst in the case of the third the peripheral neuritis took four months after his first symptoms to develop, until he became absolutely unable to walk or to stand at all. Two other cases had definite tenderness on pressure of the calf muscles.

(e) Pain was not a prominent symptom, save in the two cases of acute peripheral neuritis, in both of which the limbs were exquisitely tender.

(f) Reflexes.—Knee-jerks in eight cases were absent throughout. In one case the jerk was always present. In one case it was noted as being increased at first, diminished later, but always to be obtained. In two cases it was always most markedly and definitely increased, one of these being watched for a period of six weeks.

The superficial reflexes were invariably present.

(g) Atrophy of Muscles.—Almost all the patients showed some atrophy of their calf muscles, which was proportionate to the other symptoms.

(2) Circulatory System.—(a) Palpitation of the heart; (b) oppression in the chest; (c) shortness of breath; (d) pains in the heart region, were all common.

The pulse was frequently rapid—especially after marching a few yards—some soft cardiac bruit, especially over the pulmonary valve, was the rule. No definite evidence of right-sided heart dilatation was ever noted. One patient bled from the nose. No alteration in the general blood condition of our patients occurred. The blood counts were normal and averaged:

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total white cell count</td>
<td>5,938</td>
</tr>
<tr>
<td>Total red cell count</td>
<td>5,230,000</td>
</tr>
<tr>
<td>Percentage of red cells</td>
<td>105 per cent.</td>
</tr>
<tr>
<td>Total haemoglobin</td>
<td>97</td>
</tr>
<tr>
<td>Colour index</td>
<td>0.9</td>
</tr>
<tr>
<td>Ratio of white cells to red</td>
<td>1 to 880</td>
</tr>
</tbody>
</table>
The average differential count of all the patients was also normal and worked out as follows:—

<table>
<thead>
<tr>
<th>Leucocytes Type</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polymorphonuclear leucocytes</td>
<td>68</td>
<td>68 per cent</td>
</tr>
<tr>
<td>Large mononuclear lymphocytes</td>
<td>10</td>
<td>10 per cent</td>
</tr>
<tr>
<td>Small</td>
<td>15</td>
<td>15 per cent</td>
</tr>
<tr>
<td>Transitional cells</td>
<td>2</td>
<td>2 per cent</td>
</tr>
<tr>
<td>Eosinophile</td>
<td>1</td>
<td>1 per cent</td>
</tr>
<tr>
<td>Mast cells</td>
<td>4</td>
<td>4 per cent</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>100</td>
<td>100 per cent</td>
</tr>
</tbody>
</table>

(3) *Respiratory System.*—Attacks of oppression in the chest and of shortness of breath, as noted above, were complained of.

(4) *Digestive System.*—Constipation was the rule, two patients had diarrhoea, one vomited, and one passed slightly bloody mucous stools for two days.

(5) *Urinary System.*—The urine was measured in four cases and found to be slightly diminished, about 40 oz. daily. Every case was repeatedly tested for albumen and sugar, but these were never found to be present.

(6) *Skin and Serous Membranes.*—(a) Rashes were not noted in any patient.

(b) *Oedema* occurred in ten cases: this was limited to the lower limbs, but in two of these the patients complained of feeling "like a stick, as though their whole body were swollen." No sign of this could be discovered. In the remaining two cases, whilst under observation, the oedema was noticed to spread up over the entire body, thighs, loins, abdomen and face.

(c) Circumscribed Oedemas were not seen.

(d) *Effusions into Serous Cavities* were not noted, although repeatedly tested for.

(7) (a) *The General Condition of the Patients* was good, and, but for the cases of paralysis and those complaining of respiratory or cardiac distress, they gave no cause for anxiety. One patient suffered from malaria as a complication.

(b) *Pyrexia.*—Three of the series developed a definite high fever for three or four days, which could not be explained in any other way than that it was due to beri-beri toxin.

This conclusion, moreover, is supported by the fact that two cases out of the three developed a multiple neuritis on the second day of the fever, whilst the third showed a decided increase in the severity of his symptoms.

Five others had slight early pyrexia varying from 99.5° to 102°.
usually for one day only, without showing malarial parasites in their films, or any signs suggestive of malaria.

(c) Previous Attacks.—One patient said he had had leg edema a year ago.

(d) Mortality.—No death occurred.

(8) The Treatment adopted in every case was absolute rest, boiled rice diet, varied with mutton, eggs and extras, together with a mixture of atropine, digitalis and strychnine, and a daily phosphorus pill. The paralysed patients were massaged, and galvanism was applied to the atrophied muscles.

My best thanks are due to Lieutenant-Colonel J. T. Calvert, I.M.S., and Major Leonard Rogers, I.M.S., of the Medical College Hospital, to Major Hunter, I.M.S., of the Presidency Jail, and to Captains Megaw and Foster, of the General Hospital, for many kindnesses and much valued and instructive conversation during my visit to Calcutta in connexion with this outbreak; to Major Clements, R.A.M.C., Sanitary Officer, 9th Division, and to Major Donovan, I.M.S., of Madras, and to my colleague, Captain J. E. Hoar, R.A.M.C., at St. Thomas' Mount, and lastly to Dr. F. M. Gibson, Director of the King's Institute, Guindy, without whose kind help and invaluable assistance many of the bacteriological and cellular investigations undertaken would have been impossible.

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No therapeutic importance is now attached to the administration of this pill; it would not be given to any future case.