Thesis for the degree of Doctor of Medicine of Edinburgh University

on

Beri-Beri: A Clinical Study

by

Percy Athelstan Nightingale, Cape Colony

M.B. C.M. 1890

Etiology.

The origin and derivation of the term Beri-Beri is wrapped in an obscurity to which no author seems able to unravel, though many suggestions are made as to its probable source. These suggestions all tend to show that the term is the corrupted form of a native word referring to some chief symptom or characteristic of the disease.

Thus Bautius, writing in 1675, says that it is derived from the Hindustani Dhaye meaning a sheep, and points out that the peculiar gait of a Beri-Beri patient somewhat resembles that of a sheep; to my mind this is only worth mentioning
to discard at once, as the two styles of gait bear no resemblance to each other whatever.

Marshall in 1822 says Bhayree means weakness, and the repetition of the word, great or extreme weakness. A definite feeling of weakness or weariness is invariably complained of by nearly all patients, and is often the chief reason for their early seeking advice. I think that there is some show of reason for this derivation of the word.

Malcolmson in his prize essay in 1835 says that "Loon-Bhayree" and not "Bhayree-Bhayree" means great weakness, while Herklotz thinks that it is derived from "bhar-bari" meaning swelling; this latter is also a plausible theory, for in one of the clinical types of the disease swelling or oedema is one of the most prominent features, and would naturally attract the attention of even an unobservant native.

Barker derives it from the Arabic "bhar" shortness of breath, and "bhaari" a sailor, and hence holds that it means sailor's.
asthma; dyspnoea ought to be used instead of asthma, as the shortness of breath observed in certain stages of the disease bears not the slightest resemblance to asthma.

Secondly, B. Baill. means numbness and a rheumatism; the only objection there is to this being the difficulty of corrupting it into any word resembling Beri-Beri.

It is thus clear that various writers have attempted to derive the term from some word referring to some special symptom observed by them, and that each writer places a different value upon such a symptom. I believe that this seeming diversity of opinion has been caused by these writers observing the same disease at its different clinical stages, and of their being unaware that the prominent symptom of great weakness may to-morrow be changed into that of peculiar gait, which in its turn will give way to that of general oedema or swelling, pains of a rheumatic type, or alarming dyspnoea.

It is also equally plain that the term
Beri-Beri bears no scientific origin or value, and therefore I think that in the present state of our limited knowledge of the disease it would not be well to discard it as it does not bind us down to any definite opinion either pathologically or clinically.

Geographical distribution

The area of distribution of this disease is not only very wide, but seems to be continually increasing, owing to the power the disease has of being carried, usually by an affected ship's company, from one country to another, where it is liable to spread in an epidemic form provided certain conditions of soil, climate and mode of life are present.

From the writings of Chinese and Japanese medical men, of Bontius in Batavia in the early part of the seventeenth century, of Marshall in Guyana nearly two centuries later, and of Malcolmson in India, it is undoubtedly that the disease has its home in the East and that there it flourishes both in evidence and epidemical forms.
Whence it has spread over the greater part of the world, until today Europe alone is free from it—though imported isolated cases of it have been introduced into Greenwich and Newcastle, while lately it has appeared in the Dublin Asylum in a dangerously epidemic form.

Thus in Asia it is found in Japan, China, and neighbouring islands, in Siam, Annam, the Malacca Peninsula, Straits Settlement, and the Malaya Archipelago especially in Java, Sumatra and Borneo; in Burma, it has been noted as far inland as Mandalay. In India it is found in Assam, Calcutta, the Malabar Coast, the Northern Circars, the Coromandel Coast, Madras and Ceylon; it is prevalent among ship crews trading in the Bay of Bengal, Persian Gulf and adjacent coasts.

In Africa it is not unknown at many of the larger eastern ports and islands, such as Aden, Zanzibar, Reunion, Mauritius and Madagascar; its presence is suspected on the Congo, and in several parts of the West Coast, quite lately a report...
has reached me from Mr. P. O. Considine of Port Elizabeth, Cape Colony, that the disease has appeared in an epidemic form in that neighbourhood, having been introduced into the Colony in 1892 by a sailing ship.

In America it is said to occur among the fishermen of the northern coast, and it has been recognised at Cuba, Guadalupe, and other of the West Indian Islands; it has assumed an epidemic form in Panama, Brazil, French Guiana, and Paraguay; ships crews trading on the west coast of America suffer from it, and therefore it is probable that it already exists among many of the ports they visit.

In Australia itself, it seems only lately to have appeared, while it exists among the islands off its north-east coast, as well as among crews of ships trading in those parts—being merely an extension downwards of the disease from the islands of the Malayan waters.
Definition.

Beri-Beri is essentially a chronic disease of the nervous system, capable of prolonged latency and liable under certain conditions to acute exacerbations, is of the nature of an endemic-epidemic, multiple symmetrical peripheral neuritis, and is probably nearly allied to the malarial poison in that it, inter alia, is of helminth origin and has its habitat in tropical and sub-tropical countries, and

characterized by weakness, paralyses, and often paralysis of the muscles of both upper and lower extremities, anaesthesia and hyperaesthesia, especially of the lower extremities, skin of the abdomen and forearms; oedema, especially of the lower eyelids and over the inner surface of the tibiae; during some stage or other of the disease, a characteristic gait on a broad base, with high action of the heels and a jerking forward of the leg from muscular inco-ordination; serous
effusion into all the cavities of the body, to a more or less extent, especially into the pericardium and abdomen; atrophy of the muscles of the calf; superficial and deep reflexes diminished or totally lost; diminution or loss of sexual appetite; disturbances of the heart’s action; pains of a rheumatic type in both bones and muscles; loss of appetite; dry skin; secretions and excretions diminished; prolonged convalescence, and a marked liability to sudden and unexpected death from cardio-inhibitory paralysis, asphyxia from oedema of the lungs, paralysis of the respiratory muscles, hydro-pericardium, asthenia, or from a combination of these conditions.

Etiology.

The causes of Beri-Beri are still very obscure, though of late there has been considerable light thrown on them by means chiefly of clinical observations conducted in large hospitals, where patients admitted for other diseases and brought into contact with those suffering from Beri-Beri are
seen to contract the disease. For upwards of three years (1891 to 1894) while in the service of the Government of Johore (Malay Peninsula) I have had the opportunity of watching the disease in the Government Hospitals (the largest of which in Johore Bahru has a daily average of 270 in-patients), jails, and out-door dispensary practice, & it is from the experience thus gained that I have written this thesis.

It is usual to consider the causes of all diseases under two heads—(i) predisposing, by which as if it were, the soil is prepared and made ready for the germ, (ii) exciting, by which the germ or seed of the disease actually breaks out into action. In other words it is necessary that the vitality and resisting powers of the human frame be reduced to a certain low standard before the disease itself can affect the system.

Bentley has divided the causes of Beri-Beri into predisposing and exciting, and has elaborated them at considerable
length, winding up an enumeration of
the predisposing ones by laying great stress
on his opinion that anything that lowers the
vital and resisting powers of the system
predisposes to Beri-Beri."

It seems to me that this assertion is a medical
truism, and is no more applicable to Beri-Beri
than it is to any other disease. I even go
further and say that often predisposing
causes are quite unnecessary—in fact
that a man in a normal state of health
is liable to contract Beri-Beri if brought
into contact with Beri-Beri patients for a
certain length of time. As a proof of this
note the following:

Case 1

Awang, s/p 22, Malaya. About the middle
of August 1893 was passed by the Medical
Officer into the Johore Police Force and
was therefore presumably in perfect
health.

On the night of 25th November he com-
mitted a criminal assault, and being
caught in flagrante dicto received a
thrashing—not of a severe character.
On 26th November he was sentenced to twenty years penal servitude and was confined in the State Jail in a cell with two or three other prisoners, some of whom were suffering from the early symptoms of Beri-Beri. A week later he complained of general pains and weakness with inability to walk, so on 2nd December the leg cross-bar of his chains was recommended to be removed; this was done by the authorities only under protest as the prisoner was suspected of malingering—having on admission been in such good health.

On 15th December he was put on to light work within the jail; on 16th December he complained of pain in his knee joints and a general feeling of exhaustion and weakness. There was slight pitting over the tibial with numbness; Respiration 28 per minute; was again confined to his cell as the Jail Hospital was overcrowded with Chinese.

On 26th January 1874 dysphoeca set in with general oedema of the body; heart
sounds distant and feeble. He died the same evening with every symptom of effusion into the pericardium.

Case II

Geeow Ah Soh, aged 40, Chinese convict. Admitted into jail hospital for ulcer of the left leg on March 17th 1892, was otherwise in good health. Early in June he complained of general pains with weakness of the lower extremities, and had several attacks of feverishness; two or three weeks later oedema of the leg began, and he then rapidly developed all the signs and symptoms of the moist form of Beri-Beri. On July 17th 1892 he died with great precordial distress—evidently from effusion into the pericardium. While in hospital he was able to mix freely with the Beri-Beri cases who were then undergoing treatment.

Case III

Ho Khek How, aged 30, Chinese convict. Was admitted into the State Jail on October 24th 1892, for oedema serosi; he was a particularly well-nourished man.
powerful man: on November 24th he had oedema of the legs, with pains and general weakness; the usual symptoms of moist Beri-Beri rapidly supervened including oedema of the chest wall, and he died on November 25th 1892. While in hospital there were eight other cases of Beri-Beri under treatment, one of whom died on the same day as he did on the eighth day after admission.

Case IV

Tan Kim Eng. aged 57 Chinaman.
Admitted into the Joloore Pauper Hospital on August 16th 1892 for an ulcer of the second right toe; the ulcer being almost healed he was removed from the ulcer ward into Ward No. 1 on October 4th 1892 and by an oversight occupied a bed among the Beri-Beri patients. On October 26th he had some oedema of the legs with a characteristic brawny feeling of the calves, and on November 4th he was a pronounced case of moist Beri-Beri. The disease terminating fatally on December 13th 1892.
If by a "predisposing cause" is meant anything that reduces the system below par, so as to render it a suitable nidus for the Beri Beri germ or virus to thrive in, then it is of undoubted value to examine carefully such influences in order that they may be guarded against, and thus the rude prophylaxies against Beri Beri be arrived at.

Age. Beri Beri is essentially a disease of the prime of life, it being most commonly met with between the ages of 25 and 46. The following figures are based upon the examination of the ages of 300 cases of Beri Beri admitted into the Johore Bauer Hospital during 1892 and 1893.

1.88 per cent were between the ages of 20-20.
9.81 " " " " " " " " " 21-25
23.30 " " " " " " " " " 26-30
17.36 " " " " " " " " " 31-35
21.50 " " " " " " " " " 36-40
10.94 " " " " " " " " " 41-45
10.94 " " " " " " " " " 46-50
1.88 " " " " " " " " " 51-55
2.20 " " " " " " " " " 56-60
Sex.

Bentley argues that because Beri-Beri "is more common in men than in women; sex, therefore, is a predisposing cause," but produces no evidence to prove his. The figures I have just quoted under "age" were all labourers of the coolie class. with the exception of one. who was a prostitute, they were all imported from China by large estate owners for the labour market, but as no Chinese woman is allowed by law to leave China (with but rare exceptions) they had no wives with them, and therefore it is impossible to say that women of the coolie class, if exposed to the same conditions and influences as the men would or would not contract the disease.

Beri-Beri undoubtedly occurs to a considerable extent among Malay women, but it is difficult to get any statistics on the subject, as they object to being attended by a European, even at their own homes, and seldom or
never go to hospital. I have, however, seen a good many cases among them and have had reason to suspect many others—chiefly from the description of the illness by the husband, who applied to the dispensary for medicine, but did not wish for any personal attendance. I am strongly of the same opinion as Manson who says: *in estimating the proportion of the cases affected by any disease in Eastern countries, large allowance must be made for the social prejudices which keep females from attending hospitals and dispensaries.* This, together with the frequency with which large bodies of men, as compared to women, are crowded together in lodging-houses, factories, jails, ships and camps, probably account for a good deal of the apparent relative immunity of the female sex. Exposed to the same conditions as men, doubtless they will contract the disease quite as readily."

"Women during pregnancy and the puerperal state seem specially liable..."
to contract the disease, which may be due to their general condition being below par—especially after a post-partum haemorrhage, or to their close confinement in almost hermetically sealed rooms without ventilation, or to the virus entering the system from a lacerated perineum or cervix.

Simmons' case of a Japanese woman, quoted by Manson, being attacked in each of three successive pregnancies probably shows the latent or quiescent stage of power of this disease which was ready to burst into activity whenever there was an extra strain put upon the health of the woman. In passing it may be noted that in my experience puerperal dysentery is usually fatal in the first attack, and that a second attack is rarely recovered from.

From the geographical distribution of the disease it will be seen that few nationalities are exempt from it. Europeans living in the East are liable to contract it, though as for the most part they are of the well-to-do class their systems do not get below par, and comparatively few
suffer from it. It is, however, very prevalent among the Dutch in Java, and the Dutch troops employed in operations against the Achehese in North Sumatra. British troops have suffered from it in Burmah, and English settlers in North Borneo are not free from it. In the Straits Settlements and Malay Peninsula, the vast majority of cases occur among the Chinese—i.e., the labouring class, who live frugally and work hard.

This is undoubtedly an important factor in rendering one individual more susceptible to Beri-Beri than another. Manson very forcibly says that the liability of sailors to the disease is owing to the concentration of poisons in the damp, hot forecastle into which they huddle during bad or cold weather; thus producing an artificial incubator for the propagation of the germ. My experience seems to show that those employed in cultivating the ground, especially when preparing hitherto virgin soil for the planting of gambier, coffee, or similar tropical products, fall ready victims to
the disease, as the following figures show.

Table showing the occupations per cent
of Beri-Beri cases admitted into the
Johore Hospital.

<table>
<thead>
<tr>
<th>Occupation</th>
<th>1892</th>
<th>1893</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gambier coolies</td>
<td>70.96</td>
<td>61.26</td>
</tr>
<tr>
<td>Coffee, tea, &amp; earth coolies</td>
<td>10.75</td>
<td>9.15</td>
</tr>
<tr>
<td>Other occupations</td>
<td>18.29</td>
<td>29.59</td>
</tr>
</tbody>
</table>

Now, although the greater number of Chinese in Johore are employed in cultivating gambier (which needs the constant breaking up of fresh soil and the extension of the estates) it follows that the majority attacked would necessarily be gambier coolies but taking into account the very high per centage, out of all proportion to those employed at other occupations, of that class who applied for treatment, I think it undoubtedly proves that the occupation of breaking up the virgin soil of a tropical country renders those so employed specially
liable to contract Beri-Beri.

Impure water.

There is no evidence that impure water in itself produces Beri-Beri, but nevertheless I think it possible that water passing through dense jungle over decaying masses of organic material may not be altogether drunk with impunity, that it is commonly so used by estate coolies may determine to some degree their susceptibility to the disease.

Damp and Moisture.

All observers agree that the number of admissions to hospital for Beri-Beri invariably increase when the rainfall has been excessive. In the tropics the heat of the sun is very powerful within a few hours after the rain has ceased, which causes a damp mist to rise from the ground and I think it is highly probable that in this moist may be contained the germ of the disease itself.

Table showing the monthly admissions and deaths from Beri-Beri, &c.
The Johore Government Hospital from 1888 to 1890, with rainfall.

<table>
<thead>
<tr>
<th></th>
<th>1888</th>
<th>1889</th>
<th>1890</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Admitted</td>
<td>Died</td>
<td>Admitted</td>
</tr>
<tr>
<td>January</td>
<td>9</td>
<td>2.76</td>
<td>9</td>
</tr>
<tr>
<td>February</td>
<td>6</td>
<td>3.15</td>
<td>2</td>
</tr>
<tr>
<td>March</td>
<td>3</td>
<td>7.96</td>
<td>3</td>
</tr>
<tr>
<td>April</td>
<td>3</td>
<td>11.17</td>
<td>2</td>
</tr>
<tr>
<td>May</td>
<td>12</td>
<td>9.59</td>
<td>1</td>
</tr>
<tr>
<td>June</td>
<td>9</td>
<td>5.13</td>
<td>1</td>
</tr>
<tr>
<td>July</td>
<td>9</td>
<td>4.39</td>
<td>1</td>
</tr>
<tr>
<td>August</td>
<td>3</td>
<td>8.58</td>
<td>1</td>
</tr>
<tr>
<td>September</td>
<td>1</td>
<td>8.61</td>
<td>1</td>
</tr>
<tr>
<td>October</td>
<td>1</td>
<td>8.86</td>
<td>1</td>
</tr>
<tr>
<td>November</td>
<td>1</td>
<td>9.42</td>
<td>2</td>
</tr>
<tr>
<td>December</td>
<td>1</td>
<td>9.41</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>57</td>
<td>25.922</td>
<td>22</td>
</tr>
<tr>
<td>Month</td>
<td>1891</td>
<td>1892</td>
<td>1893</td>
</tr>
<tr>
<td>------------</td>
<td>------</td>
<td>------</td>
<td>------</td>
</tr>
<tr>
<td>Admitted</td>
<td>Died</td>
<td>Rainfall</td>
<td>Admitted</td>
</tr>
<tr>
<td>January</td>
<td>3</td>
<td>3.02</td>
<td>26</td>
</tr>
<tr>
<td>February</td>
<td>3</td>
<td>3.53</td>
<td>9</td>
</tr>
<tr>
<td>March</td>
<td>18</td>
<td>4.44</td>
<td>11</td>
</tr>
<tr>
<td>April</td>
<td>12</td>
<td>2.46</td>
<td>6</td>
</tr>
<tr>
<td>May</td>
<td>8</td>
<td>4.73</td>
<td>8</td>
</tr>
<tr>
<td>June</td>
<td>6</td>
<td>1.28</td>
<td>11</td>
</tr>
<tr>
<td>July</td>
<td>11</td>
<td>7.15</td>
<td>9</td>
</tr>
<tr>
<td>August</td>
<td>8</td>
<td>3.92</td>
<td>7</td>
</tr>
<tr>
<td>September</td>
<td>7</td>
<td>2.75</td>
<td>6</td>
</tr>
<tr>
<td>October</td>
<td>8</td>
<td>1.55</td>
<td>8</td>
</tr>
<tr>
<td>November</td>
<td>15</td>
<td>10.28</td>
<td>7</td>
</tr>
<tr>
<td>December</td>
<td>5</td>
<td>5.56</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>102</td>
<td>23</td>
<td>87.53</td>
</tr>
</tbody>
</table>
Doctor Rowell, in his report to the Government of the Straits Settlements quoted by Bentley, undoubtedly proves that the increased dampness of the soil in the Singapore Jail was followed by an epidemic of Beri-Beri among the convicts, and that when the jail grounds were more thoroughly drained and rendered dryer, by the permanent opening of the sluice gates at Raffles Institution Bridge, there was a marked decrease in the number of Beri-Beri cases.

Diet.

In that defective diet is a prime factor in the reduction of the health below a given standard it is undoubtedly one of the most important of the so-called predisposing causes, but on the other hand all the evidence tends to show that diet, in itself, is not the actual or exciting cause. Dr. Takaki of Japan, undoubtedly caused Beri-Beri to practically cease to exist in the Japanese Navy, by greatly
increasing the nitrogenous element in the dietary, but then he also improved the hygienic condition of the ships, and generally raised the health standard of the men to above par.

Rice, the staple food of the East, good or bad, cannot produce Beri-Beri, for if it did no natives, and but few Europeans in the tropics would be exempt from the disease; in times of famine or scarcity of rice (such as when the French blockaded Bangkok in the summer of 1873) the disease would be specially virulent, but this has not been found to be the case. Doctor Neil Macleod in an interesting article entitled "Can Beri-Beri be caused by food supplies from countries where Beri-Beri is endemic?" gives details of how four cases of the disease occurred on board a sailing ship from New York to Shanghai, and seeks to show that they were caused by certain articles of diet—sago, tapioca and arrowroot; but the ship itself may have been contaminated with the disease before
lying dormant; in a previous voyage to Australia, or it may have reached the vessel when in the neighbourhood of the Solomon Island, air-borne, or their Chinese servants may have infected them without they themselves showing or complaining of any symptoms of the disease.

The ordinary diet of a Chinese cooly usually consists of rice, fresh and salted vegetables, and salted or fresh fish, daily, with fresh fish, pork and pickles about twice a month, his drink is plain water. In hospitals and jails the diet is infinitely superior in every way to what he has been accustomed to outside, and yet one sees numerous cases of Beri-Beri contracted with these buildings especially within the jail where the diet is better than in the pauper hospitals (see adjoining Diet Tables).
**Diet Table in the Johore State Jail**
Chinese only.

<table>
<thead>
<tr>
<th>Articles</th>
<th>Ordinary Diet</th>
<th>Diet for short-sentenced prisoners</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Quantity/Frequency</td>
<td></td>
</tr>
<tr>
<td>Rice</td>
<td>1.8 Daily</td>
<td>Rice 1.8 Daily</td>
</tr>
<tr>
<td>Fresh Meat</td>
<td>12 Weekly</td>
<td>Vegetable 4 Weekly</td>
</tr>
<tr>
<td>Large Fish</td>
<td>10 Weekly</td>
<td>Curry Sauce 1 Weekly</td>
</tr>
<tr>
<td>Small Fish</td>
<td>30 Weekly</td>
<td>Salt 1 Once</td>
</tr>
<tr>
<td>Salt</td>
<td>2.1 Once</td>
<td>Vegetable 9 Daily</td>
</tr>
<tr>
<td>Vegetables</td>
<td>1.8 Daily</td>
<td>Beans 4 Daily</td>
</tr>
<tr>
<td>Beans</td>
<td>1.8 Daily</td>
<td>Curry Stuff 1 Daily</td>
</tr>
<tr>
<td>Curry Stuff</td>
<td>1 Once</td>
<td>Salt Pepper 1 Once</td>
</tr>
<tr>
<td>Salt</td>
<td>1 Once</td>
<td>Vegetable 9 Daily</td>
</tr>
<tr>
<td>Coconuts</td>
<td>1 Coconuts</td>
<td>Vegetable 9 Daily</td>
</tr>
</tbody>
</table>

*1 Kahi = 1.5 lb; 1 Kahi = 16 Takils*
### Chinese Diet in Johore Government Hospital

<table>
<thead>
<tr>
<th>Articles</th>
<th>Units</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>1</td>
<td>Daily</td>
</tr>
<tr>
<td>Vegetables</td>
<td>4½</td>
<td></td>
</tr>
<tr>
<td>Curry Stuff</td>
<td>3½</td>
<td></td>
</tr>
<tr>
<td>Idly</td>
<td>3½</td>
<td></td>
</tr>
<tr>
<td>Coconut Oil</td>
<td>3½</td>
<td></td>
</tr>
<tr>
<td>Beans</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Meat</td>
<td>4½</td>
<td>Twice</td>
</tr>
<tr>
<td>Fresh Fish</td>
<td>3</td>
<td>Twice</td>
</tr>
<tr>
<td>Dry</td>
<td>3</td>
<td>Twice</td>
</tr>
</tbody>
</table>

Extra: Bread, eggs, milk, sago, port-wine and gin - as ordered.

**Scorbutus**

Scorbutus, and the causes leading to it, have been credited by some debarring the forerunners of Beri-Beri, but I have seen no detailed evidence to support this; I have, however, seen a spongy condition of the gums in some cases, though I did not attribute much value to them. In December 1893 an epidemic of Beri-Beri broke out among the convicts in the Johore jail, which caused the jail hospital to be over-crowded and
to become in an insanitary condition, and it was then that there occurred the two following cases, in both of which I looked upon the condition as merely associated with the disease as an account of the insanitary arrangements with which the prisoners were surrounded and not as any factor connected with the causation of the disease itself; it will be noted that in the second case the scurvy-like condition was noticed before the symptoms of Beri-Beri, but as the patient was already in a debilitated condition and was exposed at the same time to conditions which would produce both beri-beri and scurvy, I looked upon it that the former condition would naturally show itself before the latter on account of the pathology of the two diseases, and held that it was partly justified in this view in that when the sanitary condition and diet of the hospital was improved the scurvy-like symptoms among the patients ceased while the number of those who...
contracted Beri-Beri were only lessened.

Case V

Jau ab Jok, Chinese convict, was admitted into the Jail Hospital for Beri-Beri on November 18th, 1873.

December 24th, gum of the upper jaw spongy and bleeding, when an offensive discharge.

December 25th, great restlessness, and prostration, skin dry, respirations hurried; great quantities of bloody mucous, with the most penetrating smell, pouring out of his mouth; tongue, and gums of both jaws, swollen soft and bleeding; condition of patient appears very serious.

December 26th, pulse 96, respirations 36; large purpuric patches on trunk, patient unable to speak; faint mitral systolic cardiac bruit; face swollen and bloated, lips swollen and spongy.

December 27th, easier, though face more swollen; discharge less offensive; not so much bleeding.

December 28th, much improved mucous
discharge less, little or no bleeding from the gums: teeth all very loose.

December 31st: Improvement continues.

January 1st: Discharged from the Hospital to be employed on light work within the Jail grounds.

Case VI

Lee Jeow Kay, Chinese convict, was admitted into the Jail Hospital on November 25th, 1893. for debility.

December 20th: Both lips swollen, painful and spongy, feeling like lung tissue; lower lip has tendency to bleed and crack at the angles of the mouth.

December 28th: Gums bleeding freely, inside of cheek ulcerating.

January 3rd, 1894, Leukotic symptoms better; papillary reflexes somewhat diminished.

January 20th: Pains and numbness in knees and calves; pain and tightness over abdomen; walks with locked knee joints.

February 1st: Increased numbness with cardiac distress; characteristic brown.
based gait of Beri-Beri, with other symptoms of the disease.

Exposure to cold and inclement weather. This, in the climate of the Malay Peninsula, and surrounding countries and islands, is equivalent to being exposed to the influence of malaria, and so will be discussed later on; what I wish to point out now is that a single exposure, such as a very large number of patients ascribe their illness to, can not in itself produce Beri Beri, for if it could then the disease can have practically no incubation period, and nerves can become extensively affected within the course of a few hours. The following is a typical history which many patients give.

Case III

Sing Tong Keng, aged 42, was admitted to Hospital for Beri-Beri on November 12th, 1873.

His previous history was unimportant.
except that he had lived in Johore for the last 18 years and had suffered a good deal from malarial fever.

About 20 days ago, when apparently in the best of health, he went to a Chinese theatre, and got very wet (the theatres are all out-of-doors); on returning home he felt slightly unwell, but slept soundly that night; on waking next morning he found the dorsal surfaces of both feet to be oedematous, and the soles of both feet and external malleolus to be "numb"; the oedema and numbness gradually increased till he went to Hospital, by which time he had all the symptoms of a Beri-Beri case. He remained under treatment for four and a half months and was then discharged apparently quite well.

I believe that in this case, and in all similar ones, the patient was, unknown to himself, already suffering from Beri-Beri, and that the exposure to cold merely brought on an acute exacerbation of the
disease, producing symptoms or effects to which the patient's attention was forcibly drawn. My opinion is strengthened by the following case, which shows that an attack of Beri-Beri was plainly brought about by exposure to cold and wet in a man who had had an attack of the disease some four and a half years previously and in whom it was therefore lying latent.

Case VIII

Seong Ah Kong, Oct. 23, a shop cooly, was admitted into the Johore Hospital for Beri-Beri on December 6th 1893.

He states that he came from China ten years ago, and worked in Johore for three years as a shop cooly, when he returned to China for five months. Eighteen months after coming to Johore he was attacked with general oedema of the body, numbness of the extremities, and difficulty in walking for which he spent six months in China and then returned to Johore in apparent
good health.

Twelve days ago he slept out in the streets, and on rising next morning found his face swollen; this was followed by general oedema of the body, especially of the legs, with numbness extending from the feet upwards. On admission to hospital patient stated that his condition was practically the same as when he went to bed the last time.

Patient presented the ordinary symptoms of a Beri-Beri case, with general oedema and characteristic gait; he remained in hospital until January 3rd, 1894, and was then discharged at his own request as he said he felt quite fit for work again.

Gonococcal Disease.

Persons suffering from gonococcal disease in the East often get extremely debilitated and pull down, and thus fall an easy prey to malaria or any other disease. If those influenced thereby may be exposed, even the hardy Belgian
is not an exception to this rule, for a soft bladder and pair of buboes often reduce him to a most asthenic condition, and it is under such circumstances that he may contract Beri-Beri, or the disease if already in him may undergo an acute exacerbation. It is thus a very common thing for a Chinaman to attribute his attack of Beri-Beri to a gonorrhoea or chancre; he also often seeks admission into hospital for gonorrhoea, and for what is taken at the time to be gonorrhoeal or rheumatism, but which afterwards appears to have been the early rheumatic pains of Beri-Beri. The following cases illustrate this.

Case IX

Gop Al Lam, aged 2, a gambier coolie, was admitted into the Johore Hospital suffering from his third attack of Beri-Beri on November 7th, 1873.

He states that for the last 24 years he has worked in Johore, either in the
saw mills or on a gambier plantation; with the exception of several attacks of fever he has enjoyed good health. About seven years ago he contracted a chancre and bubo; a week later his whole body began to swell, including the face, chest, penis, and scrotum; this was followed by numbness of the extremities and great difficulty in breathing, for which he was treated in Hospital for some three months. He attributes his first attack to “woman’s wind,” the second to exposure to wet, and the third to malarial fever. He was discharged from Hospital on February 8th, 1894.

Case X

Jin Hock Chek, aged 38, a gambier coolie, was admitted to Hospital for gonorrhoea and “gonorrhoeal pneumonia” on December 28th, 1891.

In March 1892 he had oedema of the legs, and then gradually showed all the other symptoms of Beli-Beiri. September 16th, 1892 died suddenly with great cardiac distress.
Case XI

Law Him Long, aged 44, was admitted to Hospital on April 22nd with gonorrhoeal rheumatism, giving a clear history of previous gonorrhoea; he was a heavy, phlegmatic, but particularly strongly built man.

In June he showed symptoms of Beri-Beri, which progressed until November 2nd when he died very suddenly and unexpectedly.

Case XII

Ganu Ah Bhow, aged 20, a saw mill cooly was admitted to Hospital for Beri-Beri on March 15th 1894.

He says that two months ago he had fever for two or three days; one month ago he felt pain and numbness in his calves which was followed by oedema of the legs and face. He ascribes his illness as entirely due to gonorrhoea.

On admission he had loss of patellar reflexes, numbness of hands and feet, with oedema over the tibia, marked
congestion of the fossae extending on to the
hard palate, and a distressed anxious look;
the heart sounds were small and distant,
but there was bruit, when walking the left
leg seems much exerted, while the foot
"hangs" on the ground more than the right;
on grasping the calves great pain is felt.
The voice was slightly husky.

Mental Depression

I am inclined to rank this somewhat
high among the so called predisposing
causes, for I think that a man suffering
from a shock to the nervous system, and
in a depressed hopeless state of mind, if exposed
to the Berti-Berti influence, is liable to con-
tract the disease in a shorter time than
if he had no mental shock and yet was
suffering from some ordinary disease,
such as phthisis or an ulcer. Case 1
which I have already quoted, is an in-
stance; it is that of a young Malay in
normal health who was caught com-
mitting a criminal act and promptly
controlled is what was practically
imprisonment for life. For the Malays are not a long-lived race, and his offence was of such a nature that no hope of reprieve was to be entertained. Even to his oblid nature the mental shock must have been considerable, for within a week of being exposed to the Peri-Peri influence he began to show the early symptoms of the disease, and within two months he was dead.

The following is an almost parallel case which is of considerable value as the various dates are accurately fixed — which is as a rule no easy thing to do when dealing with a native.

Case XIII

Reen al Leok, aged 22, a gambier-pong was admitted into the Johore Government Hospital on January 25th 1894 suffering from extensive burns of both forearms. The history was that ten days previously, while quarrelling with another coolie he had thrown him into a cauldron
of boiling gambier, and then seeing his danger he had plunged both his arms into the cauldron to try and extract him; the cooly who was thrown into the gambier died, and Abraham was apprehended by the Police on a charge of murder and sent into Hospital for treatment. In spite of the serious nature of the burns (the entire skin of both forearms from the tips of the fingers to just above the elbows peeling) he was discharged from Hospital in good health on February 16th, and was locked up by the Police in the Police Cells—which were in an insanitary condition and in which a good many prisoners had already contracted Beri-Beri. He was then fully aware that he was to be tried for murder and that by the Mohammedan law he would most certainly be sentenced to death.

On March 1st (fifteen days later) he was sent back from the Police Cells to the General Hospital suffering from Beri-Beri in an advanced stage. He said that he was never ill before.
got burnt and that he felt quite well and strong when he left the Hospital. He states that on the sixth morning in the Police Bells he had "fever" (as distinguished from "ague") which lasted four days, on the seventh morning his legs felt numb and weak, and from that time he got gradually worse so that he had to be brought back to the Hospital in a ricksha.

March 7th. Walks very slowly with the characteristic Beri-Beri gait, and has great difficulty in turning; calf muscles are flabby and painful on the slightest pressure; papillary reflexes are totally lost; the sanaeae are congested and the voice is shrill; face is very puffy, especially the lower eyelids; looks very ill and distressed.

March 9th. Cannot walk or even stand; abdomen painful to touch; no oedema of legs.

March 10th. Reaction of degeneration well marked.

March 14th. Slight oedema of chest with increased dull area of heart. Other symptoms
more pronounced.

One of the theories which gained much credence at one time was that Beri-Beri was caused by the parasite Anellylostomum dewezae - a view strongly upheld by Hynsey of Ceylon and Earl of Sumatra.

Manson deals with this theory in a way that shows it is quite untenable; why he pertinently asks, "If the anellylostomum produces Beri-Beri in Ceylon and Sumatra do we not hear of Beri-Beri in Egypt, where the anellylostomum is so common? Why is it not reported as one of the features of the many epidemics of anellylostomiasis occurring in Europe," and why is it not invariably found in the stools of patients, and in the intestines postmortem?

I believe, with Manson, that Beri-Beri has nothing whatsoever to do with anellylostomum or any other intestinal parasite, but that when the two are found together they are merely concomitants and that they bear no relation to each other, these parasites, however,
often produce anaemia, sometimes so profound as to almost simulate the pernicious form, and hence may have arisen the idea that Beri-Beri was an anaemic disease, that is, dependent upon anaemia for its origin.

"Beri-Beri does not depend on Anaemia.

Peckharing and Iundle have come to the above conclusion after a special investigation, basing it chiefly on the results of their microscopic work,² but add ³ "There will be no longer remain any doubt as to the correctness of our statement, that in Beri-Beri we can have very marked anaemia, but that anaemia is by no means a constant symptom of Beri-Beri; I had already come to this conclusion before knowing of Peckharing and Iundle's views, solely upon clinical grounds (in fact at one time I thought that the great majority of Beri-Beri patients had nearly white conjunctiva and anaemic cardiac bricks), and in
This my clinical observations differ somewhat from those of Bentley, who states that in the great majority of his cases anæmia was entirely absent. Of 38 cases of Beri-Beri examined one morning in the General Hospital, all of whom had been undergoing treatment for periods varying from four months to one week, I found that nine were anæmic in various degree, but none of them were of the pernicious anaemia type which is sometimes to be met with, though I think it is less common in the Hardy Chinese than in the “leatherback” family.
Obscure as the etiology of Beri-Beri is, the weight of clinical evidence, which is almost daily increasing, seems to point to a very close relationship between it and malarial fever, though no one seems able to define the precise connection; is it an attenuated or concentrated form of malaria, is it a ptomaine poison, is it a bacteria where is it generated, and how does it enter the body? - are still unsettled questions. Much work has been done on the subject, but the results obtained by various observers have seldom agreed, except that they are all convinced that the vein of Beri-Beri is some form of the malarial.

Further on, however, I hope to show that malarial peripheral neuritis in its characteristic form, although closely resembling Beri-Beri, still shows distinct features which mark it out as a different disease.

Bekebling and windler have isolated rod-like and granule-like bacteria from the blood of Beri Beri patients, with which they inoculated culture
butes, and from the cultivations thus obtained.

They inoculated rabbits and dogs, and produced

in them generalized adena and peripheral

neuritis, and hence they conclude that a

micrococcus obtained from the blood of Beri-

Beri patients is the cause of the disease.

Further experiments have convinced them

that "the germ is generated in the soil and

rising into the air as dust, it is inhaled,

entering the human body by the lungs."

The geographical distribution of the two

diseases—Beri-Beri and malarial fever—

are practically identical, both depending

for their production, if not existence,

upon moisture, heat, and decomposing

organic matter. Rowell of Singapore,

quoted by Bentley, gives an account of

the outbreak of an epidemic of Beri-Beri

in the Singapore prison which was caused

by the porous subsail being saturated

with foul water from the blocked sluice-
gates of the canal, and adds that the site

of the prison was on a former mangrove

swamp.
I have already given a table showing that of the Beri-Beri patients admitted into the Johore Hospital in 1892, and 1893 no less than 70 per cent and 61 per cent respectively were gambier coolies; the following figures show the striking resemblance between the occupations of Beri-Beri and Fever cases.

<table>
<thead>
<tr>
<th>Table showing the occupations per cent of Fever cases admitted into the Johore Hospital</th>
<th>1892.</th>
<th>1893.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gambier Coolies</td>
<td>69. 35.</td>
<td>76. 96.</td>
</tr>
<tr>
<td>Coffee, tea, earth coolies</td>
<td>3. 89.</td>
<td>2. 60.</td>
</tr>
<tr>
<td>Other occupations</td>
<td>26. 76.</td>
<td>20. 45.</td>
</tr>
</tbody>
</table>

In Johore, gambier estates are almost invariably situated along the low banks of the large rivers for the convenience of the water carriage. What ground is not under actual cultivation usually consists of dense virgin jungle with an immense amount of decaying organic matter. The rainfall is very heavy, drainage is a matter of secondary
importance to the Chinamen, while the thermometer daily averages 150°F in the sun and about 85°F in the shade. Here then are all the elements required for the production of malarial fever, and, as the above figures show, Beri-Beri is also present to an alarming degree under exactly the same conditions as malarial fever, painting I think, somewhat plainly to the close relationship between the two.

A somewhat curious fact is to be noted about the Johore district of Bahau Pahat—which for the most part is situated on low-lying ground some three miles from a large river and surrounded by hills which run up to 1200 feet high; the ground is extensively cultivated with coffee, gambier and pepper, by Tamil, Javanese and Chinese coolies, is surrounded on all sides by dense jungle and has, a rain-fall of about 150 inches a year, while the thermometer averages 88° to 93°F in the shade. Malarial fever is exceedingly
common in the district, and assumes an almost malignant type, causing a heavy death rate (especially among the newly arrived labor coolies), so that "Baba Pahat fever" has a special and very unenviable reputation out here, and yet Beri-Beri (even among Chinese coolies who have been for a long time on the estates and have suffered much from fever) is comparatively rare. Considering, therefore, that Beri-Beri is but seldom met with in these estates where malaria is very prevalent and severe, it seems to me that it impossible to assume that Beri-Beri may be an attenuated form or offshoot of malarial fever.

It is well-known that once malarial fever has entered into the system it is rarely, if ever, entirely eliminated; it is liable to crop up again years after, when the patient is in a cold and healthy climate, without fresh exposure to the source of infection, when-
ever his vital powers fall below par—believe the same is equally true of Beri-Beri, and have founded my belief upon the histories given by numerous patients who have had repeated attacks of the disease during a considerable number of years, from careful examination of such patients upon their admission to and discharge from hospital, and from what I consider as the pathology of the disease.

"Feverishness is a prominent symptom of both diseases; of malaria it is of course, of the so-called malarial type, with the usual rigor followed by the cold and hot stages; I believe, also, that at times the fever of Beri-Beri is identical with this, though in the majority of cases it is of the continued and not of the intermittent type. At the same time a considerable number of patients are admitted into hospital for 'fever' of the quotidian or tertian type, are discharged cured, and are re-admitted into hospital a few weeks.
afterwards with marked symptoms of Beri-Beri, the question then arises, was their original "fever" for which they were treated the fever of malaria or Beri-Beri, and if the former did they then contract Beri-Beri within the hospital? One of the main difficulties in answering this question is the impossibility to rely upon the statements made by the patient—a difficulty which I shall enter more fully into when I come to deal with the symptoms of the disease. I cannot, however, but think that in many of such cases the fever which seemed identical with that of malaria was in reality that of Beri-Beri.

The early symptoms, too, of both diseases are somewhat similar, in that the "fever" of both is followed, always in the one and very often in the other, by shooting pains in the joints, especially those of the lower extremity, slight pitting over the tabic and dorsa of the feet, and an indefinite tingling or numbness in the soles
of the feet and calves of the legs—so that the
differential diagnosis at this stage is
often a matter of some difficulty.

For it is uncommon to find cases of
mixed infection, i.e. malarial neuritis
and Beri-Beri. With these, we shall deal
in considering the differential diagnosis.

Again, in both, the spleen is almost al-
ways found to be enlarged and often ac-
deerly hard, as if from hypertrophy and
hyperplasia of the fibrous trabeculae though
it is possible that this may be due solely
to the malarial miasma.

It is a noteworthy fact that during their
residence in hospital many Beri-Beri cases
have attacks of true malarial fever which
seem to have a more disastrous effect
upon them than upon any other class
of patient—and much so in fact that after
a heavy rainfall I always watch with
special care to see that no patient in
the Beri-Beri ward has a rise of
temperature.
The conclusions of Petechiaring and Windle, quoted by Manson, that the germs of Beri-Beri are generated in the soil and enter the body by inhalation are almost equally true of malaria; I see, however, no reason to even doubt that as the malarial germ may also be taken into the body by means of water why Beri-Beri should not be also, in as much as both are to be found on the same estates where the coolies affected by both are exposed to the same climate influences, share the same broken down shanty for a house, eat of the same food, and drink of the same water from a running stream or shallow well.

Manson states that Beri-Beri is a house or town disease ---- it clings to inhabited places ---- and in this respect it differs from malarial disease, which is always more acute and more deadly on the outskirts of towns and in the open country than in the large centres of population; my experience agrees with this. No doubt that Beri-Beri, like
malaria, is essentially a disease of the swampy, lowlying, country, is found in the delta of rivers, along canals and in places built on sites which once were marshy—while in many sparsely populated gambier estates it is especially rife, but it is true that at times it abounds in hospitals, prisons, and police cells, and I think that it is undoubtedly proved that in such cases it is introduced within the building by some patient or patients, that in these buildings insanitary conditions exist, such as overcrowding, a want of free circulation of the air, from the necessary surrounding high walls, and a general depression of the mental faculties of the inmates, and that by remedying these defects the epidemic course of the disease is much stayed, if not entirely cut short.

In this respect, Manson is correct in calling Beri-Beri a house or town disease, for those who have had to deal much with it know only too well.
how extremely difficult it is to eradicate the affection from the infected locality.

In the manner in which Beri-Beri clings to the forecastles of ships the difference is marked between it and malaria. So long as the infected portions of the ship are not disinfected, cases continue to arise amongst those members of the crew who inhabit these areas. Yet all cases be cleared out, and the forecastles, or whatever portion of the ship it may be, be thoroughly disinfected, no fresh cases of Beri-Beri will originate there, so long as there is no chance of reinfection by the introduction of a fresh case from outside the ship, whereas, meanwhile, many of the crew may be suffering from repeated attacks of Malarial fever.

To sleep on the ground in malarious districts is a well-known danger, and is seldom practised by anyone except Chinese coolies whose houses, as a rule, are not built on stakes or piles, such as the Malajays and Javanese use, and whose beds are mere low couches; many a Beri-Beri patient has ascribed his illness to the effect of sleeping out on
the ground at night—when he got chilled by the dew.

Is Beri-Beri contagious or infectious?
The majority of observers hold that it is the latter but not the former, though they differ as to the exact method of communicability. Both Manson and Bentley² lay some stress on the fact that the attendants on Beri-Beri cases do not contract the disease as a proof that it is not contagious, and add as a further proof that the disease does not spread among the other patients in the hospital; I cannot agree with them about the disease not spreading among the other patients, while I think that my speaking about the attendants not contracting it they overlook the fact that there is more than one degree of contagiousness.

Beri-Beri is certainly not contagious in the same degree as small-pox, scarlet fever and others of the scabulosaætae are, which are able to infect persons by a single exposure to the virus; nevertheless I think that evidence is not altogether wanting to show that at times
its virus can be transmitted by contagion or personal infection. The following case occurred in the Johore Gaol Hospital at a time when it was a good sanitary condition and not overcrowded, and points, I think, somewhat conclusively to this one case, at least, contracting the disease by direct contact with a Beri-Beri patient.

Case XIV

Han Ali Sah, a Chinese gambier cooly, was admitted in the Gaol Hospital in June 3rd, 1892. Suffering from the tubercular ulcersous type of leprosy, he was a powerful built man. He occupied a cot alongside an old standing case of Beri-Beri who was undergoing treatment for his third attack, and who from paresis of the legs was unable to walk, and so was attended to by the leper; these two became great friends and used to spend most of their time together playing a Chinese game with little bits of stick upon the floor of the ward. Two months after his admission the lepercontracted Beri-Beri from
which he died on October 24th, 1892.

The reason why the coolie attendants on Beri-Beri patients do not contract the disease is, I think, because their health is up to a certain standard, they are constantly out in the fresh air and thus are not continually inhaling the same poisoned atmosphere, and even when they are in the ward they do not pay much attention to the patients and the medical officers back is turned; it is true these attendants sleep in the Beri-Beri ward, but their cots are placed as far away as possible from the patients, and the ventilation is very good—nevertheless I consider that even thus they are exposed to a certain amount of risk from infection, and I would certainly prefer that no one occupied the Beri-Beri ward, even for part of a day, except those actually suffering from the disease.

I might quote a considerable number of cases of patients who, admitted for
other diseases have contracted Beri Beri within the Hospital, but consider the follow-
ing general statement of more value as proving its infectiousness. When I first came to Johore in 1891, I found Beri-Beri cases mixing freely with others in a large ward containing some sixty patients in all. Knowing nothing about the disease and finding no help in the general text-books, I naturally paid some special attention to it—though from press of other work could not devote as much time to it as the subject deserved. Early in 1892 I became convinced that I had seen other patients in the ward contract it, and so placed all the Beri-Beri pa-
tients together at one end of the ward, while at the other I only kept what might be called a "floating population" of patients suffering from malarial fever, phthisis, venereal disease etc.—in contradistinction to surgical cases and others who would probably be con-
fined to their cots for some time; at the same time I kept the ward constantly
starved and white-washed. By these means I was enabled to check somewhat the spread of the disease.

However early in 1894 the Hospital accommodation became too small for the number of patients, which resulted in the Beri-Beri ward being overcrowded to the extent of 75 or 80; at the same time the starving and whitewashing was not carried out as formerly.

The result was that in March 1894 Beri-Beri in an epidemic form existed in the ward, and a considerable number of cases undoubtedly contracted the disease. The ward was therefore devoted strictly to Beri-Beri, all other cases were promptly discharged, the sanitary arrangements were attended to, and the diet slightly improved.

Here I should like to add that in October 1892 I recommended the Governor to build a separate ward in the Goal for Beri-Beri. As I was convinced from what I had seen in the Goal Hospital that the disease under certain
conditions was highly infectious, if not contagious.

The following are brief notes of a few cases who contracted Beri-Beri within the Hospital:

Case XV

G. Win Heng Soon, aged 47, was admitted into Hospital on July 30th 1893 for syphilitic rheumatism; he had pains in all the joints of the body, but none in the calves of the legs, there was a history of recent syphilis, unproved considerably under antisyphilitic treatment.

August 19th complained of numbness of the lower extremities with pain in the calves on pressure and oedema over the tibia. From this date the disease steadily progressed, until by October 1st he had paresis of the lower extremities with inability to walk, wrist-drop and incoordination of the muscles of the upper extremities.

November 24th, diarrhoea set in, and he died 24 hours later.
Case XVI

Naah ah ge, at 49, a particularly muscular and strongly built man was admitted on April 20th 1892 for aque (quotidien); his cot had another cot (the occupant of which was always changing) and the passage for a door between it and the section of the ward devoted to Beri-Beri.

In May he developed paralysis agitans from which he was slowly recovering when in October he showed symptoms of Beri-Beri; advanced general oedema and with great precordial distress set in. He finally died completely waterlogged on November 18th 1892.

Case XVII

Chin Tob Hee, at 20, Chinese gambier cooly, admitted into hospital for dysentery and bronchitis on December 26th 1893. Was sent to the Beri-Beri ward.

On January 3rd 1894, he was anaemic, face puffy, slight oedema of chest and lower extremities; numbness over the legs extending up to the lower third of
Night: pain on pressure in calves, patella reflexes present, no cardiac lesion.

January 13th: Oedema increasing.

January 25th: Ascites.

February 2nd: Patella reflexes absent; tendency to ankle drop; pain in calves on pressure but patient says this is better; atrophy of calf muscles; no congestion of fauces or characteristic gait, heart rapid and small but no bruits; ascites increasing.

February 15th: Reaction of degeneration.

Case XVIII.

May 28th, 1893, admitted for recurrent fever into hospital on September 9th, 1893 and sent to the Beri-Beri ward. Though feeling some indefinite pains in the leg was discharged on November 26th in the hope that he might get better away from the possible source of infection.

December 11th, 1893 was readmitted with symptoms of Beri-Beri; no cardiac lesion but sounds distant and small; reflexes totally lost; sexual appetite diminished.
adenos over toes and dorsum of feet; calf muscles tense and painful on pressure; gait becoming characteristic on broad base; slight pharyngitis; no hydropericardium. February 16th Reaction of degeneration in legs only; gastric troubles.

March 1st Beri-Beri gait. Acute butie condition of gums; like the majority of patients in the Beri-Beri ward.

It is certainly an argument against the personal infection theory that as a rule Beri-Beri is confined to one ward of a hospital, though the patients of all the different wards move freely with each other; it is true that occasionally a case occurs in another ward which cannot be accounted for, but this so seldom occurs that I do not think that it can affect the general rule.

I have seen no reason to suppose that the same latrine accommodation is responsible for the spread of the disease; on the contrary I have specially noted that though very many patients use the same latrine the disease is still
confined to those who live in the same ward.

In conclusion I consider that Beri-Beri is a disease distinct from malaria, although in many cases the epidemiological factors are identical and the symptoms markedly similar, especially in those cases where the malarial poison has led to neuritis.

I agree, too, with Manson that Beri-Beri as a rule spreads by soil or place inoculation, and think that at a rule it is not directly contagious and does not spread by personal infection; though there are undoubtedly exceptions to both these latter statements. I think also that the germ is generated in the soil, and is introduced into the body by means of respiration; it, under certain conditions, being liberated from the soil and suspended in the air. I believe also that the breath of a Beri-Beri patient at certain stages of the disease, contains the essential virus, which virus is capable of contaminating buildings, clothes.
and beds, rendering them all sources of danger; that the virus is very tenacious of life, and exists for a lengthened, but at present, unascertained time; and that moisture, heat, bad ventilation and overcrowding all favour its growth and development.

Idegeneracy, I think, undoubtedly plays the same part in Beri-Beri as it does in all other diseases, for in no other way can I account for the fact that some men may be exposed for a constant and prolonged length of time to the influence of its virus and yet not contract the disease, while other men under the same influences for a comparatively short time fall victims to it. The following are two such instances; it will be noticed that both men have sat in the very midst of Beri-Beri patients for periods to be reckoned by years, that they are practically bed-ridden (one is entirely so) and are much debilitated, and yet neither exhibit a single symptom of Beri-Beri.
Case XIX

Pooh Ah Sue, kept out, Chinese woodcutter, was admitted to Hospital on April 1st, 1892, for general debility; he was almost completely blind from double glaucoma of many years standing. Owing to his blindness he seldom or never leaves the ward and but rarely his bed. During the entire period of his admission he has lived exclusively among the Beri-Beri patients, and refuses to change to any other ward. In April 1894 he was still totally free from the slightest sign or symptom of Beri-Beri.

Case XX

Jan Ah Shang, aged 55, was admitted into Hospital on July 15th, 1890 for hemiplegia. He occupied the same bed in the same ward up to April 1894, being surrounded on all sides by Beri-Beri patients and never left the ward; though at times he suffered much from severe attacks of malarial fever he has never at any time shown any sign of Beri-Beri.
Incubation Period. This undoubtedly varies within very wide limits, so that it is difficult, if not impossible, to fix a minimum or maximum period after exposure to the source of infection before and after which it can be said that the persons so exposed will not develop Beri-Beri. It seems, however, to be necessary that an individual should have been a certain length of time in the country before he can develop the disease, even after exposure to the virus, but I have seen no reason to agree with Manson's tolerance of the poison may be established by long exposure.

I think that the usual incubation period may be estimated by a few months as shown by some of the cases I have quoted who contracted the disease within the Hospital; under certain circumstances, however, the incubation period can be very much shorter—in fact almost a case of days instead of months—I think case XIII shows that here the incubation period was about seven days, while in case XIX it was between seven and eight days.
I desire to draw special attention to these cases as they show, as far as I know, the shortest incubation period that has been recorded, and to add that these dates are accurately fixed by personal observation, and do not depend upon mere statements of the patients or others who were not acquainted with the disease.
Classification of Beri-Beri

I should like to state at once, and to lay very great stress on the point, that there is but one type, a variety of the disease, and that that type or variety is Beri-Beri itself. Malison has undoubtedly clearly recognised this, but Bentley has gone out of his way to differentiate the disease into no less than five types or varieties, thus unnecessarily obscuring a somewhat already obscure disease. Bentley has, however, noted the intimate relationship of his different varieties as he says, "the initial symptoms of all these varieties are remarkably similar. The differences in the symptoms of each variety being due to the particular part or extent of the nervous system which is affected by the poison."

"There is, however, I think, a certain amount of excuse for falling into the error of profuse classification, for the disease at different stages presents such, at first sight, totally different aspects, that it is somewhat difficult to realise that one
has to deal with merely different clinical stages, and not with distinct different varieties; thus, in one bed a patient may be seen so completely waterlogged with generalized oedema that he is almost inhuman to look at with his immensely swollen limbs, bloated face, eyelids so oedematous that he cannot open his eyes, neck swollen to the level of the chin, unable to move and only able to speak with difficulty so that he may ask for something to put him out of his pain for ever; while the adjoining bed may contain a skeleton-like form, with every bone clearly defined, great atrophy of the subcutaneous system, relaxation of the ligaments of the various joints, inco-ordination of the limbs, and able to move only slowly with the aid of a stout stick. No wonder that the first case would be called the acute drop- 

tical or "wet" variety, while the second would be termed the "atrophic" or "dry" variety. Between these two there is an intermediate form, showing some oedema in the lower extremities, the chest and face, but able to walk about alone with a peculiar +
characteristic gait and he might be termed of the "mixed" variety.

Undoubtedly such a nomenclature has its advantages, but it is apt to be very misleading, for the "dry" variety of today, may have pretibial oedema tomorrow morning converting it into the "mixed" variety, and in a short period later the same patient may be so completely waterlogged as to deserve the name of the "wet" variety; while, vice versa, the "wet" variety may grow less oedematous and become "mixed," and the "mixed" may lose its remaining oedema and become "dry." It is all a question of degree as to which symptom is at the time most prominent, and as these are constantly changing I cannot see that it is possible to speak of different varieties: still, at the same time, it is often convenient to speak simply of "wet Beri-Beri" and "dry Beri-Beri," meaning by this that at the time the oedema was or was not a prominent symptom. Here I may mention, what I will draw special attention to by and bye, that often in performing post-mortems there is not.
the slightest oedema to be made out in
the extremities subcutaneously, but on
opening the abdomen a considerable amount
of ascites is almost invariably found, while
there is also an abnormal quantity of
serous effusion into the pleural and peri-

cardial cavities.

I have not yet seen an "acute pernicious"
case such as Bentley makes reference to;
though I have carefully observed a large
number of Beri-Beri patients.

Many patients exhibit spasms or twitching
of various groups of muscles, especially of
those of the tongue, and calf of the leg; and
flexors generally, but I have always looked
upon these as mere symptoms, and have
seen no reason to induce me to regard
them as of a separate type or variety.
It is true that Pekelharing and Winkler
record in detail an example of Beri-Beri
of the subacute type which was also a
"well marked case of real cramp" or
spasms, and in speaking of it say that
they have "only once seen Beri-Beri of the
consulutive type that Overbeek de Meyer
has so carefully described; I think that they are hardly consistent in their own classification of this as of the convulsive type, for later on, in speaking of the same patient, they say, the case we have just cited is especially important, because it proves more than ever, how some apparent differences in symptoms can be grouped together under one heading.

I hold, therefore, that there is but one variety or type of Beri-Beri, which variety or type exhibits at different times a large number of symptoms or groups of symptoms, some of which, being more prominent than others at a certain time, give to the case at that time an apparently totally different clinical aspect to what it exhibits at another time, but that all these symptoms are almost invariably present in every case of Beri-Beri in a greater or lesser degree, and that they are not to be regarded as constituting different varieties or types of the same disease.
Symptoms

One of the main difficulties I have had in connection with the investigation of Beri-Beri is the difficulty of talking to the patients, the majority of whom were Chinese of the coolie class who spoke no language but their own; this had to be interpreted to me either in Malay or in very indifferent English, the mental faculties of the interpreters being almost on a par with that of the patient, so that I often had a doubt in my mind as to the correctness of the statements which reached me.

Again, patients would often answer questions according to what sort of answer they thought was wanted, or their statements upon the same subject would totally differ from day to day, or they would deliberately conceal part of their history, or be unable to give any history of their illness at all, ascribing it to the influence of evil spirits or to the result of wind in their bones. Finally, I should like to point out that the Chinese can bear an amount of inconvenience and actual pain...
without a complaint which would be incredible to anyone who has not had actual dealings with them. The result is that an immense amount of time and patience is needed when drawing up the cases, and that the same reliance cannot be placed upon the subjective as it can upon the objective phenomena, while the very small amount of skilled assistance at the Hospital prevented the cases from having that amount of attention paid to them which they deserved.

I propose now to describe several typical cases so as to give a general idea of the disease, and then to deal in detail with the symptoms according to the systems to which they are referable.

Case XXI

Lim Ah Leng, aged 27, Chinese coolie admitted to Hospital on November 1st 1893.

Previous History. In August 1890, he came from China to Singapore, where for a year he cultivated his own garden; for the next twelve months he worked as a ricksha cooly,
About September 1892 he came to Johore and was employed in carrying earth, among previously he had contracted gonorrhoea and chilimes.

**History of Present illness.** About twenty days after contracting gonorrhoea (i.e. towards the end of August) there began a swelling and numbness in both ankles, gradually extending downwards to the toes; after the lapse of another fifteen days the same condition appeared in the fingers and extended to nearly up to the elbow; the swelling and numbness on both occasions began simultaneously. Generalised oedema then supervened, but not to any marked degree. His gait at this time was not affected, as he walked about two miles to the hospital without any inconvenience.

**Diet.** In China rice, congee, salt fish, vegetable daily; fish twice a week; pork on feast day, i.e. about once in two months; As a gardener in Singapore, rice, congee, salt and fresh fish and vegetables daily, pork twice a month, black beans three times a week. This he considered
the best diet. As a ricksha cooly, rice, fresh fish and vegetables, never salt fish or black beans (the latter being too "cooling") pork once in four days.

In Johore (where food is more expensive than in Singapore) he says he had the lowest diet he ever lived on, viz, rice, fresh fish and vegetables daily, with pork once in two months.

Pay. In China he had only just enough for food and tobacco; as a gardener in Singapore he made about $20 a month, some of which he saved to remit to China; as a rickshaw cooly about $12 a month, and in Johore about $9.

Notes on admission. Slight general aches all over the whole body; numbness over both arms and legs; puffiness of abdomen; slight pain in calves on pressure; gleek, a healing chancreoid; slightly enlarged glands of right groin.

General appearance. A tall powerfully built man; expression dull and apathetic, not amicable.
Decubitus. Sits on cot with legs crossed. (Tiler fashion); at times put arms behind his back with palms of hands resting on the cot so as to form a sort of triangular base to rest on, and thus take the weight of the body off the spinal column.

Nervous System. Sleeps fairly well at night, but, gets up to urinate four or five times. Complains of numbness and pain in both lower extremities, extending from the toes to the lower third of the thigh; the symptoms are more aggravated over the knee joints; the right lower extremity is more numb and painful than the left. No pain or numbness over the loins and back. Over the abdomen, especially in the right epigastic region, there is some pain and numbness. Numbness and pain also extend from the fingers to about two inches above the elbows. There is marked coldness to be felt over the toes, knees, and tips of fingers. No increased pain, any part of the body at night. There is severe pain on pressure between the
radius and ulna of both forearms, also between
the metacarpal bones, especially between
those of the thumb and index finger. There is
also great pain on grasping firmly the mus-
cles of the calf, but there is no special pain
between the interossei of the feet.
Patella reflex is entirely abolished;
 cremasteric well marked, abdominal,
 slight. There is a decided tendency to both
wrist and ankle-drop.
No anæsthesia of upper lip. Slight dim-
ness of vision since present illness began.

Gait. Walks slowly on broad base, lift-
ing the heels high and jerking the leg for-
ward with the toes pointing downwards;
walks almost entirely on the outer edge
of the feet with the toes directed inwards;
turns with difficulty, and whilst doing
so has to grasp some fixed object, while
he moves the feet round little by little;
alternately, without raising them off
the ground. The gait is of a staggering na-
ture, with the eyes fixed on the ground, and
the body thrown forwards as if he was going
to fall at each step.

Integumentary System. Skin dry and hot. T. 101°F. Right trapezius muscle over shoulder greatly hypertrophied from carrying earth. Slight edema of feet up to ankles, also of face and neck, puffy of eyelids; thighs swollen but do not pit on pressure; slight puffiness of abdomen; no edema of penis or scrotum.

Circulatory System. Pulse 118 per min. feeble, small, regular; slight pulsation in neck over the region of the carotids; apex beat visible half an inch below and in a line with the nipple. Heart's action--rumbling, with a mitral systolic murmur.

Respiratory System. Watery discharge from the nose; voice slightly hoarse. Respirations, 28 per min., regular and chiefly abdominal. Cough frequent and short, causing pain over the epigas-trium; profuse watery expectoration;
a few moist rales and whistling rhonchi are
to be heard scattered through the chest-espe-
cially at the left base posteriorly; percussion
note normal. The slightest exertion produces
shortness of breath.

Digestive System. Lips swollen, with the
mucous membrane dry and scaly, corners
of mouth ulcerated, teeth good. Tonsils
swollen and red, not retracted from teeth
and do not bleed on pressure. Foul bad breath.
Tongue, moist, broad and flabby, coated
with a thin milky film; edges and tip
clean; anteriorly there is a slight longitudi-
nal fissure.

Jawes slightly congested, but congest-
ion does not extend on to the hard palate;
mandible small and pointing somewhat to
the right side. No difficulty in deglutition,
appetite for last three days lost; fullness
and heaviness over pit of stomach after
even a slight meal. Bowels xostive.
Spleen slightly enlarged; liver normal;
no ascites.
Urinary System. About 60 ozs of urine excreted daily, of which more than half is passed at night; no albumen (see chart).

Progress of the Case.

November 12th. On protruding the tongue the fibrillary twitchings are so marked that patient put up his hand to steady it.

November 13th. Slight improvement in all the symptoms.

November 18th. Slight rise in temperature, not proceeded by rigor (see clinical chart); no appetite, bowels not moved, severe headache, cough more troublesome, face and neck more swollen, tongue very tremulous; skin hot and dry; able to walk as usual.

November 19th. Patient feels so much pain and weakness in the legs that he is afraid to try and stand up for fear of falling; lower extremities are very cold.

November 20th. General oedema much less. Looks better, temperature normal. In the evening he had some pain and difficulty
when swallowing.

November 21st: Can't use his chopsticks; middle, ring, and little fingers of both hands scirr. -flazed. Paralytic symptoms markedly worse since the attack of fever three days ago.

November 22nd: Snuffling respiration as if from drooping of the soft palate—nothing is to be detected on examination.

November 24th: Can't flex or extend legs without manual help; right leg worse: congestion of fauces is spreading on to hard palate.

November 26th: cremasteric reflexes absent.

November 28th: Less pain in calves.

December 17th: Can walk slowly with aid of a stick.

December 20th: During the last two days has had a good deal of diarrhea.

December 23rd: Sexual desire completely lost since his illness began. Is bedridden again since the diarrhea.

January 3rd, 1874: Can again walk with the aid of a stick.
February 2nd Reaction of degeneration well marked.

March 7th Walks well without the aid of a stick; says he has lately had some fever which has left pains in both knees and ankles.

March 20th Walks very well alone without the characteristic gait, but cannot run for fear of falling; patella reflexes still absent.

April 1st Discharged from Hospital.

Remarks.

Note the disease appearing after gonorrhea and poor feeding that the symptoms were more marked on the right than on the left side, and that fever and diarrhea preceded the attacks of paralysis while in the hospital.
Clinical Chart of Case No. XXI

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Wine: 9.11.93, 16.11.93, 23.11.93, 30.11.93

Reaction: Acid, Acid, Acid, Acid

S.G.: 1017, 1015, 1015, 1030

Albumen: - - - -
Case XXII

see al chi, at 36, Chinese Gambier coy., was admitted to Hospital on November 13th, 1892.

He complained of slight pains of a rheumatic nature, scattered through both extremities, with almost imperceptible numbness of the legs: he walked on a broad base with somewhat high action of the heels; the patellar reflex was abolished. This condition improved till he was on the point of being discharged when on December 16th he developed a slight cough without any expectoration; he eaten well and walked about as usual making no complaint.

December 21st at 6 am. was found sitting on entrance steps to ward complaining of great tightness across the chest, with difficulty of breathing.

8 am. lips blue, pulse 130, small and irregular; respiration 40, muscles of extraordinary respiration in full action.

Said he felt as if he was going to die and implored us to help him. He spent the whole day seated on the floor with his arms and head supported on the cot, he
dyspnoea increasing if he attempted to lie down.

6 p.m. Lips black, gasping for breath, pulse small, slow and thready; heart sounds inaudible and indistinguishable from the heavy breathing; extremities cold, still talked rationally asking for help, referring all distress to the precordial region, and saying that he felt that he was just about to die.

7 p.m. died quickly, having unconscious for the last half hour, after suffering great agony for 13 hours.

Post-Mortem. Body muscular and well-nourished, no oedema of extremities: pericardium contained 6 ozs pale serous fluid; heart enlarged much distended with dark fluid blood (no clots), especially the right ventricle, slight effusion into the pleural and abdominal cavities. Lungs much engorged and oedematous; all other organs normal.
Case XXXI

Ying al Ing, at 28, Chinese gambier, coolly admitted into hospital for ulcers of both legs and itch on October 24th 1893. Case taken on December 13th; died December 14th.

History. Came from China ten years ago and went to Batu Pahat as a gambier cool; six months after his arrival he fell ill with general numbness over the whole body accompanied by loss of power in both lower extremities; for this he was bed-ridden for eight months, after which he slowly regained the use of his limbs and the numbness disappeared. From time up to last year he remained in good health; he then left the Batu Pahat district and worked on another gambier estate. Five months ago the numbness returned in both upper and lower extremities, together with pains in his back for which he was treated in the Johore Hospital for about three weeks. Having broken out with itch and ulcers with slight numbness, he returned to hospital on October 24th 1893, but on admission did not make any mor.
tion of the numb feeling.

Pay. In Babi Pahat ten dollars a month, with food, most of which he spent in gambling; on the other gambier estate eight dollars and his food.

Food. In China - 3 meals a day - rice, congee and potatoes daily, also fresh and salted vegetables, fresh fish twice a month and pork once. In Johore 5 meals of congee, salted and fresh vegetables, rice, salt fish and pickles daily; black beans three times a week; fresh fish once a month and pork once a fortnight.

General appearance. A well built muscular man, eyes sunken, face drawn, expression dull and listless; slightly anaemic.

Zonurus. Lies on either side with equal ease; if he lies on his back he immediately experiences great dyspnea.

Herodius System. Sleep disturbed owing to the distress of breathing; numbness of both legs, especially in soles of feet, extending upwards to six or seven inches about the knees - the tips of the fingers are also numb, but otherwise the upper extremities are not
affected. Great pain in the calves on pressure, also between radius and ulna, and the interossei of the hands. General diffuse tenderness over the abdomen on gentle percussion. Patella reflex abolished, cremasteric and abdominal present. No wrist or ankle drop.

Gait: walks very slowly and carefully, with short paces, bringing both feet flat and gently on the ground; has to be supported on either side while he walks, locks both knee-joints firmly together and keeps his eyes fixed on the ground, the whole body being inclined forwards.

Integumentary System. Body covered with itch, superficial sores over both great trochanters. Skin dry and harsh. Oedema of both feet and legs, extending slightly over the thighs; paws and serotine oedematous; abdomen puffy, slight oedema over sternum; no oedema of upper extremities. Face puffy and swollen.

Circulatory System. Radial pulse 120, small, soft and regular; no pulsation in neck; heart heaves slightly,
and there is a mitral systolic bruit, the area of cardiac dullness is not increased.

Respiratory system. Voice natural, tongue slightly inflamed and relaxed.
Form of chest round and symmetrical, respirations 30 per min., jerky, complains of great dyspnoea, and refuses to distress feeling to the precordial region and pit of stomach. Over the left lung anteriorly a few cooing rales are to be heard, otherwise the lungs are normal. The breath is very offensive, and at the angles of the mouth there are small white mucous patches—such as are very common in most natives.

Digestive system: lips slightly everted, teeth irregular and dirty, gums normal. Tongue pointed, moist, coated with white fur in the middle, edges clean and glazed, papillae prominent, at tip, fine fibrillary twitchings when tongue is protruded. Stomach slightly congested, appetite lost, no nausea, vomiting or diarrhoea; sense of fullness and distension even after a very slight meal.
he says that everything he eats turns to wind.

Genito-urinary System. Urine Acid, Sp. Gr. 1010, no albumen or abnormal constituents.

For about the last six months has entirely lost all sexual desire.

December 11th. Penis and scrotum more edematous; some dyspnoea.

December 12th. Dyspnoea easier after getting the bowels well open with croton oil.

December 13th. Same taken condition as above.

December 14th.

Refused his breakfast and vomiting a small quantity of yellowish matter. 6 am Pulse 108, small and regular; heart sounds distinct, careful examination of the chest gave only negative results, except that there were a few scattered moist sounds throughout both lungs. Respiration 28 a minute, a good deal of dyspnoea, with a "tight" feeling in the precordial region. 1 pm. Patient sat up on his cot as usual during the dinner hour but did not eat anything, and soon afterwards without making
any complaint seated himself on the floor somewhat on his right side propped up by means of a low stool. 3 p.m. donated a large quantity of watery fluid; cold and clammy; great dyspnea, gasping for breath, alas! widely dilated; quite unconscious; pulse imperceptible, and heart sounds entirely obscured by the laboured breathing. 

4.35 p.m. Died quietly.

Postmortem. 17 hours after death.

Rigor Mortis nearly all passed off; great hypostatic congestion of skin.

Lungs. each pleural cavity contained about seven ounces of clear fluid: no pleuritic adhesions; on section lungs very frothy and congested.

Pericardium - a good deal of fat scattered on anterior surface; hydropericardium = 3/4 oz.

Heart much enlarged; anterior surface consisted almost entirely of the left ventricle and was covered with a thick layer of yellow fat.

Right auricle dilated, half filled with a firm auto-mortem clot which...
extended through the tricuspid valve.

Right ventricle much dilated, walls abnormally thin and contained a good deal of fat, muscle pale but not friable; full of firm white ante-mortem clots with some dark fluid blood. Tricuspid valve very thin and fractured, edges thickened and rough.

Left auricle, small ante-mortem clot extending through the mitral valve.

Left ventricle, somewhat enlarged; walls hypertrophied, muscular papillares very strong; muscle of good color and not fatty; ante- and post-mortem clots. Mitral valve thickened and rough at edges. Other organs healthy.

Remarks

Note that this was the third attack of Beri-Beri that the patient appeared only really ill for three days, and that the death occurred somewhat suddenly within three hours; the right side of the heart was fatty, while the tricuspid and mitral valves showed traces of same former endocarditis.
Case XXIV

Beri-Beri. (dry)

Jup ah How, at St, Cooly, jahore sawmill.

Duration of illness 50 days.

Admitted 27th October 1893. Case taken 16th Nov. 93.

Discharged 24th March 1894.

History. Left China three years ago for Singapore. During his life there suffered only from trivial diseases. Did not stay long in Singapore but left almost immediately for Rhuo, where he worked as a gambier cooly for 7 months. After this he came to jahore and worked in the sawmills carrying planks etc. A month previous to his admission he suffered from severe attacks of fever unaccompanied by rigors. At the end of this month he felt numbness in both legs, which gradually increased, confining him to bed. He attributes his illness to exposure and damp.

Diet. In China this consisted of rice and boiled vegetables, very seldom pork and fish. In Rhuo there was an improvement in the food—salt and fresh fish, vegetables, etc.
a week and pork occasionally in addition to the usual rice. In the other he only had rice and black beans, fish and pork very seldom.

**General appearance.** A medium sized man, fairly well nourished, sallow but not anaemic, face slightly drawn, otherwise cheerful. Lies on back naturally, when sitting an edge of bed toes point to the ground, and touch it, heels raised, and hands grasp edge of cot as if to take weight of body off the spinal column.

**Integumentary system.** Dry and smooth, no oedema. Temperature normal. Except at hands up to wrist, and feet and leg up to knee, which feel distinctly colder than the other parts of the body.

**Circulatory system.** Pulse full and regular, slight pulsations in neck, and superficial veins on right side fairly engorged; apex beat of heart invisible, but impulse to be felt a little below the nipple to the inner side; cardiac

Digestive System. Lips separated. Teeth well formed, regular; not decayed. gums of both jaws red, slightly ulcerated. a faint blue mark along the edges of the gums of the upper jaw. Tongue. broad, moist, covered with a thin coating of fur at back part; tip and edges devoid of epithelium. No difficulty in deglutition. Intense congestion of farces extending forwards to hard palate. Appetite good. No nausea or vomiting. No pain or uneasiness at pit of stomach before or after meals. Bowels move twice daily, costive; no pain in defecation. Spleen slightly enlarged.
**Nervous System.** Kinniness over legs; patient lies down easily, but on attempting to rise to a sitting posture is only able to do so by placing both elbows on the bed as a fulcrum, and thus raising his head and upper part of trunk up with a sharp jerk. When sitting on the edge of the bed he cannot stand up without assistance, either by means of a stick or by someone catching him by the arm. He says this is due to weakness in his ankles and a dragging pain in his calves. There is marked paralysis of extensors of both legs. Ankle drop: toes are practically immovable. He can't flex or extend them in the least.

Feels intense pain in the calves, when grasped the skin is painful and hangs in folds showing the great atrophy of the muscles, when the muscles of the calf are grasped they are felt to be much atrophied and hard and give intense pain.

No wrist drop, but fingers are tremulous on extension.

He is unable to straighten his back on standing, as it feels weak as if it
it would give way; stands supported by a stick with his head forward and feet wide apart—thus forming a triangular base. Dumbness over dorsum of both hands, ceasing at wrist, also from both knees downwards.

Experiences a feeling of cold in both legs, especially at night; on palpating the anaesthetic parts (i.e. hands and legs) there is a marked coolness to be felt compared with the other parts.

Gait. Walks on broad base by means of a stick, which he places in front of him, and then walks or shuffles up to it; he throws his limbs out and feet inverted, placing the whole of the sole on the ground; the outer side, however, touching the ground first; the heels are raised up when walking in order to get over the toes, and the foot is somewhat jerked outwards as if proper control over it had been lost. In walking or shuffling along he first puts the stick forwards, then he draws one foot after it, and then draws the second.
foot up to the level of the first foot but does not seem able to place it beyond the first foot — 1 = 1 — 1 = 1.

All reflexes are absent.

Pain on pressure in both forearms between the radius and ulna about half way up. Light and sleep unimpaired.

No albumen. Slight phosphates (16. 11. 93).

Short notes since admission. 16. 11. 93.
Patient bedridden, intense pain over thighs and legs. Relaxation of ligaments of ankle joints & paralyses of extensors of the legs.
Looks careworn and as if in great pain. Travel slightly ulcerated.

November 15th. 1893. Fine fibrillary twitchings of tongue, which in the middle line anteriorly resembles the strawberry tongue.
21st November 1893. Eyes slightly prominent.
24th November 1893. Can walk about without assistance or stick, but in a staggering manner.

From 2.30 to 4.30 p.m. he was feverish, without any antecedent rigor, lower lip swollen and inflamed, tongue furred and angry looking.

25th November 1893. Swelling of lips less.
28th November 1893. General improvement; skin moist.

9th December 1893. Has had ten stools within 24 hours - somewhat dysenteric in nature; 99°F.

11th December 1893. Cardiac sounds extremely distant and feeble; some moist sounds at upper half; right lung posteriorly.

12th December 1893. Sexual desire diminished since illness, but not completely lost; when in good health has an erection every night. At present scarcely twice a week.

4th January 1894. Improving steadily; walks fairly well without a stick.
9th January 1894. Walks on a base of 16 inches, and complains of pain in the calves.

7th March 1894. Improvement continues, gap in base not so broad; still has high action of the feet, and falls on attempting to run. Patellar reflexes still absent.

24th March 1894. Asked for his discharge, walking on a good firm base.

Urine:

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History. Patient states that he left China 8 years ago proceeding to Batu Pahat, where he worked as a gambier cooly during that time. He was 25 years old when he left China, where his occupation was that of a gardener; has no recollection of ever falling seriously ill in his native place. At Batu Pahat he has had several attacks of malarial fever off and on these past 8 years—his 1st attack occurring two months after his arrival. Four months before admission into hospital, the first symptom of his present illness made its appearance, which he attributes to exposure to rain.

His illness first took the form of swelling in his both feet extending upwards gradually.
implicating whole body, followed by numbness in the same order a few days after the swelling on the estate where he had been working. He knows of another case of the same type, who got ill before him, and with whom he frequently came in contact, and in fact they lived together, and he also states that they used to cook for each other by turns. He was sent down from Batu Pahat, together with the case he alludes to above, by the Police authorities as being members of some secret society. When in Batu Pahat he states that the generalised oedema was slight, but that the swelling and numbness decidedly increased after his arrival in Johore. Here he and his friend were confined in the same room for 20 days and nights at the Police lock-up. His friend was in a very bad state, quite swollen out and gasping for breath. He died on the 20th night; after the death of his friend, the next day he was sent to hospital by the Police. About a month after admission, great difficulty of breathing set in, all the other symptoms became aggravated.

General Examination: There is generalised oedema of the whole body, including feet and
peritonum as also face and neck. Marked ascites, subconjunctival edema, wellmarked over scrotum, edema of both lungs, normal posture in bed lying on either side; complained of general numbness.

Patellar tendon reflexes abolished as also cremasteric and abdominal.

Treatment from beginning together with short notes.

3rd September 1893.
Given a mixture containing iron, quinine, arsenic, and digitalis, with warm fo-mentations to the scrotum and penis, and a drachm of compound jalap powder every second morning.
November 12th 1894. A strong diaphoretic mixture every four hours.
November 15th. Trouble some cough began today, so was put on an ordinary cough mixture containing carbonate of ammonia and senna.
November 19th. Urine scanty, with some feverishness; given an ounce of fresh infusion
of digitalis every four hours, with quinine
and antifebrine as required.

November 23. Bedsores forming over great
trochanters, scanty urine, very constipated.
Given drachm doses of bitrate of potash,
and one minim doses of eroton oil
every second day.

December 5. Oedema greatly diminished,
the potash and eroton oil stopped; pulse
very weak.

December 19. Has had a great deal of
diarrhoea the last few days; oedema
continues to diminish.

January 2nd, 1854. Since the last note
patient has been getting gradually
weaker, dyspnoea much, and
is now more like a case of "dry"
beri-beri.

January 3rd, 1854. Died quietly at 3 a.m.
without any precordial distress.

Postmortem - see next page.
Postmortem 11. am. 3. 1. 94.

Body flabby and pale, general edema all over but to no very marked degree; bedsores over great troCHANTERS and iliac iocchi.

Pericardium, clear fluid.

Heart of about normal size: the right side was seen to be of markedly gelatinous-myomatous form of degeneration; this was especially marked along the right free border from the base to the apex, and along both right and left auriculo-ventricular grooves.

Right auricle-normal size, contained no clots; its walls were so thin that they seemed to be on the point of rupturing, it being almost totally devoid of muscular tissue and consisting only of the visceral layer of the pericardium, through which the degenerated pale muscular papillaries were plainly visible from the outside.

Right ventricle-slightly dilated, walls extremely thin, especially towards the apex; the muscle was very pale and friable.

Tricuspid valve perforated; muscular papillaries and cords tendineous pale and small. Pulmonary valve competent.
No clots only dark fluid blood.

The left heart as it lay in situ was hard and firm like a cricket ball but not so large as one.

Left auricle - no clots.

Left ventricle - markedly concentrically hypertrophied, muscle of good color, not friable and no fat. Contained no clots.

Aortic valves allowed of slight regurgitation, two of the cusps were so thin that at their edges they had become slightly fenestrated.

Abdomen 200 cc clear fluid.

Stomach contracted and pale.

Liver not enlarged, firm and hard.

Spleen somewhat enlarged, very friable.

Pleural cavities contained about 5 oz.
of fluid each; lungs, small, pale and floating on the fluid; both lungs on section were markedly spotty; the right had no adhesions, the left was firmly adherent throughout except to the pericardium.
Remarks.

This is a case where symptoms were markedly aggravated by incarceration in the insanitary Police cells of Juhore.

Body at one time was completely waterlogged; under treatment the oedema began to go down, and there was some chance of his developing into the dry form of Beri-Beri.

Bedsores formed freely for some weeks before his death.

The condition of the heart was due to the prolonged strain in it, on account of the congested and waterlogged lungs.

Death was eventually due to asthma; the Right heart muscle having atrophied and wasted so much that it could no longer contract and simply stopped working from pure inanition.
Beri Beri (dry)

**Case xxvi**

Name: Hul Ab Sam

Age: 30.

Occupation: Rickshaw Cooly

Duration of illness: 2½ months

Admitted: 1.10.93

Case taken: 18.11.93

Died: 18.11.93 2.30 p.m.

Previous History: Came from China six years ago and has been working ever since in Johore as a rickshaw cooly.

In China he was a carpenter; while there he had two attacks of fever, lasting about three months each time.

Two and a half months ago for the first time since he came to Johore he became ill; numbness and swelling of forearms and legs; the swelling and numbness began in the feet, and extended upwards to the thighs. About the same time as the feet the hands also became numb, extending upwards to the arms. About a week after numbness set in the same parts began to swell, but not
to any marked extent.

A week before admission to Hospital he felt a sense of oppression or "tightness" in his chest in addition to his other symptoms. Had chancre and bubo about a year ago.

On admission the following symptoms were taken: numbness in both lower limbs, as also in upper extremities; oppression at chest & difficulty of breathing. Bubo scar. Left groin. Heart sounds quick and thumping. Pain in both calves. Difficulty in walking.

Pay. In China, dollars six a month with food, most of which he spent on opium. Has smoked opium for 10 years.

In Johore he got 25 cents daily - 14 of which he spent on opium.

Food. In China he had rice and boiled potatoes, salt fish once or twice a week, and pork in feast days i.e. once in two months. In Johore, where the food was better, he had rice, vegetables, and salt fish daily. Fresh fish very seldom, pork once a week, no black beans.
General appearance - fairly well nourished able bodied man; face somewhat drawn, conjunctiva anaemic.

Decubitus. In bed lies on his side mostly; if he lies on his back he feels as if he would be suffocated in the chest by the "wind," rushing up from his stomach and choking him. Out of bed he is not able to stand owing to numbness of his lower limbs.

Integumentary System. Skin dry and cold, no oedema present. Bubo near left groin; large ulcer 5 cm. on right leg. No cutis anserina.

Circulatory System. Pulse hardly perceptible, intermittent and flickering. Heart's action irregular and intermittent, with cardiac sounds distant & accentuation of the second sound.

Respiratory System. Voice natural, but distressed, movements of chest regular, but respiration slow and laboured. Great retraction of intercostals during inspiration; also nasi broad and dilated.

Refers pain and difficulty in breathing
to bit of stomach at each respiration, tossing about from side to side, throwing his arms about as if he were being suffocated.

Answer all questions coherently and correctly.

Digestive System — lips separated, cold and livid, teeth well formed, gums blue, not retracted, tongue dry coated & viscid saliva at its middle, tip and edges clean, anterior papilla prominent.

Jawless congested, including tonsils and palate - not hard palate. No difficulty in deglutition, no twitchings of tongue.

Nervous System — no paralytic symptoms present, no wrist drop, no ankle drop, no contraction of flexors or extensors of any of the extremities.

All reflexes absent. No anaesthesia of upper lip. Severe pain on dorsum of both feet between intersossei, also in calves and anterior surfaces of thighs on pressure. Insensibility in lower extremities from the feet upwards - marked numbness over loins and over whole anterior surface of abdomen.
On admission on 1st October 1893 had rheumaticy pains in knees joints, and put on mixture of lord: salicyl: & liniment jerebith for leg.

7th October Puls Jalapa Co. every other morning.

30th October Gargle of Pot. chlorat: with true myrrh.

13th November. Mixture beri-beri. 1st.

16th November. Vomiting, no nausea but stomach irritable - loss of appetite.

Diarrhoea with blood and mucus, about 12 motions, after each motion had pain at pit of stomach and lower part of abdomen.

17th November Great dyspnoea and cardiac distress, almost total loss of sleep: sit up constantly, not able to stand; feels very cold, temp. subnormal. Pulse = flickering, heart still regular. Respirator 32.

Dry cupped precordial region. Amyl nitrite inhaled. Respiration fell to 28, and pulse at wrist improved very slightly. Talks rationally. Sense of impending death.
18th November. All symptoms of yesterday aggravated; radial pulse not to be felt; heart intermittent and tumultuous. Treatment repeated & mustard plaster to heart.

Respirations 32 long and full. Answered all questions while his case was being drawn up.

2.30 p.m. Died of great cardiac distress.

Post-Mortem 19th November 1895. 22. hours after death.

Rigidity not very well marked. Good deal of lividity, partly from hypostatic congestion.

Heart. Pericardium, 3 xiii pale straw colored fluid.

Heart; large veins dilated, slight patch of superficial fat.

Right auricle and appendix completely occluded & firm white ante-mortem clot, extending through the tricuspid valve into right ventricle.

Right ventricle, two elongated ante-mortem clots; distended & dark venous blood, and a few small post-mortem clots.
Valve and muscular papillae healthy and prominent.

Right auricle \{ \text{somewhat distended with blood and post-mortem clot.} \\
Left ventricle \}

Valves - all normal.

Heart was a large one, chiefly due to the large Left ventricle; heart muscle good, not friable or fatty.

Lungs - normal - no pleural effusion.

Abdomen, liver greatly engorged with blood, and bile stained. Stomach distended; all vessels engorged; no effusion or only a few drachms into the abdominal cavity.

Brain. Some effusion into cranial cavity and also into vertebral canal. Superficial vessels much distended. Some tough adhesions between coverings of Right hemisphere. The Medulla Oblongata and Pons were pale.

Spinal cord not examined.
Notes

General distension and engorgement of vessels probably due to the inhalation of amyl nitrite the day previous to death and the day of death.

From the small amount of pericardial effusion death may have been primarily due to the cardio-inhibitory action of the vagus, and secondly to the large acute-mortem clot slowly forming in the right auricle.

The amount of effusion in the pericardium (3% of) was comparatively so small that I doubt if it held any thing but a tertiary place in the causes of death.

The vomiting which set in two days before death probably also pointed to implication of the vagus.

The diarrhoea may have been due to loss of nerve control over the sphincters and lower bowel.
Beri-Beri (Moist)

Case. xxvii

Lee Ah Keng at 23. Shop boy
Admitted 6.12.93.
Base taken 9.12.93.
Duration of illness 15 days
Discharged (at request) improved 3.1.94.

History. Came from China 10 years ago and worked in Johore for 3 years as shop boy; after spending the next five months in China he returned to Johore to his old work for one and half year. He then fell ill with general edema and numbness, and went back to China about four and half years ago; after remaining there five months he became perfectly well and came back to Johore where he has been in good health for the last five years.

About 12 days ago he slept out in the yard and on rising next morning found his face swollen. He consulted a Chinese doctor and took his medicine for two days; finding himself getting worse,
both his feet and legs becoming acedemic, 
he came to Hospital.

He states that his condition on admission 
was practically the same as when he went 
to bohia last time; he says the sea voyage 
alone did him a great deal of good, for when 
he landed in bohia he had no swelling, but only numbness scattered over the upper 
and lower extremities, and that after few 
days in bohia even the numbness disappear.

Day Dallar six food, sends a portion 
to bohia and saves the remainder, of 
alike, he has taken to gambling but still 
recuits money home though he saves now.

Bohia in bohia, three meals a day, con- 
sisting of rangee, salted vegetables, salt + 
fish and fresh vegetables daily, fresh fish and 
pork twice a month.

In johore three meals, rice, fresh fish, 
salt fish, pork and pickles daily.

General appearance - fairly well nourish-
ed young man, stout and compe, face drawn, 
expression anxious.
Decubitus lies on either side with equal ease; if he lies on his back he experiences great difficulty in breathing.

Walks on a broad base and as if about to fall, knees firmly locked, throws feet heavily forwards, his heels touching the ground first; turns with great difficulty, owing to partial loss of control in both limbs.

Integumentary System: skin smooth and dry, never perspired now; oedema of both feet, legs and thighs, puffiness of abdomen. No oedema of upper extremities, face swollen, both lower eyelids puffy; slight oedema of chest.

Circulatory System: Pulse 60, small, regular, markedly diastolic; no pulsation at root of neck or higher up; apex beat of heart visible about 1 inch to the inner side of left nipple.

Heart's action slow and weak. Marked mitral systolic murmur at apex.

Respiratory System: voice has become shriller and more effeminately - tongues and fancies inflamed.
Respiratory movements regular: experiences great difficulty of breathing referring the distress to over the procoroidal region. Cough rare, not painful, no expectoration only coughs when he feels suffocated.

Digestive System: Lips separate, mucous membrane of lips and buccal cavity reddened and inflamed; gums reddened, not retracted. Several teeth extracted for caries. Tongue broad and moist, edges clean, tips glazed and raw-purred. Glands congested, extending over to hard palate. No difficulty in swallowing; appetite impaired, feeling of fullness over pit of stomach after eating even a small quantity. Bowels constipated. Liver and spleen normal.

Nervous System: Very restless, sleep disturbed from the great dyspnea he experiences. Innumbness in both feet, legs and thighs-less at groin. Innumbness over back and loins, also over abdomen especially at epigastrium, innumbness over front of chest, also in upper extremities from fingers to axillae. Innumbness increased
at night. Patella-tendon reflex abolished, abdominal and spermatic present.
No pain in calves on pressure, or between radius and ulna.
The swelling preceded the numbness. Upper lip not anaesthetic. No special sen-
sation of coldness, no goose skin. Can flex legs on thighs, and thighs partly on abdomen.
Numbness more marked over flexors than extensors in both upper and lower extremities.

[Underlined: Sexual desire totally lost since illness.]
[Underlined: no previous ever.]
Progress of Case

24th December 1893. Oedema of feet going down.

26th December 1893. Oedema of legs and puffiness of face much diminished.

28th December 1893. At noon became febrile, at 6 pm. the temperature being 104°.

29th December 1893. Morning Temperature 98°. Evening 100°.

30th December 1893. Morning Temperature 99°.

31st December 1893. Morning Temperature 98.0°. Evening 100°.

1st January 1894. Temperature normal, no paralytic symptoms.

2nd January 1894. Patient said he felt so much better that he insisted on leaving Hospital.
<table>
<thead>
<tr>
<th>Date</th>
<th>Pulse M</th>
<th>Pulse E</th>
<th>Respiration M</th>
<th>Respiration E</th>
<th>Temperature M</th>
<th>Temperature E</th>
<th>Diarrhea in 24 hours</th>
<th>Weight Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dec 11</td>
<td>60</td>
<td>72</td>
<td>18</td>
<td>20</td>
<td>98</td>
<td>98.4</td>
<td>1 H</td>
<td>64</td>
</tr>
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<td>52</td>
<td>70</td>
<td>16</td>
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<td>97</td>
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<td>70</td>
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<td>96.8</td>
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<td>48</td>
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<td>29</td>
<td>96</td>
<td>108</td>
<td>16</td>
<td>20</td>
<td>98</td>
<td>100</td>
<td>1 S</td>
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<td>99</td>
<td>100</td>
<td>1 S</td>
<td>54</td>
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<tr>
<td>31</td>
<td>90</td>
<td>108</td>
<td>18</td>
<td>20</td>
<td>98</td>
<td>100</td>
<td>1 S</td>
<td>42</td>
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<tr>
<td>Jan 1</td>
<td>72</td>
<td>90</td>
<td>18</td>
<td>20</td>
<td>98</td>
<td>98.4</td>
<td>1 S</td>
<td>48</td>
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<td>2</td>
<td>72</td>
<td>90</td>
<td>18</td>
<td>20</td>
<td>97.8</td>
<td>98</td>
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</table>
Remarks.

Note that this is the second attack of Beri-Beri, the first being about six years ago; for the last five years he has been in good health, this points to the prolonged period during which the disease may be dormant. The present attack was brought about by sleeping out in a yard—that is, not off the ground or guarded by a mosquito curtain; in this respect it bears some likeness to an attack of malarial fever.

Note also the initial systolic bruit—which is found in the great majority of Beri-Beri cases.


Case XXVIII

Beri-Beri (mixed)


Sambier Kooy.
Admitted 31. 12. 92.

Disease Beri-Beri (dry).

Died 27. 11. 93.

Post mortem 28. 11. 93.

An admission was unable to walk without the help of a stick, numbness and pain in lower extremities. Reflexes abolished.

14th February 1893. Complete relaxation of ligaments of ankle joint; complete loss of motor power in both lower extremities. Paralysis of extensors.

About this time oedema of feet began and extended upwards to the knees, also began in both hands and extended half way up both upper arms.

5th March 1893. Accentuation of 1st cardiac sound.

About middle of March chronic diarrhoea set in, and lasted about three months when all oedema had passed.
away and patient was left as a mere skeleton. Patient then began to pick up a bit, complaining merely of indefinite pains throughout the body—especially in the muscles and joints of the lower extremities.

16th June 1893. Had an attack of dysentery, after this patient never rose from his bed—not even to sit up for nourishment; the dysentery lasted till about the end of June.

From July patient went markedly downhill, gradually losing his appetite.

About the middle of October bed sores formed over sacral regions and great trochanters.

20th November 1893. Paralysis of vocal cords
27th November 1893. Died quietly at 2.10 pm.

Post-mortem. 28th November 1893. 10 am.

Body greatly emaciated, bedsores and dirt; no anaemia. Heart, small; pale.

Hydro-pericardium = 3 m3

Right auricle. No post-mortem clot; some
Right ventricle—what distended & dark venous blood. Walls of ventricle abnormally thin.

Left auricle—apparently normal.
Left Ventricle: Walls concentrically hypertrophied; muscle in good condition. Valves and state of vessels normal.

On base of Right Ventricle was a small milk-white patch the size of a 20 cent piece; it was quite superficial, and the muscle underneath was totally unaffected.

Right Pleural cavity: 3/4 fluid.

Right lung - adherent to pleura; on section was frothy and adenomatous.

Left Pleural cavity: 3/4 fluid.

Left lung entirely covered down to parietal pleura; in parts empty, in others on section frothy and slightly emphysematous.

Abdomen contained 100 oz clear fluid. All organs healthy but pale, the gut especially being of a glistening white. Kidneys apparently normal. Spleen very slightly enlarged. Liver pale but almost of normal size. No adhesions between gut.
Remarks

This is a case of "mixed" Beri Beri, originally being moist and then becoming of the dry or atrophic form. Death was probably partly due to asthenia following exhaustive diarrhoea and dysentery.

Note again the large amount of ascites without any anuricca or of the catarrhics.

Diarrhoea in 16th June 1893, greatly aggravated all the symptoms.

General Facts.

Muscular System. Great atrophy often rapidly follows an attack, which is best seen, and as a general rule begins, in the muscles of the calf, but may extend to the entire muscular system. Pain, varying from tenderness to intense agony, is invariably present at some stage of the disease, especially when superficial or deep pressure is made on the muscles—and this is so characteristic that it may almost be said to be pathognomonic.
Dropy or oedema is another characteristic symptom, and it also, in some degree, invariably present at some stage or other; it begins, or is rather first noted, on the anterior aspect of the tibia, and in the tissues surrounding the ankle joint; from thence it may spread over the entire body, filling all the cavities and stretching the skin almost to the cracking point, until the patient is completely waterlogged and resembles nothing so much as a prize pig.

In these severe cases of "wet" beri-beri, the serotum is often enormously swollen, while the penis is so twisted that urine can scarcely be passed. It is specially to be noted that even in the "dry" form, when the body is terribly emaciated and the muscles so atrophied that all progression is out of the question, a considerable quantity of fluid may be found in the chief cavities—especially the abdomen, though it is often not recognised until the post-mortem is
The general appearance and expression of the face is often so peculiar that a diagnosis may be made as the patient walks into the consulting room; the cheeks are full and glossy, the expression dull, and the general idea is one of hopelessness and helplessness.

Other cases again have nothing characteristic about them—which perhaps is not saying much, as the majority of patients are Chinese who are practically expressionless.

The attitude is only noteworthy when the patient walks on the broad base with body thrown forward, supporting himself with a stick, or any near object, which is usually known as the "Beri-Beri gait."

The temperature as a general rule may be said to be normal or subnormal; undoubtedly the onset of the disease is usually marked by fever, running up to 310° or 103°, but it is noteworthy that this rise of temperature is not preceded
by a rigor, as in malaria, but that the
body gradually gets warm. Hence the natives
of the Straits Settlements are in the habit
of distinguishing the fever of beri-beri
as “paulas” (Indlay=hot) from the fever
of malaria as “denam” (Indlay=fever), so
that in the early stage of the disease,
when one does not know whether one is
dealing with malabarian fever or beri-beri
one is led to pay some attention to the
word the patient uses, and often finds
it helpful in an early diagnosis.

Alimentary System.

The gums may be swollen and tender, as in those cases (90, 10, 111) with perio-
dontal symptoms, or they may be retracted from the teeth which may be covered
with sordes, but as a rule this varies so
much that it is not a symptom of any
value.

The tongue is dirty, and white fur
anteriorly, with the red papillae some-
what prominent; posteriorly it is very
yellow and foul looking; at times
fine fibrillary twitchings are well marked on protrusion; in case 92, it being so much so that the patient had to steady his tongue with his hand; it is almost always indented by the teeth.

James. Bentley claims that he is the first observer who has drawn attention to the frequency with which the fauces were inflamed. In the Bihore Hospital I specially enquired into this by examining 94 patients in one morning—nine of whom were specially selected—with the following result:

<table>
<thead>
<tr>
<th>Condition</th>
<th>No.</th>
<th>Beri-Beri</th>
<th>Pre-Beri-Beri</th>
<th>Beri-Beri</th>
<th>Pre-Beri-Beri</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total R. Exam.</td>
<td>94</td>
<td>30</td>
<td>30</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Normal</td>
<td>31</td>
<td>48.43</td>
<td>20</td>
<td>66.66</td>
<td></td>
</tr>
<tr>
<td>Slight pharyngitis</td>
<td>22</td>
<td>34.37</td>
<td>3</td>
<td>10.00</td>
<td></td>
</tr>
<tr>
<td>Marked pharyngitis</td>
<td>11</td>
<td>17.18</td>
<td>7</td>
<td>23.03</td>
<td></td>
</tr>
</tbody>
</table>

These 94 Beri-Beri cases were all undergoing treatment and represented all stages of the disease; the whole 94 cases
(all of them Chinese) were smokers of 
opium, or tobacco, or tobacco impreg-
nated with a small quantity of arsenic.
From these figures I am unable to agree 
with Bentley's observation, while it seems 
probable that if there usually was much 
inflammation of the pharynx it would 
also be extended to the alimentary system 
in general—producing diarrhea and va-
tious digestive troubles—which are not 
found.

About the only digestive trouble which 
patients complain about is the feeling 
of fullness after taking even a small 
quantity of rice; they state that eating 
produces a ball in the stomach, follow-
ed by distension, shortness of breath, and 
emissions of gas by the mouth. On percus-
sion a certain amount of flatulence is 
detected, but not sufficient to account 
for the symptoms complained of.

The bowels are regular or slightly consti-
vociting seldom occurs, except towards 
the end, it then consists not only of the 
food taken, but of considerable quan-
Sticky of a sour-smelling fluid— and is always to be looked upon as a most grave symptom, usually indicating that death will take place in a few hours; however, otherwise, the patient may appear at the time. There is nothing special to note about the rest of the alimentary system.

Hæmopoietic System. Bekelhaïing and Warkler made an exhaustive investigation into the properties of the blood, with a view of detecting the pathogenic organism of Beri-Beri, but with practically negative results. Personally, I was not in a position, from lack of time and apparatus, to attempt any scientific work in this direction.

Circulatory System. This is almost invariably affected to a greater or less extent, and careful attention has to be paid to the cardiac sounds, and pulse, both for the purposes of diagnosis and prognosis.
The patient usually complains of palpitation, increased on the slightest exertion, with occasional attacks of dyspnoea and faintness, hot flushings and cold hands and feet. On inspection the area of pulsation is seen to be much increased, the apex beat is displaced outwards and downwards, till it may be even beyond the nipple line, while abdominal pulsation in the region of the ensiform cartilage is extremely common. The pulsation of the carotids in the neck can be well seen and felt, while the radial pulse, even when the patient is at perfect rest, is found to be rapid and small, varying between 90 and 120 per minute.

The area of cardiac dulness is enlarged, chiefly, at the expense of the right ventricle, in all directions.
The following figures show the condition of the heart in 175 consecutive admissions (Chinese) into the poliose Hospital from January 1st 1892 to February 16th 1892.

<table>
<thead>
<tr>
<th>Total No. of hearts examined</th>
<th>175</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot; &quot; &quot; affected</td>
<td>35, 20</td>
</tr>
<tr>
<td>&quot; &quot; &quot; Beri-Beri cases</td>
<td>32, 18.28</td>
</tr>
<tr>
<td>&quot; &quot; &quot; &quot; affected</td>
<td>19, 57.37</td>
</tr>
</tbody>
</table>

Cardiac Lesions found in Beri-Beri:

<table>
<thead>
<tr>
<th>Lesion</th>
<th>No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral systolic leak</td>
<td>19</td>
<td>57.37</td>
</tr>
<tr>
<td>Aortic</td>
<td>1</td>
<td>3.12</td>
</tr>
<tr>
<td>Tri cuspial</td>
<td>4</td>
<td>12.30</td>
</tr>
<tr>
<td>Carotid pulsation</td>
<td>4</td>
<td>12.30</td>
</tr>
<tr>
<td>Intermittent pulsation</td>
<td>3</td>
<td>9.37</td>
</tr>
<tr>
<td>Irregular pulsation</td>
<td>3</td>
<td>9.37</td>
</tr>
<tr>
<td>Accentuated 1st sound</td>
<td>2</td>
<td>6.25</td>
</tr>
<tr>
<td>&quot; &quot; 2nd</td>
<td>8</td>
<td>25.00</td>
</tr>
<tr>
<td>Reduplicated 1st</td>
<td>4</td>
<td>12.30</td>
</tr>
<tr>
<td>&quot; &quot; 2nd</td>
<td>5</td>
<td>15.60</td>
</tr>
<tr>
<td>Epigastric pulsations</td>
<td>4</td>
<td>12.30</td>
</tr>
<tr>
<td>Truffled &amp; Distant sounds</td>
<td>9</td>
<td>28.12</td>
</tr>
<tr>
<td>Shrinking sounds</td>
<td>4</td>
<td>12.30</td>
</tr>
</tbody>
</table>
From this it will be seen that whenever there is a cardiac lesion in Beri-Beri, it is invariably, apart from other conditions, a mitral systolic murmur, due in the vast majority of cases to a dilated right ventricle with a disparity in size of the mitral valve—though at times it may be purely coronary in origin.

Respiratory System. With the exception of a peculiar bleating or somewhat nasal voice, which is found in a considerable number of cases and when met with is almost diagnostically of the disease, the respiratory system is primarily never affected. In the milder forms of Beri-Beri, and when the heart is enlarged and a valvular lesion present, it naturally follows that there is considerable edema of the lungs with moist sounds scattered throughout. While later on, in such cases, the pleural cavities become choked with serous fluid, as much as 8 to 10 ounces in some instances (as § 27 XVII), the breathing becomes laboured with all...
muscles of extra-ordinary respiration brought into action, and the patient, with a cyanotic bloated and distressed face, succumbs to asphyxia.

**Integumentary System.** The skin as a rule is harsh and dry, though a moist skin may be met with in the wet forms of the disease.

In the extreme stages of the moist forms the skin often cracks, where it folds on itself, as in the distended and twisted penis in the groin and in the neck, from sheer overdistension.

I have never seen any characteristic eruption such as some observers have met with.

**Urinary System.** This is practically normal; the urine is acid, the Sp. Gr. about 1020, while I have never detected albumen or any other abnormal constituent; it is passed in considerable quantities, and there is never any retention or frequent micturition.
Reproductive System

As in locomotor ataxia there is loss of sexual desire, and sometimes even of ability to have an erection. In the female there is nothing special to note, except that as the case progresses towards death the catamenia become scanty, lose their normal red colour and finally stop.

Nervous System.

This is the most important of all the systems to pay attention to, but unfortunately, owing to the peculiar characteristic of the Chinese in being able to stand a great amount of pain without complaining, and to their inability or dislike to express their real feelings, it is extremely difficult to get an accurate account of their real condition—especially of the subjective phenomena. So say nothing of the difficulties one contends with in having but an indifferent interpreter.
Sensory Functions. Sensation is invariably affected at some stage of the disease, and is usually amongst the initial symptoms; the patient complains of a pain, often of an intense and exacerbating character, in the calves of the legs, with pain also in the soles of the feet—but not of such a bad character.

In such cases, owing to the agony produced, it is impossible to apply any pressure to the gastrocnemii, but even those who have but little pain on walking or sitting suffer greatly when these muscles (which are felt to be brawny & rough) are compressed.

While the pain is chiefly found in the muscles of the leg it is also present, but to a lesser degree, in those of the thigh; the penis, scrotum, abdomen and muscles of the back are not affected, but over both pectorals anteriorly the pain is often so great that one can only apply the stethoscope with care. The muscles of the upper extremities are affected to some degree, which as a rule is only well marked.
when pressure is made on the interosseous space between the radius and ulna.

I have never heard any patient complain of muscular pain of the head and neck.

There is some alteration in the sensibility to heat and cold, chiefly in the direction of not feeling heat, but this is so difficult to determine with a clinician that not much value can be placed on it as a symptom.

Tremulation over the dorsum of the feet may be met with, but it is uncommon; I had one case, a Tamil, who complained of a burning sensation of the soles of the feet; he had some symptoms of Beri Beri, but after watching him in the wards for over two months, I came to the conclusion that he was malingering, being anxious to have his agreement as a coffee coolie, an unhealthiestate cancelled.
Mummehess is met with in the soles of the feet, so that the patient complains he cannot feel the ground when he walks—so that his gait is rendered unsteady; it is common in the lower extremities and is met with in the fingers, especially the ring and little one; there is also a feeling of "pins and needles," and a dull aching pain in the deep muscles near the long bones.

The numbness or tingling soon gives way to anæsthesia of the skin, more or less complete in nature, in scattered patches throughout the body, especially well marked in the lower extremities and forearm; I have not seen a case of anæsthesia of the lips, though Bentley mentions several, and Simons says it is common in Japan—and thus I bear out Franson and Pekelharing's observations.

I cannot say that my attention has been specially drawn to the tenderness of the nerve trunks, as some writers describe.
Cramps in the calf and thigh are often met with, and cause great pain, but they cannot be compared with the cramps in cholera, nor do the muscles stand out in knots in these paroxysms.

In one case there was a slight tendency to opisthotonos with clenching of the fingers; in another, a Siamese man in Bangkok in 1897, the slightest touch threw the patient into an almost cataleptic condition, so much so in fact that when I first saw him I thought it was a true case of tetanus; unfortunately I was only able to see this last case twice, under most unfavourable circumstances in a small native house, so that beyond satisfying myself that it was a true case of Beri-Beri I was unable to make any notes of his most interesting condition.

The other senses of taste, smell, and hearing are not affected, though most Celestials are slightly deaf owing to the native habit of cleaning the wax out of the ears with sharp painted sticks.
Slight.

Cedema may be seen in the lids and
in fact in some cases of wet Beri-Beri
it is often so extreme as to cause the
palpebral fissure to assume the form of
a mere slit.

Ptosis was noted in one case. Paralysis
or palsy of the motor nerves of the
lids or globe have been noted by other
observers. I, myself, have noted nerve
injuries in one case, and a slow canulac.
ing atrophy of the optic nerve in another
case.

Motor Functions. I have never seen
any case in which any of the various
organic reflexes were primarily affected.
The skin reflexes are nearly always
more or less affected, especially those
of the abdomen and lumbar region,
which are early diminished and some-
times entirely lost.
The tendon reflexes are most important, especially those of the patella, in the very early stage of the disease, so early that it is seldom met with or if so is difficult to diagnose, this reflex is undoubtedly exaggerated— but this exaggeration may last only for two or three days, is followed by rapid diminution and soon total loss.

This early abolition of the patella reflex is a most important diagnostic point— and is often found in patients in whom the characteristic gait has not yet developed, and who only complain of slight pains and numbness in the leg. Once abolished it will take months or years to return— in fact I have never yet seen one return though I have watched cases carefully for months, and have seen them periodically even after they were discharged from the hospital as "cured."

Next in importance is the ankle drop with pointed foot and toes, which is well seen only at a later stage:
Brisk drop, too, is very common, especially in old standing cases of the dry variety, and is most persistent. The cremasteric reflex may be completely lost—but this is somewhat uncommon.

The various other reflexes are seldom affected.

In-co-ordination of the lower extremities is always well seen in well-marked cases, and helps to give rise to the characteristically peculiar walk in those affected with the disease—as will be noted later on.

I have had no opportunity of going into the question of the electric irritability of the muscles and nerves, but Peckham and Wrinkler have gone into the subject exhaustively, and claim that they can by it diagnose a case of Beri-Beri. When even the patient himself feels scarcely indisposed, ... and often before he has any distinct symptoms of oedema. They add that they are, in regard to...
quantity as well as quality some important variations in the muscular and nervous reactions, which, if they cannot all be ranked in the reaction of degeneration, can nevertheless, taking the words in their widest signification, almost invariably be so classed.

Vasomotor Disturbances are seen in the coldness of the extremities, the peculiar shiny pallor of the face in the wet variety, the edema, and the cardiac symptoms.

The nutritive functions are not much interfered with; in some cases bedsore form, but it is astonishing how seldom they are seen considering that patients often lie for months on a bare plank bed covered only with a thin Chinese mat; when they are seen it is almost only in the advanced stages of the moist variety.

Perspiration is usually diminished, so that the typical goose-quill skin is not at all uncommon.
The cerebral and mental functions are never affected.

Locomotory System.

Pains in the bones and joints, deep-seated, are often complained of; in fact, it is usually the reason the patient goes to hospital. He describing it merely as 'wind.' (Chinese "wunghhee," Malay "angin," Siamese "lone").

Effusion into the knee joints is commonly seen, but not to any great extent; the surrounding puffiness of the joint often makes this difficult to determine.

The mobility of the joints is often most striking, especially the lateral movement of the ankle; this is probably due not only to the relaxed external ligaments, but in a great part to the atrophy of the surrounding muscles and tendons; an holding up the patient's leg and shaking it, the foot "waggles" in a strangely helpless fashion.
The various conditions of the muscles have been already dealt with, and their placidity, twitchings and atrophy are noted.

The gait is pathognomonic of the disease: the foot is lifted high off the ground so as to prevent tripping over the toes, and jerked forward in a helpless style, the toes reaching the ground first, the sole and heel following with a kind of "flop": the base is a broad one, even up to 18 inches, and the patient often can't walk at all without the support of a stick which he places in front of him to make a triangular base of support, with the body bent forwards over it.

Unlike locomotor ataxia he can stand with his feet together and eyes shut; he turns slowly in a large area with his eyes fixed on the ground.

The attitude is well seen in the accompanying photographs.

The locking of the knee joint with a bowing backwards of the leg is most characteristic.
Case xxix

Meri-Meri (dry)

Along Law at 34. Gambier booty
Admitted 12. 12. 93.
Absconded 25. 12. 93.

Fell, rather slight, Chirac, man of over 60 years of age, complained of both legs, pain on pressure in calves, numbness over loins and hands. Expression natural. No giddiness. Some precordial distress. Fullness of stomach after meals. Reflexes diminished.

On admission had a racing heart, irregular and intermittent, too rapid to make out any bruits; pulse was too small, irregular and difficult to feel to allow of counting; some difficulty heard to beat about 180 per min.

He was put on a mixture containing five minim of tincture of digitales, and liquid extract of ergot, every four hours; which two days later was added tincture of nux vomica ten minims, with an additional ten minims of digitalis.
17th December 1893. Pulse 60, regular, mitral systolic bruit; on waking up the ward heart immediately became irregular, intermittent and rumbling, resembling the uneven racing of a ship's engines when the screw comes out of water.

Reduce the digitalis to m7 thrice daily.

22nd December 1893. Pulse 60, mitral systolic well marked; after running a little Pulse 120, irregular, intermittent heart beats not reaching the radial pulse.

Reduce the digitalis to m7 thrice daily.

24th December 1893. Pulse 48, hard high tension; heart sounds irregular and intermittent; even while sitting quiet; irregular and intermittently increased on exertion, when pulse rises to 100 but quickly falls again to 48.

Heart to-day resembles the firing of a bullet at a target - the 1st sound resembling the explosion of the cartridge, the burst the whistling of the bullet through the air, and the sharp clear
2nd round the "ring" of the bullet against the target.

Stop Digitalis entirely.

Spleen slightly enlarged. Reflexes diminished. No special gait. calves still tense, but patient says they are much better. Swellness of legs much improved since admission. Fusses slightly congested. No edema.

25th December 1893. Patient felt so much better that he absconded before the morning ward visit.

Remarks:

This is an undoubted case of Beri-Beri in which the chief symptom was the marked cardio-inhibitory action of the vago, completely throwing the heart out of gear; under heavy doses of Digitalis and 

Vermic the heart regained its normal action to a considerable extent, but before any permanent good could result the patient absconded.
Pathology

The general post-mortem appearances found in those cases already detailed are as follows:

Anaemia, in moist cases, is universally present in all the tissues and organs to a more or less degree, in the very dry forms the tissues may appear abnormally dry and pale, but one is often surprised to find even in such cases a considerable quantity of serous fluid in the abdominal cavity, which is probably a change which occurs shortly before death and is not diagnosed during life (Case No. XXVIII. shows this well).

Echymoses: have been found as petechial patches under the viscidal layer of the pericardium - and Mausser describes similar ones under the pleura, and about the serous coat of the abdominal viscera; haemorrhages may also be found between the muscles and the sheaths of nerves.
I have not seen minute hemorrhages in the brain which have been described by some authors.

The blood is found to be remarkably fluid and dark in color, while firm coagulations are rare: in many of the cases noted ante-mortem clots were well seen in all the cavities of the heart, and especially in the right ventricle.

The muscles are atrophied or swollen, brownish red or pale red, according to whether the affection is in an early or late stage.

The lungs are usually edematous, and as death so often occurs from cardiac failure they are often found to be engorged with dark red blood; ante-mortem clots may be found to extend from the heart into the pulmonary artery and vein. Fluid is generally found in the pleural cavities. In one case the fluid was sanguineous.
Pericardium: a good deal of fat may be scattered over the anterior surface. The pericardium is invariably present in every case, and varies from \( \frac{3}{7} \) to \( \frac{3}{17} \). I believe the latter amount (Case No. Xx) is the highest yet recorded.

The heart is usually much enlarged, especially the Right Ventricle, and is often covered with a thick layer of yellow fat. The muscle tissue in general is changed in appearance; fatty degeneration is the rule, and the muscle is pale but not usually anaemic.

Cavities of heart. Right auricle always dilated, with thin walls, and usually contains ante-mortem clots in one case (No. 25) its walls were so thin that they seemed to be on the point of rupturing; the muscular tissue being almost absent and the wall consisting only of the visceral layer of the pericardium through which the pale muscular papillae
were plainly seen from the outside.

Right ventricle: here dilatation is most marked, and in many cases is extremely so; in no case have I seen hypertrophy, muscle degeneration always shows itself more clearly in the walls of this ventricle than in any other portion of the heart muscle.

Left auricle shows nothing remarkable.

Left ventricle is often somewhat dilated. Hypertrophy is present in most of the cases—in some markedly so: the muscle tissue itself is usually normal in appearance to the naked eye.
Abdominal Cavity

Nothing characteristic is to be noted except that ascites is usually present. In some cases the spleen was found to be enlarged, which seemed to be due to engorgement.

Nervous System

With the naked eye nothing abnormal was noted in the brain and spinal cord, though here, as elsewhere, ample fluid serous was common.

Owing to press of work and the absence of proper facilities microscopical investigation of the peripheral nerves and other tissues was not carried out; these, however, have been worked out in detail by Bailey, Pekelharing, and Winkler.
Beri Beri Jaunt.

Man of Lang. at 37. Gambier C.,
Admitted 11th December 1893
Disease Ulcer right foot
Died of Beri Beri 14th February 1894.

Well, tall, muscular man. Sent to Depot and paid close to Beri Beri quarter hill.

28. 1. 94. Left eye punctiform keratitis
1. 2. 94. Fortic Injuries
1. 2. 94. Puffiness of abdomen, slight oedema of chest, face puffy and swollen especially lower eyelids; slight oedema of legs with some numbness. Initial collapse brisk.

No characteristic gait or calf pains.

14th February 1894. General oedema, especially of chest and head and neck, increasing. Heart sounds very distant. Hydropericardium. Dull - dull note extending to right border of sternum.

11. 45 am. Temperature 98° but felt hot; patient looks ill, but beyond some prostration, dyspnoea does not complain; some shortness.
14th February 1894. 8.45 am. Patient lying on right side on floor (since 6 am) in great distress, moaning. Respiration 44. Intercostal muscles greatly indrawn at each inspiration. Face bloated, both eyes very oedematous, the left being completely closed. Lips blue and cyanosed, body cold, pulse imperceptible, heart sounds obscured by heavy breathing; quite sensible, referring pain to pit of stomach.

8.53 am. Died immediately after speaking, sensibly and asking to be dry-cupped. As he died a considerable quantity of yellowish fluid gushed out of the nose and mouth.

Liq. strychn. on X injected hypodermically. Tabloid of nitro-glycerine given by mouth, any cupped and mustard plaster 10 hours before death.

Post-Mortem

14th February 1894 10.15 am (one and a half hours after death).

Head and neck and abdomen greatly swollen, very slight oedema over tibia; eyelids extremely congested; lips blue.

Chest wall fatty, serous fluid oozed out in considerable quantities on incision.
Lungs: Both healthy, no adhesions, emphysema with watery, swollen & fluid and black blood-waterlogged; considerable effusion into both pleural cavities.

Pericardium: slightly fatty an outer layer contained 17 ounces of clear serous fluid.

Heart - vessels over it distended and tortuous; organ enlarged, somewhat flabby; Right side dilated, left side normal except valves which was somewhat hypertrophied. No blood clots, or signs of endocarditis.

Spleen enlarged and congested.

Ascites about 80 ounces.

Other organs normal as well developed.

Remarks

Note that patient complained of nothing till within 3 hours of his death which was caused by asphyxia from edema of the lungs.

Hydropneumothorax - 17 ounces highest recorded.

No ante-mortem clots from rapidity of death, no post-mortem clots because Post-mortem tone was one and half hours after death when body
was still warm.

Only those well acquainted with the disease could have diagnosed it, and have looked for a sudden end for patient did not know that he was seriously ill.

Contracted in Hospital after six weeks residence.

Diagnosis

In a malarial country the idea of Beri-Beri ought never to be out of one's mind when a patient complains of any symptoms in the lower extremities referable to a symmetrical nervous lesion; if there is pain or numbness of the muscles of the calf, with a feeling of weakness in walking or standing, and edema, however slight, over the tibio-posterior, a provisional diagnosis can be rapidly made which can be confirmed at the time or very soon afterwards by finding the patella reflex absent or greatly diminished; the pulse abnormally rapid, and some peculiarity of the heart—such a mitral bruit,
enlarged area of percussion or a beating and thumping action.

The only difficulty which really presents itself is to distinguish it from malarial neuritis - the two diseases being thus compared.
Malarial Peripheral Neuritis

Anæmia usually marked—always present.

Heart symptoms, if present, are not very marked, and are usually due to the anæmia.

Spleen usually enlarged

Local œdema rare

Serous effusions very rare.

Fever precedes neuritis for a long time and often accompanies it.

Fever distinguished by Malays and Malay-speaking races as "djamam; malarial fever and ague.

Symptoms practically never acute in onset. Incubation period usually of some months' duration.

Beri-Beri

As often absent as present.

Heart symptoms an important and almost constant feature.

Uncertain. Present at one time at least in the history of the case, often chronic.

A common cause of death. Fever need not precede neuritis for long, but often accompanies rapid onset of symptoms. Called simply "pans" = warm or hot, as distinguished from fever and ague. Often acute.

Often apparently only a few days.
Symptoms often show striking recrudescence of a daily persistent type. Gait of a shuffling type in paresis or paralysis.

Malarial
During attacks of fever plasmodium may be found cases readily cured, Quinine + Arsenite curative

Paralysis does not show nightly increase as in multiple malarial neuritis. Gait equinice and typical i.e. with loose knees, ankle drop, broad base.

Beri-Beri.
Absent, unless malaria present as a complication sudden death commonly. Not so in Beri-Beri.

Prognosis.
Prognosis is always uncertain and usually grave; one sees cases showing but few symptoms die in a few hours (case No.) while others who are completely waterlogged, or whose muscles are atrophied and paled to an alarming extent, gradually begin to recover when all hope is practically given up. Precordial anxiety, with extension of dulness of the cardiac area, and dyspnea are grave symptoms, while vomiting, especially of a quantity of fluid as in cholera, almost always points to impending death - in fact when this latter symptom comes on I have not seen a case recover.
The mortality is difficult to estimate and can only be judged approximately from the cases one meets within Hospitals and Gaols.

My experience is follows:

### Johore Gaol Hospital

<table>
<thead>
<tr>
<th>Years</th>
<th>Total Admission</th>
<th>Total Died</th>
<th>Beri-Beri Admitted</th>
<th>Beri-Beri Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>1888</td>
<td>53</td>
<td>5</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>1889</td>
<td>84</td>
<td>7</td>
<td>13</td>
<td>5</td>
</tr>
<tr>
<td>1890</td>
<td>93</td>
<td>4</td>
<td>15</td>
<td>2</td>
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<tr>
<td>1891</td>
<td>162</td>
<td>8</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>1892</td>
<td>226</td>
<td>11</td>
<td>26</td>
<td>9</td>
</tr>
<tr>
<td>1893</td>
<td>277</td>
<td>15</td>
<td>57</td>
<td>12</td>
</tr>
</tbody>
</table>

| Total  | 895             | 50         | 127                | 54             |

= 558 = 26.77

### Johore Government Hospital

Beri-Beri Admissions only

<table>
<thead>
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<th>Year</th>
<th>Admitted</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>1888</td>
<td>57</td>
<td>21</td>
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<tr>
<td>1889</td>
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<td>1890</td>
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<td>120</td>
<td>35</td>
</tr>
<tr>
<td>1893</td>
<td>180</td>
<td>35</td>
</tr>
</tbody>
</table>

| Total | 508 | 131 = 25.78 |

= 508 = 131 = 25.78
Treatment

Prophylaxis. Beri-Beri being undoubtedly closely allied in some respects to malaria it follows that the prophylaxis is the same. Houses ought not to be built over stagnant swamps, or in the neighbourhood of soil which is being constantly filled for the purposes of cultivation, such as in a gambier and coffee plantations: they ought to be raised on piles not less than five feet from the ground, and the rooms ought to be freely ventilated and over-crowding avoided. A hospital ward or ships forecastle once infected ought to be kept rigorously clean with white wash and tar, and Beri-Beri patients isolated by themselves.

The diet ought to consist largely of fresh meat, vegetables, fish, and wheat flour—in preference to the almost exclusively rice and salt fish so common among Asiatics.
Once a patient is infected he ought promptly to leave the Beri-Beri area, and if possible go for a sea voyage; colds, chills, and exhaustive work ought to be avoided and the patient live a regular and quiet life.

I have met with no drug that is anything like a specific; broadly speaking it may be said that the best routine treatment is a tonic composed of iron, quinine and strychnine: of the latter I have constantly given in beer, of the liquor thrice daily with good results. The galvanic battery and massage ought to be used daily.

The various symptoms must be treated as they arise: thus, blistering, dry and wet eczema over the heart for precordial distress and forming hydropericardium; nitroglycerine for sudden and grave cardiac danger I have not found of much value.

Paracentesis abdominis for ascites I have repeatedly performed in the same patient with temporary relief only.

Cathartics, in the moist forms, of caustic and ipecac, gamboge, creton oil but especially...
of Puls. Jalap. &c. (the latter in doses up to two drachms daily) are most useful.

Digitalis and strychnine or mixtures are most useful in steadying the heart.
Diuretics and diaphoretics are at times of great value.

Papping the pericardium is so difficult to do, when the heart is heaving and tumbling, that I have never felt justified in attempting it, especially since no successful cases have been recorded.

I hereby certify that the above thesis has been composed by me.

Florence Nightingale

in B. C. in 1870

H. B. M. Legation
B. angrek
Swan
The following original photographs were taken by me in Phnom in 1872 & 1873; unfortunately, most of them, together with the negatives, have been spoilt on account of the hot moist climate.

**Beri-Beri.**

The general expression and edematous condition of the body are characteristic.

**Beri-Beri.**

The triangular base of the foot and firmly set legs are well seen.

**Beri-Beri.**

Diagnostic characteristic gait. The downward gaze, the balancing effort with the arms, the high elevation of the right leg, the locked left knee joint, and the firmly planted left foot.

**Beri-Beri.**

Ankle and wrist drops; the left hand also shows the wrist drop, but is not seen here, as it is used for support.
Beri-Beri.

Notice, in the first the high outward and uncertain action of the right leg, with the balancing effort of the left arm, in the second the landing on the outer side of the right foot, and in both the locked knee joints with the eyes fixed on the ground.

Beri-Beri.

The firmly locked knee joints, downward gaze, and short careful gait are well seen.

Beri-Beri.

The broad base, locked knee joints, and general position of the bodies are characteristic.

Beri-Beri.

Note the forward stoop of the body, the eyes fixed on the ground, and the position of the stick as it forms a broad triangular base for walking.
Beri-Beri
The locked knee joint (right),
high action of the left heel,
and position of the stick are
well seen.

Beri-Beri
Note the well-marked ankles
and wrist drop in the adae-
matus stage; cannot move
or even sit up right without
support.

Beri-Beri
The broad base of support
is well seen in all these
cases.

Beri-Beri
Instantaneous photograph of a
convalescent patient walking.
Note the broad base (knuckles)
on which he walks, the high ac-
tion of the right leg, the left look-
ed knee joint and the general
balancing efforts of the body.
Beri-Beri.
Note the high action of the right knee and leg, the balancing efforts, and the downward gaze. In the second picture the locked knee joints are well seen even when the patient is supported against a pillar.

Beri-Beri.
Shows the characteristic gait. Note the firmly locked flexed knee joints of the foremost patient with the bowing back and side of the leg.

Beri-Beri.
Note the general attitude, and the broad base of gait.

Beri-Beri.
The locked knee joints, feet wide apart, and forward stoop of the body are well seen.

A group of Beri-Beri patients.
A convalescent patient rising off his bed. Note the brace of support formed by the hands and the support of the left leg.

Beri-Beri

Note how he supports himself on his hands with legs wide apart; he is not a patient and is seated naturally.

Beriberi

Side view of the mode of progression of a patient whose legs could not bear the weight of his body; this had been his only method of moving about for the last twelve months on his buttocks and hands.

Beriberi

Front view of patient whose sole mode of progression is on his hands and buttocks.