Thesis for the Degree of M.D

Beri - Beri.
Introduction

I choose Beri-Beri as the subject of my Thesis on account of the frequency with which the disease is encountered in tropical and sub-tropical countries. Beri-Beri has been recognized since the seventeenth century as a distinct disease but comparatively little has been done in elucidating its true nature.

The difficulties which beset one in making a research in the tropics, are numerous, for not only have we to speak a language foreign to ourselves, but also, in many cases, foreign to those from whom we are trying to elicit information. Again, we do not always have at hand the material requisite for carrying on scientific investigation and we are often brought face to face with native prejudices, such as that of granting a necropsy.

This Thesis is based upon 300 cases which have come under my supervision in the course of Hospital practice. The majority of the cases occurred in Chinese coolies working at the Coal Mines in Labuan and Sarawak as well as in coolies engaged in opening jungle in the interior of British North Borneo.
Definition

Beri Beri may be defined as a non-febrile multiple peripheral neuritis, occurring endemically and epidemically, chiefly confined to tropical and subtropical countries and characterised by:

1. Anaesthesia, hyperaesthesia and paraesthesia of the skin.
2. Local or general oedema.
3. Muscular hyperaesthesia and paralysis.
4. Muscular atrophy.
5. A tendency to a sudden termination by paralysis of the cardiac or respiratory muscles or by both.

Etymology

We are still without any definite meaning of the word Beri Beri. Each country seems to have its own name. In Borneo it is generally known amongst the Malays as "sakit lumpoh"; "sakit" in the Malay language meaning illness and "lumpoh" numbers.

In China, as far as I am able to gather,
the disease is placed in the same category as Rheumatism, there being no special name by which it is known unless it be by "pee" which means numbness. To whichever language the word Beri-Beri can be attributed, it would appear that its meaning indicates a prominent symptom of the disease. If derived from the Singhalese, beri means weakness and a reduplication of the word intensifies its meaning, hence Beri-Beri comes to mean intense or great weakness.

The word Beri-Beri as used by the Malays means sheep, but it is difficult to understand the connection between the peculiar gait of a Beri-Beri patient and the gait of that animal.

We now know that Beri-Beri is similar to the disease Kakhe of Japan.

Geographical Distribution

Beri-Beri has its habitat in tropical and sub-tropical countries. It has been met with in the temperate zone. The disease is more...
prevailing in the eastern than in the western hemisphere.
Beri-Beri prevails endemically, more or less,
throughout the Malay Peninsula and the Malay Archipelago.
In Borneo, Java and Sumatra, Beri-Beri claims
many victims and in Borneo some ten years ago, it caused such tremendous havoc amongst
the coolies that estates were closed and trade
almost paralyzed.
In Malaya, the disease is endemic, confined
mostly to the Chinese coolies and causing a
death-rate second to malaria.
In the Celebes, Moluccas and New Guinea
severe attacks are frequently experienced.
Beri-Beri is also known in the Sulu Island,
Mindanao and the Philippine Islands.
In India the disease occurs along the
Malabar and Coromandel Coasts and in
the neighbouring island of Ceylon.
It is present in Burmah, Siam and Indo-China.
In the Federated Malay States it is endemic
and epidemics have occurred at Singapore.
In China, the disease occurs chiefly at the
coast ports, notably Shanghai, Foochow, Amoy.
In Japan the disease is endemic and under the name of Kakki was responsible for a high death-rate in the Japanese navy. Beri-Beri has been met with in the South Pacific Islands. The natives and Chinese coolies of Australia have suffered from it. The East and West Coasts of Africa have experienced sharp attacks and South America is not immune. Beri-Beri has been known to break out in epidemic form in hospitals and barracks where a large number of persons are congregated together. Ships calling at ports where the disease is common, may, after many weeks, show cases amongst the crews and it is in this way that most of the patients in European hospitals come. Epidemic waves appear in countries where the disease is endemic.

The distribution, therefore, of Beri-Beri might be considered as almost universal; many diseases which were registered in former
days as cardiac, renal, material and obscure nervous are now recognized as
Keri-Keri.

**Symptomatology**

I have thought it advisable before entering into a detailed analysis of Keri-Keri, to
record six cases, giving the notes of each as they were taken at the time each patient
was under observation. The notes are not so abundant as they might have been
but pressure of other duties prevented me from entering into as full an account
as I should have desired. It is quite
impossible to trust to one's Chinese dressers and apothecaries for accurate information.
I hope, however, that the cases recorded will
show a general outline of the disease as it occurs to one engaged in hospital
practice.

Of the six cases recorded, three recovered and
three died. They show clearly how uncertain
the course of the disease is.

In one case (case 6) the disease was of long
Standing, yet there was every sign of recovery but sudden symptoms set in which proved fatal.

Case 5, of equally long duration as case 6 and perhaps of a more severe type, recovered and the patient was able to continue his occupation.

Cases 3 and 4, of almost equal duration as regards hospital treatment, took on different symptoms, the one dying and the other recovering.

Cases 1 and 2, of different duration and working in a country together under the same conditions and entering hospital on the same day with a somewhat similar history, shewed a marked contrast in the ultimate result: the first dying after a short stay in hospital, the second able to withstand the poison and ultimately recovering.

Case No 1.

Cham Wing, male, age 26. A Cantonese coolie engaged in making a new road through the interior of British North Borneo.
Admitted into Habana Hospital 11th December 1895
Died 20th December 1895.

History:—Patient states that he has been ill for one and a half months and that he had to stop work on more than one occasion on account of stiffness in his legs. There was also some swelling of his feet but this disappeared when he kept quiet. About three weeks after the stiffness came on, he experienced a feeling of numbness in his feet and legs, and some little time after, numbness appeared in the fingers and hands.

State on admission:—There is numbness from fingers to wrists and from the toes to the ankles. There is no oedema. There is pain on pressure of the muscles of the calves of the legs. The muscles are hard and firm. The throat is dry and inflamed. Spleen much enlarged. There is tightness and pain in the chest and some discomfort in the epigastric region. The eyes are painful at night. Knee-reflexes are lost.
Patient is able to stand and walk. He can flex his feet and move his toes but cannot stand on tip-toe.


Progress of Case:

16th Dec. 1895. No numbness has increased and there is some in the chest.

20th Dec. Some oedema has appeared on the dorsum of feet. The pain and tightness of chest is worse.

23rd Dec. Complains of shooting pains in shoulders and arms, but worse at no special time. Throat is painful. The condition is not improving. Appetite bad.

30th Dec. Feet are more swollen. Patient suddenly attacked with vomiting, pain and distress in chest and difficulty of breathing. He is very restless and tosses about. He died in the evening.

Treatment: Tonic treatment, when bad symptoms set in, digitalis, stimulants or cupping.

Eat: As usual scale with a diminution in the quantity of rice, beans being substituted.
A post-mortem examination was held two hours after death and the following were the chief points noted.

Here is slight swelling of chest and abdomen.

Slight bluish discoloration of skin on back.

Chest: Right pleura adherent to chest wall by recent and old adhesions. Left pleura also slightly adherent.

Heart: 2 oz. of fluid in pericardial sac.

Both auricles and ventricles and large bloodvessels filled with dark fluid blood.

Abdomen: Liver congested and extending slightly below the ribs. The left lobe touching the spleen.

Spleen is congested and enlarged, extending downwards to a point midway between lower ribs and iliac crest.

Case No. II.

Shau Ram, male, age 26, a Bantowee coolie employed in making a new road through the interior of British North Borneo.
Admitted into hospital 11th December 1895.
Discharged cured 6th June 1896.

History: Patient states that for two months he suffered from periodical attacks of fever, which were followed by gradual increasing weakness in his limbs and twenty days before admission into hospital, he felt numbness in his legs and ultimately lost all power of locomotion. He never noticed any swelling. The country in which he has been working is covered with dense jungle and the rainfall is considerable.

State on admission: There is numbness, more or less, over the whole body, with the exception of the back and face. The patient states that it began in the toes and gradually extended upwards affecting the legs, thighs, abdomen, chest and arms until it reached the neck. There is no oedema of the legs and patient has never noticed any.
The skin of the body has a yellow waxy look and that of the legs feels drawn and tight.
There is a tingling sensation in the hands and occasionally paresthesia is present.
There is pain in the muscles of the leg which is much intensified by pressure.
The muscles of the forearm also are painful.
The superficial and patellar reflexes are absent. Although no tightness of the chest is complained of, there is a certain amount of dyspnea, worse during the day and when lying down. Palpitation is present but beyond this, there is no cardiac trouble.
There is fullness and distension in the epigastrium which is increased after meals.
Patient cannot stand without support and is quite unable to walk and lies in bed on his back a helpless mass.
Tests to heat & cold of leg. The sole of the foot can distinguish warmth but not cold.
Inside of leg to thigh normal. At ankle and lower third of leg on the outside, warmth feels cold and cold feels warm; other parts normal. On the dorsum of the foot, close to the toes, the sensation to cold is
normal, but that to heat is impaired. When tested with a pin, a patch situated about the middle third on the outside of the leg is insensitived.

Right leg: The role of the foot is normal to cold. Warms cannot be recognized.

Dorsum of foot normal. On the inside of the leg extending from the ankle to just below the knee, heat and cold cannot be detected; above this, sensation is normal. Outside of leg recognizes cold but cannot recognize warmth.

When tested with a pin, all parts are normal except the role of the foot which is anaesthetic.

Progress of Case:
26th December 1895. The general condition has improved.

2nd January 1896. The numbness and pain in hands and feet is lessened. The tingling sensation of fingers is less. The chest does not cause much trouble.

16th January. The fingers can be straightened. The numbness in its upper part of the body has gone.

17th January. Patient has a severe headache.
otherwise the condition is the same.

25th January. The numbness is more severe in the legs and there is tightness behind the knees. Patient is not so helpless, being able to use his hands.

4th February. Improvement. Numbness and tightness is less. Patient can stand but he requires support.

11th February. There is much tightness and weakness of knees. The pain into muscles of the legs is not so severe.

14th February. Patient has a rise of temperature and numbness and pain of muscles are rather increased. Knees are complained of as being very weak.

18th February. Had several watery motions mixed with blood and mucus. Vomiting. Pain in legs less.

26th February. Motions less in number and thicker, and there is an absence of blood. Vomiting ceased. There is little or no pain in the abdomen. Numbness is greater.

5th March. Numbness and pain in legs less. Patient is able to walk about with the aid of support and has greater
confidence in himself when standing.
17th March. The use of the hand is nearly restored. Indurations in the gastrocnemius muscles can be detected.
28th March. Numbness only complained of in the toes and outer sides of the feet.
Tightness of muscles has almost gone.
Chest causes no trouble.
4th April. Improvement is steady. There is still some tightness behind the knees.
Numbness have quite recovered.
25th April. Numbness only present in the toes.
Patient is walking well.
3rd May. Tightness behind the knees is rather greater and numbness extends to the heels. There is no pain in the legs.
8th May. Tightness is better. Left foot is slightly painful.
22nd May. Numbness of feet has nearly gone and tightness is only complained of in the morning. Patient can walk without the aid of a stick.
3rd May. Patient is improving rapidly. Tightness and pain have gone and patient can go up and down stairs.
6th June. Patient appears to be quite well and is discharged cured.

**Treatment:**

Magnesium sulphate was given in the morning when occasion called for it. Iron, Arsenic and Strychnine as tonic treatment.

Liniments rubbed into legs gently to relieve the muscular pains.

**Diet:** Milk. Rice replaced by beans to a large extent. Fish and meat.

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**Temperature Chart of Case II**

[Graph showing temperature readings over a period of days and months.]
Case No. III.

No. 49. Male. Age 27. Bootie at Huara coal mines.

Admitted into hospital 20th January 1902.

Died 14th February 1902.

History: Patient states that fourteen days before admission (i.e. 16th Jan 1902) he suffered from fever, which, after an interval of five days was followed by weakness in his knees and by pains in his legs; shortness of breath when walking and a feeling of tightness in his chest; numbness extending upwards from toes to hips and from finger tips to the shoulders.

State on admission:

There is numbness in his legs extending from his toes to his thighs and from the finger tips to the middle of his arms. There is also slight numbness of his face. The muscles of the calves of the legs are tender to pressure, but when lying down and keeping quiet, the pain is not very great. The patient is able to stand and when walking, complains of weakness in
his knees and is uncertain about himself.
not caring to trust himself far without
support in case he should lose his balance
and fall. There is a sense of fullness
in the epigastrum, more marked after taking
food and this causes difficulty in breathing.
His tongue is clean and the appetite is good.
His bowels are moved every other day.
Urine normal. Temperature 99°F. Fahl.
Progress of Case:

5th February 1902. His condition of the patient
is the same as on admission, except that
in addition, there is some numbness in the
chest.
7th February. Patient feels weaker. Walking
is very irregular and has more of the
characteristic beri-beri gait about it.
12th February. No pain in the muscles of the
 calves of the legs is very severe, and when
the muscles are pressed, the patient cries out.
Walking causes extreme pain. There is
considerable tightness in chest and great
difficulty in breathing.
18th February. Patient states that the numbness
is "all over his body."
No tightness in chest and difficulty in breathing are more severe. No heart is beating violently and pulsation in the neck is visible. The cardiac impulse is diffuse and there are signs of failing circulation.

12th February. Patient is suffering from intense tightness of the chest and breathing is laboured. The whole chest being lifted up under great difficulty. Patient is sensible and says that he feels a great weight on his chest. The lips are livid; the hands, feet and nose are cold. The patient died during the course of the day.

![Graph Image]

**Treatment:**

Regulation of the bowels with occasional doses of Mixture Atta. Application of Liquor Equisartian to the cardiac region to relieve chest distress. Mixture containing Soda and Magnes. Blisters with Digitalis Liniments rubbed into legs. Wet: chiefly milk...
Case No IV.

Coh Chin, male, age 33. Cook at laticum coal mines.

Admitted into Hospital 21st December 1900.
Discharged cured 11th January 1901.

History: Patient states that he has been ill for one month suffering from fever which lasted twenty days, after which period his feet began to swell, and numbness appeared in his toes, which gradually spread upwards to his knees; about the same time he had pain in the muscles of the calves of his legs. After taking Chinese medicine the swelling disappeared, but there was no change in the numbness or pain.

State on admission: Patient complains of numbness in the legs which spread upwards from the toes to the middle of the thighs; there is also numbness extending from the fingers to the shoulders. The muscles of the legs are painful to pressure and there is pain in the muscles of the forearms when they are pressed between the radius and ulna. The hands are not strong and he cannot hold any weight.
The patient can stand and experiences no inconvenience from doing so, but his walking is slow and he usually keeps his eyes fixed on the ground. The patellar reflexes are lost. There is some tightness in chest and at night he feels warm and is thirsty. Appetite is good, tongue clean and bowels regular.

Progress of Case:

26th December 1900. Numbness is somewhat less. At noon, he complains of pain and tightness in his legs.
31st December. Numbness only extends from the toes to the middle of the calves of the legs and from the fingers to the elbows. There is tightness and diminished pain in the legs.
4th January 1901. Improvement is marked. Numbness is less and there is no tightness in the chest.
6th January. Has a headache. Numbness only present in hands and feet. Patient walks without any difficulty.
11th January. Patient feels very well and is discharged cured.
Treatment:

General tonic treatment, consisting of quinine, iron and strychnine. Linements well rubbed into the painful muscles.

Diet: Modified rice diet, with fish and meat.

Case No. V.

Lee Sing, male, age 24. Cookie at Mura coal mines.

Admitted into Labuan Hospital 6th June 1895.

Discharged cured 29th June 1896.

History: Patient states that he has been ill for two months, during which period he has had occasional attacks of fever. He noticed, also, that a gradual increasing weakness of his legs prevented him from moving about without the aid of a stick.
and at the same time he experienced numbness in his legs. The weakness increased to such a degree that he lost all power of locomotion and then he sought admission to hospital. He never noticed any swelling.

**State on admission:** Patient is a muscular and well-fed man. The muscles of the calves of the legs are firm and hard and on pressure there is pain.

He lies on his back usually with his legs flexed, because when he keeps them extended for any length of time, there is great tightness at the back of the knees.

He is quite unable to walk.

He has pain in his chest and a girdle of tightness.

Temperature 98.5°F.

Appetite fairly good. Tongue is slightly coated with a white fur. Bowels are constipated. Urine normal.

Plantar and knee reflexes are absent.

Anaesthesia is complete in both legs and extends from the toes to the middle of the thighs.

Right Arm: sensation is perceptible to a pin in the palm and palmar surface
of fingers; on the back of the hand, sensation is present only on the fingers. The front and back of forearm and arm give negative results except a few points round the front of the elbow where sensation is normal.
Left arm: The palm and back of hand are normal. In the forearm, sensation is somewhat impaired, while in the arm, it is normal. There is no oedema anywhere.

Progress of Case:

11th June 1895. The condition is much the same. The pains in chest and legs are more severe, and patient states that he feels as though he were bound with ropes from his legs to the umbilicus.

17th June. Patient feels slightly better; the numbness is not so great.

23rd June. Numbness is not so extensive, and the pain in the chest is less.

6th July. The arms are weak. He says that his legs are stronger, and he does not complain of so much tightness behind the knees, when his legs are extended.
17th July. Sensation in legs and arms is returning, but is irregular in distribution. Patient can just stand with support.

3rd August. Pressure of the muscles of the calves of the legs and the tendon Achilles causes great pain. Patient is very weak.

10th August. Muscles of legs are flabby and wasted. Unable to flex or extend the toes, nor can the foot be flexed at the ankle.

21st August. Pain is less and patient can stand more steadily.

29th August. Condition the same.

10th September. Improvement is slow. There is more power in the hands and arms, and objects can be grasped better. The chest pains are much less and the feeling of being bound with ropes has gone.

26th September. Numbness is much less. Ankle drop is well marked.

11th October. Pains in legs are severe and occasionally patient has muscular cramps. Ankle drop not so marked.

20th October. Has a sensation of pain and
needles" in his fingers. Throat is dry.
30th October. When standing, can move his feet, but dare not attempt to walk. Tightness is still severe behind the knees.
3rd November. Temperature rose in the evening to 105° Fahr. Patient feels very ill and weak. There is some congestion at the bases of the lungs. Pulse is frequent, 120 per minute. Breathing is somewhat laboured.
4th November. Temperature has fallen slightly and patient feels more comfortable. He has a slight cough.
6th November. Temperature rose to 104° Fahr. after which patient perspired profusely and the fever abated.
8th November. Temperature normal and lungs clear.
16th November. Hitherto much less and patient feels stronger.
24th November. Pains and tightness of chest greatly lessened. Muscles are firmer and there is every appearance of the patient putting on flesh. Patient can move about with the aid of crutches.
1st December. Numbness present in hands and feet only. Sensation returning, and patient can feel the ground when walking.

2nd December. Tightness of chest and muscles behind the knees nearly gone. Can walk about with greater confidence.

12th December. Patient in attempting to go downstairs fell and bruised his ankle.

23rd December. Slight swelling of feet brought about no doubt by weakness and the attempts of the patient to move about too much. He is tired of lying down.

29th December. Hands have nearly recovered. Pains of tendo Achilles and muscles of the calves much less.

2nd January 1876. Swelling of feet persists. Numbness of the feet has not altered. Here is considerable tightness of abdomen. A small abscess above the left eye, causes swelling of eyelid and cheek, and a slight rise of temperature.

4th January. Swelling of face is much greater. Numbness, where present, is more severe.
There is considerable tightness in the chest, and in every way, the patient appears to be worse.

6½ January. An improvement has taken place.

10½ January. Swelling of face less. Complains of pain throughout the body, thereby preventing sleep.

12½ January. Has a sharp attack of fever and does not feel so well.

19½ January. Abscess nearly healed. Numbness is still bad, but condition generally is better.

1st February. Tightness in chest and legs still bad. Great improvement in walking.

6th February. Swelling of feet gone. Abscess completely healed. Pain and tightness of legs greatly improved; there is still some tightness in the chest. Appetite is bad and there is a feeling of distension in the stomach.

10½ February. Still want of appetite for food. Legs have improved since last note.

13½ February. Numbness in feet and tightness of chest rather more severe.

21st February. Patient can walk well
but it causes considerable pain in the legs. Patient has put on weight.

20th March. Some pain over the instep of right foot. Numbness of feet has gone.

Pain in legs less and walking can be accomplished without the aid of a stick.

5th March. Patient feels very well, and does not feel much pain in the muscles, except when they are pressed.

28th March. Still has pain over the instep. No numbness. Feels tightness behind the knees. Patient is remarkably well.

5th April. Improvement.

9th April. The pain over the instep is worse at night. No slight numbness along the tibia.


8th May. Patient can move his toes, except the large ones. Tightness is easier. Patient can walk well.
There is a slight return of knee reflex.
10th May. There is no pain over the instep. Numbness seems to come one day and go the next.
23rd May. Rather more pain and tightness in muscles of the calves.
31st May. Improvement is marked. Chest is not at all troublesome and no distension in epigastrium is complained of.
6th June. Tightness, numbness and pain nearly gone. Big toes can be moved, and patient has hardly any difficulty in walking.
22nd June. There is no tightness and no numbness and only a little pain is complained of.
29th June. Patient feels very well and is discharged cured.

Temperature: As it would be useless to give in detail the temperature chart of such a prolonged case, it is only necessary to state that the course throughout the illness was more or
less normal, except during the periods when there was some febrile disturbance due to intercurrent troubles and these have been fully detailed in the progress of case.

Treatment:

Regulation of bowels with occasional doses of calomel.

Keller’s tonica and Aconite liniments for the muscular hyperaesthesia.

Morphia for the general pains throughout the body which occurred on Jan 13th 1896.

Symptomatic treatment for bruise of ankle and for abscess above eye.

High temperatures treated with quinine.

Tightness of chest and congestion of lungs treated with digitalis and other stimulants.

General tonic treatment with iron, arsenic, and strychnine.

Diet: as occasion occurred, a low and milk diet in contrast to the usual full diet of hospital custom.
Case No. VII.

Chau Ah Yong, male, age 22. Fireman on a coasting steamer.
Admitted into Hospital 20th November 1901.
Died 23rd March 1902.

History: Patient states that he has been ill for one week, first noticing swelling of his feet which spread gradually upwards till it reached the face, and at the same time there was a gradually increasing weakness of his legs.

State on admission: There is some oedema along the right tibia and to a slight extent swelling is present on the dorsum of left foot. There is no general oedema. There is numbness in hands and feet reaching as high as the wrists and ankles respectively. The pain and tightness of muscles of calves are considerable and muscular hyperaesthesia is well marked when pressure is applied. Patient cannot stand well and he is unsteady in his gait. There is tightness in chest and
difficulty in breathing is complained of. Heart sounds are clear. Plantar and knee reflexes are lost. Appetite is good and tongue clean. Bowels are regular. Temperature 98.4° F. All other systems appear to be normal.

Progress of case:
24th November 1931. Patient has kept to the recumbent position since admission and in consequence all swelling has disappeared. All power of motion in the legs is lost. Patient is a helpless being. Considerable weakness of arms has developed.

28th November. Condition the same.

The skin of the leg was rendered aseptic and a blister raised and agar-agar tubes inoculated and incubated.

29th November. Swabs are swollen, sore and bleed slightly. The agar-agar tubes remain sterile.

2nd December. Patient is most helpless.

10th December. Sensation has increased and extends to the elbows and to the knees. There is some pain in the
Throat and voice is weak. Sius are better.
The pain in the muscles of the legs is
still severe. Hands are quite paralysed;
it is impossible for the patient to hold
anything and there is in coordination
of muscular efforts. Toes cannot be
moved and the ankles hang flail-like.
18th December. Painless is slightly im-
proved. His feet are painful.
21st December. Bowels constipated. The
temperature rose to 101.5°F.
28th December. A slight improvement is
noticeable. There is no tightness in the
chest. Numbness still present from
elbow to fingers but only from ankles
to toes.

2nd January 1902. A change for the better.
21st January. Numbness extends only to the
wrists. Ankles and toes cannot be
flexed.
12th January. Patient says that he has much
pain in the bones of his legs. He is
able to stand if supported, but cannot
do so for long out account of the pain
and tightness of muscles. Sleeps badly.
21st January. There is just a trace of edema on the feet along the lines of the metatarsal-phalangeal articulations.

23rd January. Fullness in epigastric region.

24th January. Fullness in abdomen gone after a dose of aperient medicine.

25th January. Slight tightness in chest.

Condition remains the same.

29th January. Slight improvement. No numbness in fingers. Fingers can be used. There is numbness in the thumbs; thumbs cannot be flexed.

1st February. Patient has greater confidence when standing, but there is much pain and tightness behind the knees.

6th February. Legs feel stronger, but patient cannot stand yet without support. He is able to move his ankles.

23rd February. Vomits frequently. No appetite, takes milk only. There is a good deal of discomfort in the chest. Heart beats are rapid and thumping and cardiac impulse is diffuse.

Temperature is subnormal.

24th February. Vomiting ceased. Chest is
easier, though there is some pain in the left side.

20th February. Numbness in feet. Sensation has returned everywhere in the legs, except in the toes. Legs feel tired. When sitting up, patient feels giddy. The pain in the chest is less, but breathing is somewhat troublesome.

7th March. Can stand better but still requires to hold on to a support, and complains of much pain and tightness in the muscles of his legs. The face is puffy along the border of the lower jaw. Patient is anaemic.

12th March. Hands have recovered perfectly. Swelling of feet less.

14th March. Patient is able to walk with the aid of crutches, but feels his ankles very weak. The ankle drop is much improved.

19th March. Can walk tolerably well and can feel the ground, sensation having returned in the feet. There is very little numbness in the feet, but considerable tightness of muscles of legs.
22nd March. The moving about seems to produce a slight general oedema. The swelling of feet has increased and has extended upwards. There is oedema of chest and abdomen and the puffiness along the lower jaw appears to make the cheek too hang too low. The neck is full.

23rd March. In the evening, the patient suddenly vomited and complained of fullness in the epigastrium and much tightness in the chest causing difficulty in breathing. The condition continued to get worse and patient became extremely restless, rolling about from side to side, heating his chest as though by doing so he was enabled to get more air into his chest.

The tightness and oppression in the chest are very severe, and signs of impending death are supervening, as indicated by the lividity of the lips, coldness of the toes, fingers and nose and by a profuse, cold, clammy perspiration. The agonising pain
in the chest is increasing and causes the
patient to utter cries. At 9 p.m. the
temperature fell to 96.5° Fahl and shortly
afterwards he died.

Temperature chart of case VII

Nov 1901

Temperatures:

Jan 1902

Temperatures:
Treatment:-

Occasional doses of calomel and mixture alba, were necessary to correct constipation, sometimes combined with cascara sagrada.

Potas. Acetat and Citrate combined with digitalis to relieve the swelling and tightness in chest.

Tonics of iron, strychnine and arsenic.

Camphor monobromatum was given in 2 gr doses three times daily with gradual increases, and under this treatment, patient felt much better.

The battery was applied to the muscles when the pain was less, and bathing the legs in salt water was indulged in when possible.

 Diet:- a low diet consisting chiefly of milk and chicken broths. Rice was avoided as much as possible on account of the distension and fullness of stomach, which it caused.
Analysis of Symptoms of Keri-Keri.

Premonitory.

These are uncertain and are not definitely defined. There may be sensations of heat and cold throughout the body. Frequently a feeling of lassitude is complained of and an inability to carry out the day's work satisfactory. The patient is easily fatigued and the legs do not appear to be so strong as they should. - a feeling of tightness being experienced in the muscles behind the knees. He may notice that in carrying heavy loads or in walking up steep inclines that he pants for breath and sooner or later he takes to his bed and applies for medical relief.

Fever is frequently complained of at the commencement of the illness, but many cases run throughout their entire course without a rise of temperature. When the disease is ushered in with fever, the temperature is never very high.
Integumentary System.

A marked peculiarity about the skin, is the inactivity of the sweat glands, and only in exceptional circumstances are there any signs of perspiration and these are present in local patches, such as, the plantar surfaces of the toes. Even when drugs are administered to stimulate the sweat glands, they seem to be slow in their action. Perspiration is often profuse when death is approaching. It is on account of this absence of perspiration that the skin usually presents a harsh, rough, dry and scaly appearance, causing thereby a loss of the natural gloss which is so well-marked in the dark-skinned races such as Indians.

Certain cutaneous eruptions are considered by some authors to be peculiar to Beri-Beri, but when carefully analysed, it will be found that they are usually accidental. In my experience, eruptions are generally absent, but when present, they take the form of petechiae and herpes. I have seen a very severe case of herpes zoster in
a Beri-Beri patient.
The colour of the skin depends largely upon
the existence or not of anaemia and the
presence or absence of oedema, and when
there is much oedema, the skin is put on
the stretch and has a pale look, and at
the same time it is somewhat shiny.

Oedema: This is almost a constant symptom
of the disease and usually first makes
its appearance along the tibiae. In
cases which have come under my notice
within a week or so of their commencement
and in whom no history of oedema could
be obtained, the oedema, when it did show
itself, was noticeable on the dorsum of the
foot along the line of the metatarsophalangeal articulations of the 3rd, 4th,
and 5th toes.

The reason for attributing the initial
swelling to the skin over the tibiae can be
explained, I think, by there being an under-
lying hard bony surface against which
the integument can be easily compressed
and perhaps the oedema is sought for
in this situation than in any other.
The oedema varies greatly in extent from a mere local trace to a general oedema, sometimes only present on the dorsum of a foot, the back of a hand, along the border of the lower jaw, or even over the sacrum.

The oedema is of firm consistence and may develop slowly or it may develop rapidly; its duration may only be a day or it may last for many weeks. When oedematous symptoms are more pronounced than the nervous symptoms, it is then classified as Oedematous Beri. Beri and may involve all the serous cavities.

Nervous System.

Numbness is one of the earliest symptoms and may be first noticed in a foot, or in the fingers, lips and extending as far as the ankles or the wrists, it may remain there for sometime. Should it continue to spread, it does so upwards until it may involve the whole body. Anæsthesia is seldom complained of in the back.
Patients sometimes complain of a girdle of anaesthesia round the abdomen on a level with the umbilicus. Simoons remarks that numbness round the mouth was a common feature in Beri-Beri of Japanese. In my experience, I have found it of frequent occurrence amongst the Chinese. Anaesthesia is irregular in its onset and follows no special paths. The sensation of tingling and pins and needles may and may not be experienced. Formication in various parts is common. Sensation to heat and cold may be increased or diminished, and in some cases heat may be recognised as cold, and cold as heat. Paraplegia may affect one or more limbs at a time; sometimes only the legs are involved, sometimes only the arms; and in severe forms both legs and arms are completely paralysed, such patients lying on their beds as utterly helpless beings, unable to move a finger or a toe. In this extreme condition, bedsores sometimes form. When the fingers are paralysed, they are usually flexed upon the palm and it
becomes impossible to lay the hand flat on a surface, and objects are grasped with difficulty. When the lower extremities are involved, the patient is very unsteady in his gait, is unable to turn round sharply and if asked to stand with his eyes closed will sway from side to side. The larger motor nerves are affected. Implication of the vagus nerve accounts for the vomiting. Occasionally the laryngeal nerve is involved causing complete or incomplete aphonia.

Shortly before death, twitchings, tremors and choreic-like movements are observed but chiefly when the fatal termination is slow and prolonged. Vaso-motor fibres would appear to be involved and implication of them would account for the oedema. Mental faculties are not as a rule affected, the patient remaining conscious up to the very last.
Muscular System.

The muscles, or groups of muscles, that are most commonly affected are those of the legs and arms; perhaps, next in frequency, we might place the laryngeal group.

The muscles may be affected in one of two ways: a. by being swollen. b. by being atrophied.

Swollen and atrophied muscles may appear together in the same patient. Atrophy may be more marked in one leg than in the other. When the muscles are swollen, they are hard and firm, and in long-standing cases, one can often detect, especially in the gastrocnemii, oval-shaped indurations.

Atrophy of the muscles is shown more markedly in certain groups such as those of the calves of the legs which may consist of a mere band of muscular tissue. A similar condition is met with in the muscles of the thenar and hypothenar eminences.

Muscular cramps are of frequent occurrence and are to be seen in the muscles of the calves and thighs.

In addition to these cramps, patients often
complain of tightness in the muscles, more marked when walking and especially severe when assuming the erect attitude. The tightness is usually referred to the tendons at the back of the knee by the gastrocnemii and hamstring muscles, and also to the tendon Achilles.

I have, in the hospital, under treatment, a patient who has tender, hypertrophic and contracted muscles of his calves, a thickened and shortened tendon Achilles, and although he can walk with the aid of a stick, yet he is only able to touch the ground with the balls of his toes. This condition is very chronic and in all probability, it will never entirely disappear.

Muscular hyperaesthesia is more or less present from the commencement of the disease and is easily elicited by compressing the muscles against an underlying hard bony surface, causing the patient to wince or even cry out. The hyperaesthesia is more marked in the calf muscles than in any other group, though it can be demonstrated by...
pressing the thenar and hypothenar muscles and the muscles of the arms and thighs.

Gait: this is characteristic and is usually known as the Keri-Keri gait. The patient having steadied himself in the erect attitude, lifts his leg with difficulty high from the ground, shoots it forward and then lets it down heavily on the ground; the foot wobbles about and appears as though it were badly jointed at the ankle. The eye is usually kept on the ground all the time and the legs are spread apart in order to get a broad base and so the better to balance himself.

Reflexes: as a rule, the superficial reflexes, such as the abdominal and epigastric are present and normal, though they have been known to be increased, diminished or even disappear. There is great difficulty in obtaining a plantar reflex owing to thick, hard and horny condition of the skin. The patellar tendon reflex is lost usually at an early stage of the disease. I have never
observed an exaggeration of it.
Ankle clonus is never obtained.

**Circulatory System**

Precordial uneasiness and palpitation are very frequent symptoms in Beri-Beri and are exaggerated on the least exertion, such as going up and down stairs. The carotid arteries may pulsate violently and pulsation of the abdominal aorta may be visible. The cardiac impulse, usually feeble, may be diffuse and towards the right side when there is increased precordial dullness as indicating dilatation of the right heart.

Bruits heard over the different valve areas come and go during the progress of a case. Accentuation of the second sound at the pulmonary area is of frequent occurrence, but re-duplication, in my experience, is not common.

Precordial pain associated with epigastric fullness and distension should be hailed with suspicion. It is a serious symptom and is usually described as though a heavy
weight were pressing upon the chest, giving rise to a sensation of constriction and oppression, and at the same time the patient is unable to draw a full breath. The pain is sometimes noticed after partaking of a full meal, especially one of rice, and in many cases this would appear to be a distinct cause. It is constantly present when the disease is about to terminate fatally. It is a sign of failing circulation and must be considered as one of very great import, as before long irregularity of the pulse will show itself, then followed by coldness and a purple hue of the lips and extremities, a violent pulsation of the veins of the neck, breathlessness and irregular respirations. The period of onset of heart failure is, by none, said to be most frequent towards the end of the first month of the disease and before muscular paralysis shows itself. I have not found this true in the case.

It is worthy of notice that the sudden heart failure is analogous to the heart failure of diphtheria - a disease in which these
is also a peripheral neuritis.

The Pulse, naturally, in such a disease as Keri-Keri is accelerated on the least exertion and a marked difference may be studied in it during the erect and recumbent positions of a patient.

There is a lowered pulse tension and a feeble pulse wave.

Retardation of the pulse is rare, but it must not be forgotten that it does sometimes occur.

Below will be seen three sphygmographic tracings.
The Blood.

Microscopical examination of the blood reveals nothing characteristic. Agitation of the corpuscles, said by some observers to be peculiar, has, in my hands, been due to a faulty preparation of the film. There is no increase in the number of white corpuscles.

Haemorrhages from the nose and gums are sometimes observed.

Anaemia is a frequent concomitant.

Cyanosis is present when death is about to take place.

The Temperature.

Patients usually give a history of febrile disturbance before the actual numbness and other early symptoms set in; whether one should regard this rise of temperature as a prodromal stage of the disease is doubtful. I am inclined to look upon the rise of temperature as a derangement of the heat centre connected with malarial fever or perhaps with some disturbance of digestion and that an initial rise of
Temperature is not to be expected. Throughout the whole course of a case of Beri-Beri, from commencement to finish, we can say as a general rule that there is no rise of temperature and any that there may be can be accounted for by some intercurrent ailment such as a superficial abscess, stomach troubles or malarial fevers.

It has fallen to my lot to treat patients in the very earliest stages of Beri-Beri — in what might be termed Beri-Beri Incipiens and no rise of temperature has been noticed. Any variation that does occur consists in a slight morning fall and an evening rise. The temperature charts given to Cases 1, 2, 3, 4 and 6 will explain.

In some cases where a definite cause cannot be given for the increase of temperature, we may find a reason for it in an exacerbation of some of the symptoms viz a sudden increase of the pain in the muscles or along the course of the larger nerves such as the peroneal and radial nerves. Temperature charts usually show a normal
or a subnormal reading during the progress of the case and when the disease is about to terminate fatally, there is invariably a subnormal temperature.

Respiratory System.

Aphonia, resulting from paralysis of the laryngeal muscles, is not present in all cases of Beri-Beri. It may come on early in the disease or it may be late in its onset. In many cases it amounts to a mere whisper and when associated with paresis of other muscles of respiration, it is of grave importance. In the cases which have come under my observation, aphonia when present has always recovered. Shortness of breath frequently accompanies palpitation and in many instances dyspnea occurs. Occasionally, nasal, laryngeal and bronchial catarrhs come on during the course of Beri-Beri.

Edema of the lungs is generally present when Beri-Beri ends fatally. It gives rise to
great restlessness and dyspnoea and patient usually complain of great weight in the chest at the same time.

**Digestive System.**

The tongue usually shows signs of weakness digestion, being more or less coated. The fauces are often congested and a feeling of dryness in the throat and thirst are frequently complained of. Constipation has, as a rule, to be corrected. Sometimes diarrhoea is present. The motions may be passed involuntarily towards the end, but usually occur in those cases of complete paralysis when death is slow and the patient more or less in a state of unconsciousness.

The appetite remains good throughout the course of the disease, as a rule, but a full rice diet is frequently followed by fulness and distension in the epigastric region, giving rise to palpitation. In two or three cases I have noticed that patients who have been living on a meat diet exclusively and then partaken of rice, have suffered severely.
from abdominal distension and that symptoms of cardiac failure setting in have proved fatal. It may simply have been a coincidence but it must be kept in mind that a rise diet may produce disastrous results.

In addition to the epigastric uneasiness, heartburn and pyrosis are not uncommon symptoms.

Vomiting, apart from being due to an over-loaded stomach, often heralds death. If associated with cardiac and respiratory troubles, we may expect a fatal issue. It may come on quite suddenly and without any warning.

Hiccough is sometimes troublesome. Liver, spleen and other organs are usually healthy and take part in a general congestion when such is present.

Genito-Urinary System.

This system is not affected in any special way. The urine is normal in reaction and specific gravity and is free from albumen.
when sedema is present to a great degree, the quantity of urine per day is diminished but suppression is rare. I have not seen a case of suppression. The urine may be passed involuntarily under the same conditions as the motions are.

A case is reported from India in which altered blood corpuscles were detected in the urine by aid of the microscope, but until there is convincing proof that the urine is albuminous in a large majority of instances, we must regard the presence of albumen as being a stray constituent and not at all peculiar to the disease.

One or two cases have come under my notice of Beri-Beri patients who have been cured, and on their return to hospital have suffered from albuminuria. What the relationship between Beri-Beri and albumen in these cases, it is difficult to say. It must be considered a sequela. Seldom is paralysis of the bladder observed. Sexual impulses are generally impaired and may be entirely lost.
62.

Termination of Beri-Beri.
The tendency of Beri-Beri is to a cure, but in many cases when patients return to their work, one or two symptoms are apt to return. As sequelae, we can consider as important, weakness of the legs with contraction and induration of the muscles of the calves. Weakness and palpitation of the heart. Fullness in the epigastrium, absence of knee-reflex and albuminuria.

Diagnosis.
In early (Beri-Beri incipiens) and isolated cases, diagnosis is somewhat difficult. When a patient complains of puffiness of the chins and feet accompanied with numbness in the fingers and the toes and weakness at the knees with perhaps, some discomfort in the chest, we may on further questioning, elicit the information that others, in the same district or ship from which this man came, suffered from similar symptoms and therefore we must always keep Beri-Beri in
our mind when dealing with patients in tropical and sub-tropical regions as such symptoms as the above are almost certain to turn out to be Beri-Beri. In advanced cases, the diagnosis is somewhat easier; the pronounced numbness, oedema or only a history of it, absence of patellar reflex, peculiar gait and thumping cardiac impulse with bruits which come one day and are gone the next, should be a sufficient guide to form a diagnosis on.

**Differential Diagnosis.**

We have to differentiate the disease from:

1. **Malarial Neuritis**: this is rare and our knowledge is imperfect. We would expect rises of temperature and pains would be less constant. In all probability, there would be splenic enlargement. In malarial neuritis, local oedema is rare and heart symptoms are not marked; a cure is readily affected with quinine and arsenic, whereas in Beri-Beri there is usually a history of oedema and cardiac
symptoms are well marked and quinine and arsenic do not affect a cure.

II. Alcoholic Neuritis: The question would chiefly arise in the case of Europeans as natives and Chinese are not large consumers of alcoholic drinks. The history and remors would act as a guide.

III. Locomotor Ataxy: Lightning pains. Various crises and ocular symptoms together with electrical reactions and trophic disturbances would be strong points against Beri-Beri.

IV. Progressive Muscular Atrophy: In this disease, atrophy commences in the upper extremity - the lower limbs remaining unaffected until a very late period. Cutaneous sensibility, as a rule, remains intact.

V. Renal Disease: Absence of albumen and other abnormal constituents in the urine in Beri-Beri mark a strong contrast between the two diseases.

VI. Cardiac Disease: The suddenness with which bruits come and go is a peculiar feature of Beri-Beri.
VII. Arsenical Poisoning:—this is distinguishable from Beri-Beri by the presence of skin pigmentation varying in colour from brown to black; by the cutaneous eruptions, by pruritus with local perspirations and a furfuraceous desquamation of the cuticle. Amongst the gastrointestinal symptoms, vomiting and diarrhoea are present, and although vomiting is a prominent symptom in Beri-Beri, it usually occurs late in the disease and often heralds a fatal issue, whereas in arsenical poisoning, it extends over a lengthened period and occurs early. In Beri-Beri, there are no characteristic mental symptoms such as one sees in arsenical poisoning and again, in Beri-Beri there is a much larger proportion of dilated hearts.

Prognosis.
This must always be guarded. Beri-Beri is a most treacherous disease, and patients have been known to be discharged cured
from hospital and before they have gone a
hundred yards have fallen down dead.
I have known of cases completely paralysed
in which the patients were absolutely
helpless, unable to raise themselves from their
beds or feed themselves and yet have
recovered; on the other hand, patients who
appear to be much less affected and indeed
recovering have developed sudden tightness
in the chest, dyspnoea and vomiting and
before many hours have passed are dead.
In giving a prognosis much depends upon
the system which is seriously affected.
One may safely say that a case in which the
muscular system is solely affected while the
respiratory and cardiac muscles remain
tolerably free, that that patient will recover,
whereas a patient in which the cardiac and
respiratory systems are the chief seats of
disease, the prognosis is bad.
Vomiting, apart from indigestion, is of very
great omen, and should always be looked
upon with the greatest suspicion.
The mortality will depend greatly upon
whether we are dealing strictly with the mor
acute forms or with the minor forms.
In my own experience of Hospital practice in this country, in many of whom the patients were far advanced in the disease and moribund on admission, the mortality has been as high as 27%. This is a very much higher percentage than recorded by some observers.
Patients who are removed from the endemic area have a much better chance of recovery than those who continue to live in the place where they contracted the disease.

**Treatment.**

There is no specific cure for Beri-Beri.
The most that we can do is to treat it symptomatically; it is convenient, therefore, to study it under three headings, viz.

1. **Prophylaxis**
2. Local
3. General

1. **Prophylaxis:**
   Great attention must be paid to prevent
overcrowding: thorough ventilation should be insisted upon. Houses should be built
on rising ground and damp flat land avoided, and when this is impracticable
the ground immediately underneath the building should be cemented. Proper
regard must be had for efficient drainage.
Buildings, such as hospitals, gaols and barracks
in which large numbers of patients are
frequently housed, must be subjected to
periodical thorough cleansing and disinfection. When space will allow, it is
advisable to remove the patients from one
room to another, so that the one can be
properly cleansed and disinfected while
the other is being occupied. I am in the
habit of adopting this plan and after
fumigating with sulphur, the walls and
floor are washed with a 1 in 1000
solution of perchloride of mercury and washed
again with fresh water containing some
disinfectant such as Jeyes Fluid, Physo or
Carbolic Acid.
The clothes of Beri-Beri patients must be
carefully washed and disinfected.
It is advisable and very necessary to remove all patients from the infected area, as continuance of residence in the district lessens chance of recovery. When cases break out on board a ship, those affected must be sent on shore when possible, or isolated from the rest of the crew in the meantime, and their quarters not occupied until cleansing and disinfecting have been carried out.

The general rules of personal cleanliness must be enforced.

Local:

Gentle friction of the extremities with anodyne liniments will relieve much of the pain and tightness of the muscles. When the pain has been intense, I have given hypodermic injections of morphia but they have afforded little permanent relief.

Bandaging of the arms and legs lessens the oedema. The precordial pain and sneezing is best relieved by the application of liquor epicaptaris. In the more chronic cases, electricity is useful as it helps to give tone to the muscles, but
it should not be applied when there is much muscular hypaesthesia.

3. General:

This must be of a tonic nature. Patients when able to get out should be encouraged to live as much as possible in the open air and sea-bathing, without exertion, should be indulged in. For those who can afford it, and when their condition permits, a sea-voyage should be recommended.

In cardiac cases, active movements must be avoided and rest insisted upon.

As to diet, this must be of a light and nourishing nature. Milk is excellent. It is customary for natives to have two meals daily, but it is preferable in Beri-Beri patients to split these meals into three, or even four, daily. In this way we avoid overloading of the stomach and perhaps avert some of the unpleasant symptoms which are known to arise from partaking of a full meal. Rice which enters largely into the dietary of Asiatics and causes considerable distension and fullness in the epigastrum
is best substituted by beans and this method should be followed in all cases of Peri-Peri.

Tea from its contained tannin is said by some to be beneficial. It is the custom with patients in Babuna Hospital to be supplied liberally with tea but I cannot say that there is any very special beneficial result therefrom. It is given as a drink and the quantity allowed is two quarts daily.

Drugs: what has succeeded in the hands of one medical man, has failed in another. The bowels should be kept freely open—two or three motions daily, especially in dropsical cases—with mixture alba, sulphate of soda, calomel or pulse jalapae Co.

Perspiration should be encouraged by surrounding the patient with hot water bottles and covering him up with blankets and administering a mixture containing nitre, ammonium and jaborandi.

Cardiac and respiratory failures are to be treated with suitable drugs and when these are severe, inhalations of nitro-glycerine may be given; we may even have to resort
to cupping and resection but they can only hold out a temporary hope of relief. Digitalis or Stephania are the two drugs upon which one can depend in cases of palpitation of the heart and in dropsy. It requires care in its administration and must be watched.

Applications of Belladonna and glycerine to the cardiac region also relieve palpitation. Lately, I have been giving patients monobromated camphor in 2 grain doses in pill form, three times a day, and gradually increasing the dose weekly until 12 grains have been taken daily. I have not noticed any unpleasant effects from it, such as a lowering of temperature and pulse, and it does not cause gastric irritation nor have any marked soporific action. Indeed, one would rather say that the camphor stimulates the nervous and vascular systems and acts as an antiseptic in the bowel and perhaps it is to this latter action that camphor has acted beneficially in my hands in cases of Beri-Beri. After giving the camphor for about a month
I usually stop it for a few days and substitute another nervous tonic, and then resume its administration. For the most chronic cases, iodide of potassium was tried but it is disappointing. Silver nitrate and chlorides of gold give more hopeful results. A mixture containing iron, arsenic and strychnine is useful when acute symptoms have subsided and the patient enters, so to speak, upon the stage of convalescence. When patients are suddenly seized with cardiac distress and oppression in the chest, it might not be inadvisable to resort to tapping of the pericardium, and although I have never carried out such heroic treatment, my observations at post-mortem on such cases almost convince me that such a course would be advisable, for it will be found that there are several ounces of fluid in the pericardial sac.

The native treatment of the disease is interesting and varies with different tribes.
In remedious cases, the people living on the coast subject the patient to a severe sweating process. The man is seated on a log enveloped in a mat, below which a fire is made of the rubbish which collects at the mouths of the rivers.

The Dyaks of Sarawak try to reduce the swelling by making small incisions in the skin covering the calf muscles and skin, but little benefit seems to be derived from this method of blood-letting.
The foregoing photograph shows three Bari-Bari patients suffering from muscular hyperaesthesia. In them the oval-shaped indurations of the gastrocnemius muscles are well marked. There is no oedema. Although they are able to walk, there is much pain in doing so. They require the aid of a stick.

The above photograph shows a Bari-Bari patient in the act of walking. He is fairly well advanced towards recovering, and although he requires the aid of a stick to steady himself, yet he does not require a broad base.
The above photograph shows a Beri-Beri patient who is unable to stand or walk. Swellings are general. The wasting of the muscles of the legs and the weakness of the ankles are shown. There is intense hyperaesthesia of muscles of arms and legs.
The same patient as the foregoing photograph to show ankle drop which is perfect. It is vitally painful to the patient to sit in the position shown.
Aetiology.

This can be studied under two heads, viz. A. Predisposing, B. Exciting.

A. Predisposing:

1. Sex: generally speaking, Peri-Peri is less common in women than in men, probably because they are not so much exposed to the influences determining the necessary conditions for the entrance of the poison into the system. It is difficult to estimate in what proportion the two sexes are attacked as this must vary in different countries. In my own experience only 2 or 3% of women are attacked, and most of these are Chinese women who work at the coal mines in Labuan. Women just after childbirth are very susceptible to the disease, and the mortality is high.

2. Race: there is no actual racial immunity, but Europeans suffer much less frequently than natives and this can be accounted for by the improved and hygienic conditions under which our European lives.
After nine years residence in the East, I have had under my observation only three Europeans affected with Peri-Peri - two of whom recovered, and one died. I believe, however, that if Europeans were to live under the same conditions as the Chinese in this country, they would be just as liable to contract the disease. Europeans are more liable to the disease than Europeans.

A point worthy of consideration is that the native of this country is not stricken down with Peri-Peri in anything like the same proportion that the immigrant Chinese is. It is the Celestial who furnishes the bulk of the number of cases in our hospitals. This may be accounted for by the fact that the native is an indolent creature and will do no work when he can possibly avoid it.

5. Age. Peri-Peri is a disease of adult life. Cases have been reported as occurring in children at the age of 2 years and in old men of 77 years of age.
Three or four years ago, an epidemic was reported as having broken out in the German Blind School and Foundling Hospital for children in Hong Kong.

Occupation. The disease is chiefly found amongst the coolie class, especially those engaged on estates or in opening new jungle as was witnessed in Borneo some ten years ago. Miners are frequently attacked and those working in the tin mines in the Federated Malay States and in the phosphate mines on Christmas Island are responsible for the greater number of cases in the hospitals there. In Labuan and the neighbouring territory of Sarawak, the majority of the coolies affected are those who work underground cutting coal being at the time exposed to damp, heat and vitiated air.

Of sedentary occupation, it is more common in tailors, clerks, teachers and mechanics. It is well known that soldiers and sailors are liable to be attacked.
Amongst coastal divers the disease is fairly frequent.

5. Sanitary conditions - these include clamps, filth, overcrowding, bad ventilation and defective drainage. These, as a rule, are always present when large numbers of coolies are housed together. Insufficient regard is paid by employers of labour in insisting upon having their coolie houses built on high ground. They imagine that anything is good enough for a coolie until a severe epidemic of Beri-Beri breaks out amongst them and it is only then that the short-sighted policy is discovered, but perhaps too late in avoiding a high death-rate and a hampering of the work on which they are engaged.

It is not uncommon to see coolie houses built on low, flat, damp land, badly constructed and almost devoid of any ventilation and the natural filthy habits of the dwellers therein combined with general insanitation favour the growth of any organisms requiring heat, filth and damp.
6. Meteorological conditions - the disease occurs more frequently during the wet season and one constantly observes that patients complain more of their pains during a wet day.

7. Previous attacks - persons who have been attacked once, are liable to a second or third attack.

8. Wounds and sores - an individual with a wound is more liable to contract the disease than one without a wound. Patients in hospital being treated for wounds have occasionally developed Beri-Beri, but not until they had been a month or so in the hospital; whether they had previously been exposed to the poison, it has been impossible to ascertain.

B. Exciting:

Many theories have been advanced as to the real cause of Beri-Beri, but many of them should be relegated to oblivion. The chief theories are.
1. The Anaemia Theory.
2. The Intestinal theory.
3. The Rice theory.
4. The Arsenic Theory.
5. The Carbonic Acid poisoning Theory.
6. The theory of a specific bacillus present in the blood, or in the soil or in dwelling houses.

Anaemia Theory: This theory is not recognized at the present day or if it is, it can only be by those who have no knowledge of Beri-Beri. It is true that one does occasionally see a mild form of anaemia in Beri-Beri patients, but the same condition would occur in any individual who was compelled to remain in bed for weeks together. Anaemia does not produce Beri-Beri, and Beri-Beri does not produce anaemia. The co-existence of the two diseases is due entirely to a separate causation and when the two are present together, careful investigation will prove that its anaemia results from malaria, should the patient have lived or be living in a malarious
district, or it may be due to some intestinal parasite as the ankylostoma duodena or to other causes. Examination of the blood of Beri-Beri patients does not reveal a condition of anaemia. The treatment of the two diseases is different; arsenic which is such a valuable drug in at least some forms of anaemia is practically useless in Beri-Beri except as a general tonic.

2. Intestinal Theory: this is based on the frequency with which one finds the presence of the ankylostoma duodena in the intestine and the successful results which are obtained in its treatment. The intestinal and the anaemic theories go hand in hand - they both produce great weakness and pallor, and the oedema resulting thence along with other symptoms such as palpitations and haemice murmurs might suggest Beri-Beri but a careful search should guide one in changing his idea and looking elsewhere for a real causation. Possibly there may be a parasite living in the intestine.
producing a toxin which becoming absorbed into the system causes nerve degeneration.

3. Rice fever: the two principal exponents of this theory are Dr. Hoce of Sarawak and Captain Hoce of the Indian Medical Service. Dr. Hoce holds that the disease can be traced to mouldy rice and is introduced into the system as a minute fungus or mould which finds a suitable medium in rice which has been kept for some time exposed to damp. He further states that the action of the fungoid growth may be increased by the excrement of a small beetle which is always to be found in the rice bags, after the grain has been husked and stored in the bags for several weeks or months. To justify this assumption, he adduces the following facts:

1. That the people who suffer most from Beri-Beri are those who eat imported rice which is sold in bags and comes chiefly from Siam and Java.
2. That those who live on freshly husked padi (padi is rice with the husk still
surrounding it) scarcely ever develop Beri-Beri and that the women who live at home and hull their rice as it is required, are rarely attacked by the disease.

3. That those who have to make long journeys into the jungle suffer from Beri-Beri because they have to take with them imported rice which in course of time develops the mould or fungus.

4. That a large percentage of those who have developed Beri-Beri will recover without medicine if fed upon freshly husked padi, whilst those who continue to live upon imported rice, an equally large percentage will certainly die and gives as an illustration the case of 89 male Dyaks who had contracted Beri-Beri during their journeys in the jungle and who had been living upon imported rice. They were placed in villages where none but freshly husked padi was used. Of the 89 attacked, 33 recovered and 6 died, whilst out of 128 similar cases, where the people lived on imported rice, 47 died.
Two monkeys fed upon mouldy rice developed some of the characteristic reflex actions of Beri-Beri and at the same time showed a loss of energy but there was no edema.

Capt. Root draws attention to the possibility of infection being caused by a microcosm inhabiting rice. Various rice liquors from different sources of rice were made and the organisms in each proved the identical. He also found the same organism in the yeast which is used to ferment the rice liquors with and further adds that the blood and cerebro-spinal fluid of Beri-Beri patients contained the same organism. The organism is described as a motive diplococcus developing by spores, the spores splitting into two, becoming elliptical and grow out into rods.

The rice theory has a great deal in its favour but we require greater evidence to determine it as a definite cause. Rice is the staple diet of the Asiatic and it would be interesting...
to know the actual numbers who live on
imported rice, compare them with those who
live on freshly husked padi and ascertain
the number of persons attacked under the
two conditions. If rice is such an important
factor in the causation, it is reasonable to
expect a much larger proportion of Beri-Beri
patients than is accounted for. We would
also almost expect, amongst the imported
rice eaters, men and women to be affected
equally, but this is far from being the case.
There are certain tribes who live largely on
millet, sago and potatoes and yet they are
attacked with Beri-Beri - it is possible that
the organism is capable of growing and
thriving upon these articles of food in the
same way that it is supposed to do on rice.
In Manchuria, millet is the staple article
of food but Beri-Beri is not unknown.
The Hadzays - natives of Lakeu - grow their
own padi and husk it as it is required, but
I have never known a case of Beri-Beri amongst
them, largely due to the fact that they do
not take European medicine. Their mode
of life is different - being an isolated one.
They do not engage in mining nor in opening new land.

Are we not entitled to expect more cases of Beri-Beri in Europe where all the rice that is used is imported?

Rice, per se, cannot be considered as the cause of the disease.

It is advisable to recommend those who have been attacked with Beri-Beri to remove from the infected district and in all probability they continue to live on imported rice in their new surroundings and the improvement which does take place in such instances cannot be judged as being due to change of diet, for there has been none, but it is due to change of locality and so we are inclined to regard Beri-Beri as a place disease and not a food disease.

In support of the rice theory, Baron Sanyoeki, in publishing statistics of the prevalence of Beri-Beri in the Japanese Army, states:

1. That rice eaters are the only persons affected with Beri-Beri.

2. That rice eaters transport the disease to places where, before their arrival, it
3. That Beri-Beri and rice are inseparably connected, lack of nutritive substance being the cause of Beri-Beri.

4. The Arsenic Theory: The theory that Beri-Beri might have an origin in arsenic was first published by Surgeon-Major Norr and Dr. Reynolds in the British Medical Journal for October 1901, in which they detail the case of a lady suffering from peripheral neuritis almost identical with arsenical poisoning and Beri-Beri. In this case there was no possibility of an alcoholic origin as the patient was a total abstainer from alcoholic beverages, and a material origin can also be excluded. Although the clinical picture presented marked symptoms of Beri-Beri, yet many of them such as the erythema, pigmentation of skin with desquamation of the cuticle and absence of circulatory and respiratory symptoms point rather in favour of arsenical poisoning. The abnormal sensations in the legs in the early part of her illness might be accounted
for by the existence of pregnancy as the patient was confined of a dead child at full-term. Another point in favour of arsenic is the fact that the patient had lived largely on tinned foods and arsenic was proved by analysis to be present in the hair. It is worthy of note that some of the cases diagnosed as Beri-Beri may really be due to arsenical poisoning as grain dealers have been known to put arsenic in rice to preserve it from the attacks of beetles and in Aceh it is recorded that arsenic is spread on the rice fields to destroy the rats. This subject requires further enlightenment and all workers in districts where Beri-Beri exists would do well to keep this in mind and have the rice from time to time subjected to analysis. If Beri-Beri is proved to be due to arsenical poisoning, I think it hardly possible, we must desprease the term Beri-Beri from our nomenclature and substitute a peripheral neuritis due to Arsenie.
5. Carbonic Acid poisoning theory - Dr. Ashmead of New York maintains that Beri-Beri is due to poisoning with carbonic acid gas. His experience of the disease in ships is considerable and he holds that he has always found a source where the carbonic acid gas came from. The point, he states, is due to carbonic poisoning of the red blood corpuscles and peripheral nerves, and further adds that rapid recovery is due to removal from the vitiated air of the ships to a poorer atmosphere and is similar in effect to removing the sick from low-lying plains to higher land and thereby increasing the supply of oxygen.

He denounces the theory of food as a cause, its only effect can be insufficiency of nutrition.

Dr. Ashmead asks the question, "If the Manchester cases of Beri-Beri were caused by arsenical (beer) poisoning, why are not all cases of Beri-Beri due to some chemical poison?"

An examination of the symptoms caused by carbonic acid poisoning fully disprove this.
Theory, and are in no way connected with Keri-Keri.

Carbonic acid gas is a product of respiration of animal life, that it exists in combination in many minerals we know, and that it is formed on the complete combustion of carbon in the presence of an excess of oxygen.

When diluted with air, carbonic acid gas is more or less respirable and inhalation of air with one half of carbonic acid produces a pricking sensation in the nostrils and larynx, together with headache, tinnitus aurium, giddiness, constriction and oppression in the chest followed by unconsciousness and a fatal congestion of the brain.

Should the proportion of gas be still less in quantity, it causes giddiness, feebleness of the legs and precordial distension, followed by slow and shallow respirations and more or less confusion of the mind and senses.

I do not agree with the exponent of this theory as to the cause of Keri-Keri except in so far that it may be due to a chemical poison, but that poison is certainly not carbonic acid gas, but in all probability a chemical
poison or toxin originating from bacteria, in other words, an organic poison.

6. The theory of a specific bacillus existing in the blood or outside the body, in the soil or surroundings of buildings. I infinitely believe that it is to this source that we must look for a real and definite cause.

Having limited means at my command, it has been impossible to carry out the researches in such a manner as I would have desired. In order to ascertain the existence of an organism in the blood, I selected, at different times, patients who were suffering from various stages of the disease and who had been removed from the place of infection. The method adopted was as follows.

A finger was well scrubbed with soap and water and washed with turpentine, then washed in 1 in 1000 perchloride of mercury solution and finally sponged with ether. The finger was then pricked with a needle which had
previously been sterilised by heat, and films of blood made on cover-glasses which had been cleansed by a solution of trichromate of potassium and kept in a solution of alcohol or in equal parts of alcohol and water. Some of the films were examined in a fresh and unstained condition, others were rendered permanent by exposing them to 120° Centigrade in a dry-air steriliser. They were stained with methylene blue, fuchsin, thionin and gentian violet. Sometimes a double stain was used. In no instance was I able to discover an organism either in the interior or exterior of a blood corpuscle.

After going through the same process of cleansing the skin of a finger, tubes of peptonised broth with agar-agar were inoculated with blood under the greatest precautions, then incubated. In one or two instances only did a growth show itself. This growth was white and spread itself very thinly over the surface of the agar-sloped tube. It did not liquefy the agar, nor did it penetrate into the substance of the growing medium.
Films were made and stained and examined with 1/12 oil immersion lens, revealing rod-shaped bacilli with rounded ends, a clear centre but deeply stained bodies at either end; occasionally, a body less deeply stained occupied the centre of the bacillus. Micrococci were also present.

Plate cultures were made from the tubes and after 24 hours incubation, the growth had the same characteristics as that of the tube growths. It spread rapidly over the surface but microscopical examination detected more cocci than bacilli.

In all its instances, one could not come to any definite conclusion as to the nature of the organism and as to whether it had anything to do with the disease or not.

I conducted the experiments in series of six cases at a time but the majority of them gave negative results.

I am inclined, therefore, to look upon this growth as one due to an organism which existed in the air of the ward in which Beri-Beri patients were living, and it
entrance into the tube at the time of inoculation was purely accidental. Tubs were also inoculated with dust from the Beri Beri wards, and the growths obtained were very similar to those described above, only that there was usually present more than one colony and microscopic examination proved them to be cocci - no bacilli could be found. Tubs were also inoculated with the soil surrounding the hospital buildings, but the growths obtained were quite different to those of the blood and dust inoculations. They were finger-shaped and penetrated into the interior of the growing medium.

I am fully convinced that the organisms found in the three instances have nothing in common with Beri Beri and that to meet with any degree of success, one must make the inoculations of tubs in the district where the disease prevails and also at the very earliest signs of the disease appearing, and not sometime after the patient has been removed from the endemic area.
Several workers at this subject describe organisms present in the blood. Fishers of Canada mentions the presence of an unicellular organism both inside and outside the red blood corpuscles. It is smaller than the parasite of malaria and apparently forms pigment granules and has a cycle of development.

Rikets and Winkler are the two greatest advocates of the bacillus theory and their treatise on Beri Beri takes up the subject in an exhaustive manner. They describe granular and rod-like bodies existing in the blood of Beri Beri patients and to demonstrate them, it appears necessary for the patient to be living in the endemic area at the time of examination; at least one must not allow too great an interval of time to elapse between removal from the infected area and the time of examination. Otherwise negative results are sure to be obtained.

That these organisms probably have some causal connection with the disease is proved.
by examining the blood of healthy persons a few days after arrival in the infected district when no bacteria will be found, but examine the blood of the same persons after a few weeks residence, and organisms will be found. Their theory is further strengthened by inoculating sterilised nutrient media with a drop of blood, obtaining growths of organisms and infecting solutions of these growths subcutaneously in dogs and rabbits and producing some of the paralytic symptoms of the disease and demonstration of degeneration of several nerve fibres.

Pathological Anatomy.

The external appearance will depend very much upon whether the patient has died of the more acute tropics form or of the more chronic paralytic type. Still, as a rule, when a patient dies of Beri-Beri, there is a more or less general oedema.

Post mortem lividity shows itself early. In acute cases, the body is swollen, the neck and back are cyanotic and on cutting into the
Connective tissue, there is considerable venous engorgement. The blood is dark red and fluid, lungs, these, and more especially the bases, are congested and edematous; on cutting into them, dark fluid blood escapes. One or both pleuræ may show signs of recent pleurisy, and sometimes several ounces of fluid may be found in a pleural cavity.

The heart: On opening the pericardial sac it will occasionally be found filled with fluid, and in some cases, so great is the amount that, by its pressure, it causes the heart to lie almost transversely. The arteries, especially the right, are engorged, and the right ventricle is almost always dilated. The myocardium is soft and congested. The large arteries leading from the heart have nothing peculiar.

In the abdomen, a certain amount of fluid in the peritoneal cavity may be met with. It is of recent origin.

The liver: As a result of the general engorgement, this organ is enlarged and congested. Its enlargement extends in all directions, reaching close to the spleen.
The Spleen: This organ, likewise, participates in the general congestion, and is enlarged and congested.

The Kidneys: These show a general congestion. On peeling the capsule, a smooth, nor or less, congested surface is exposed.

The Intestinal Canal: shows indefinite venous hyperaemia. The parasites, such as antrophotes and frequently found in cadavers, are not pathogenic.

The Brain and Spinal cord: The appearances consist of venous hyperaemia of the cerebral and spinal meninges, with sometimes slight congestion of the brain and spinal cord substance.

It has not fallen to my lot to obtain sanction for a necropsy in many cases and the above observations are obtained from a few cases only. Usually, the examinations had to be conducted at unusual times and at great inconvenience.

The coarse pathological conditions reveal nothing peculiar to the Disease and therefore it is
to the minute pathological changes that we have to look to such as, the degeneration of nerve fibres and changes in the spinal cord but my observations are not yet complete enough to record in this thesis.

The following photographs will show pretty clearly atrophic and tropial form of Beri-Beri.

The above photograph of Tropical Beri-Beri is very typical. The oedema is general and of two months duration. The patient is quite unable
to stand or walk. There is severe cardiac
dyspnoea due to effusion in pericardial
sac.

This photograph depicts a patient with Acute
'Cardiac Beri-Beri'. There is no oedema
and there is no effusion into the peri-
cardial sac but there is intense cardiac
distress. The patient is unable to walk.
This photograph shows three patients suffering from the Atrophic form of Beri-Beri. The one on the right has nearly recovered, the man in the middle is quite unable to walk, whilst that one on the left can move about with the aid of a stick.
This photograph shows three contrasts. The patient on the left is suffering from malaria cachexia. The middle man is a case of Atrophic Beri Beri on the road to recovery, whilst the man on the right is recovering from Tropical Beri Beri.